# **SUNLIGHT ROBBERY:**

Health benefits of sunlight are denied by current public health policy in the UK



## **Oliver Gillie**

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The author has no connection with commercial interests that might profit from any aspect of this work. In particular the author has never worked for or accepted payments from makers of sunlamps or vitamin supplements. The author has financed the research for this work himself and paid for the printing and distribution of this document. The only payments received have been for journalism connected with the work.

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## Foreword: Sunlight and Survival

#### A magic shotgun

Most people in Britain and other northern industrial countries have a level of vitamin D in their bodies that is insufficient for optimum health, especially in winter. These low levels of vitamin D are now known to be associated with a wide spectrum of serious disease much of which leads on to premature death. The diseases associated with D deficiency include more than a dozen types of cancer, multiple sclerosis, diabetes type 1, heart disease, high blood pressure, and schizophrenia as well as the classic bone diseases: rickets, osteoporosis and osteomalacia. Evidence that these diseases are caused, at least in part, by a deficiency of sunlight and vitamin D has accumulated at an accelerating pace over the last 10 years but is not well known, even to professional scientists. This review outlines the evidence showing that these diseases are linked to deficiency of vitamin D and the serious implications this has for public health policy in the UK and the Republic of Ireland.

"If what you say here is true then vitamin D is a "magic bullet", said one colleague. I had not thought of it before, but he is right. Vitamin D can fairly be compared with the first 'magic bullet', the antibiotic Salvarsan, discovered by Paul Ehrlich in Germany in 1910 and used to treat syphilis which was at that time a scourge similar to HIV today.

Ultra-violet radiation and the vitamin D it generates produced the first cures of tuberculosis of the skin in 1903. The results were seen by the world as miraculous and earned the Danish physician Niels Finsen the Nobel Prize. Soon afterwards, rickets, a common disease causing devastating deformity, was also being cured with ultra-violet radiation. Within a few years, foods such as cod liver oil, which contain vitamin D, were being used to cure rickets. Again the results seemed miraculous. So sunlight and vitamin D qualified as a magic bullet almost 100 years ago.

Now we are confronted with a wealth of new evidence, varying in strength and quality, suggesting that vitamin D deficiency is important in causing a wide spectrum of chronic disease. The effect of D deficiency goes way beyond the diseases of the bone that have long been known to be linked with vitamin D. But this new evidence is unfamiliar to most doctors and scientists and quiet rightly they are sceptical. At first sight it seems unlikely that vitamin D deficiency could cause so many entirely different diseases. And we still have little idea how D deficiency acts, together with other risk factors, to cause one disease in one person and other diseases in other people. However the accumulated evidence linking D deficiency to chronic disease is extensive and consistent. So it has great strength as a coherent body of work.

In the next decade vitamin D may be recognised once again as a magic bullet – or more likely as a magic shotgun, able to do much more than a single bullet. If an optimal level of vitamin D is maintained throughout life many tragic diseases that presently destroy lives may be prevented. We should not be surprised at this. Northern Europe is not man's natural environment. Recent studies of human DNA tell us that man evolved in central Africa where the tropical sun provides plentiful ultra-violet light for vitamin D synthesis in the skin every day of the year. Lack of sunlight resulting from our northern location and our maritime climate makes the British Isles an extreme habitat compared with the tropical regions where human beings first evolved.

#### Survival of the pale

Some 50,000 years ago small bands of people, almost certainly dark skinned, moved into Europe via Turkey, a story that has been reconstructed by Professor Stephen Oppenheimer in his authoritative book '*Out of Eden*'[1]. The virgin territory of Europe provided plentiful food in summer, however in winter not only was food in short supply, but also low levels of vitamin D must have increased the susceptibility of these pioneering bands of people to disease and reduced their fertility. Vitamin D deficiency causes infertility, and during pregnancy and lactation stunts the growth of the brain and other organs in the foetus and newborn. How this occurs, causing common diseases such as schizophrenia, is only now beginning to be understood and is explained in some detail later in this review.

A pale skin exposed to the sun makes vitamin D six times faster than a dark skin and so pale skin has an important advantage in northern countries. Natural selection, powered by infertility and disease, took its toll

on the migrants heading north. Human skin colour changed by genetic mutation over a period of thousands of years to a lighter shade. These changes in skin colour actually occurred several times in different human races in various parts of the globe. Not only do native peoples everywhere have paler skins the further they live from the equator, women and children always have paler skins than mature men of the same tribe, a neat adaptation to provide the maximum vitamin D that is needed for fertility of women and growth of children [2].

The first Europeans probably spread along coasts and rivers where fish provided an important supplementary source of vitamin D. But as the pioneers pushed further into the interior of Northern Europe those with paler skins were more likely to survive and contribute to the following generation. The British Isles and Scandinavia were the end of the trail, further north than human beings had ever lived before. The cloudy maritime climate of the region often blocks out sunlight even in high summer adding to selection pressure for a pale skin.

This evolutionary story, which has only been fully established recently, helps us to understand our needs today. The pale European skin enables the first weak rays of spring sun to be used to make vitamin D and the last weak rays of autumn to be utilised before winter comes. This extends the period during which vitamin D may be synthesised and so must have extended the period of optimal health and fertility providing important survival advantages. Children with British ancestry are often blond even though their parents have darker hair and skin, an adaptation allowing maximum uptake of weak spring and autumn sun at an early age.

Sunbathing to take advantage of the most sunny times of day and the earliest and latest sun of the season is a natural and necessary activity for human beings who have evolved to survive in this northern environment. Tanning also provides a benefit. Pale skin in spring gives a maximum response to the weak spring sunlight. As spring turns into summer and the sun gradually becomes stronger, pigment is deposited in the skin providing some protection from the harsh midsummer sun and allowing longer exposure without burning. This process involves the death of skin cells but there is no reason to believe that this should be alarming or anything other than a wholly natural process.

Sunlight is the primary source of vitamin D for people everywhere except for communities living within the Arctic Circle who obtain the vitamin from oily fish and other marine animals. Now that we live in cities and spend little time in the open air many people in the UK suffer from serious deficiency of vitamin D caused by insufficient exposure to sunlight. This has been exacerbated by a fear of skin cancer which has dominated national policy on sunlight.

#### **Sunlight policy**

Since 1994 the United Kingdom government, backed by a number of cancer charities and other organisations, has organised a campaign for prevention of skin cancer that warns against exposure to sunlight. This policy, which is currently based on an Australian model, gives advice to cover the body with clothes or suncream, wear a hat, seek shade between 11am and 3pm, and avoid tanning. The policy cautions the public to avoid exposure to the sun while making no concessions to the health benefits of sunlight. In fact any benefit derived by this policy in prevention of skin cancer is greatly outweighed by the disease deficits incurred by the loss of vitamin D.

British government policy on sunlight advocates a lifestyle particularly unsuited to the UK. It is an unnatural lifestyle and is recognised as such by a large section of the public who have rejected it. Continuing with these government recommendations can only increase vitamin D deficiency in the population and so lead to an increase in ill health and premature death.

A new public health policy on sunlight and vitamin D, taking in the views of a wide range of British and foreign experts, is badly needed in the UK. It is suggested here that any such policy should include a recommendation for regular sunbathing in strong sunlight whenever possible while taking care not to bake or burn. There is no sound scientific basis for alleging that a tan is ever unhealthy and so the slogan 'There is no such thing as a healthy tan' should be dropped.

People enjoy sunbathing. It is the normal and natural way to obtain vitamin D. Sunlight costs nothing and has very great health benefits. Sadly in the UK we get too little of it. So it is folly to discourage sunbathing in the way that has been done by government policy over a period of 10 years or more. A wise government policy on sunlight should encourage sunbathing while advising people how best to sunbathe safely. Such a policy should also recognise that for six months of the year sunlight in the UK is too weak to provide any vitamin D. For optimal health most people in the British Isles need to take a vitamin D supplement, at least in winter, and probably year round. This is particularly important for special groups such as mothers and babies,

people with dark skins, people who are overweight or stay mostly indoors, people who work at night and sleep during the day, and the elderly.

Present government policy on sunlight is the product of limited specialist knowledge, particularly of dermatologists. Bone specialists and others have been ignored. A considered policy on sunlight, taking in a wide spectrum of views, is badly needed in the UK. This report reviews the detailed scientific and medical evidence that must be considered in developing a broadly based national policy on sunlight for the UK.

Oliver Gillie

## Summary

**1.** Many people of all ages in the UK have undesirably low levels of vitamin D. In winter the majority of the population are at risk of serious vitamin D deficiency or insufficiency which makes them vulnerable to chronic illness.

**2.** Sunlight is the most important source of vitamin D for people in the UK providing about 90% of the total vitamin D in the body. Small amounts of vitamin D are obtained in the diet from fortified margarine, butter, meat, eggs, oily fish and some breakfast cereals and from supplements.

**3.** After synthesis in the skin vitamin D is processed into its active form in several different tissues of the body. Vitamin D plays an important role in regulating growth of some 30 or more tissues. Vitamin D works in several ways. It controls calcium absorption, influences differentiation and maturation of cells, triggers cell death (apoptosis), and switches genes on and off. Despite being described correctly as a vitamin, vitamin D is also a hormone. It alters growth signals to cells, inhibits growth of blood vessels which is an important part of tumour formation, and has a modulating activity on the immune system.

**4.** Accumulating evidence now suggests that vitamin D deficiency increases the risk of many chronic diseases including 16 different cancers, several nervous system diseases including schizophrenia and multiple sclerosis, diabetes types 1 and 2, as well as being a contributory cause of heart disease, raised blood pressure (hypertension), inflammatory bowel diseases, polycystic ovary disease, menstrual problems and infertility, infections and dental decay. The cancers that have an increased risk as a result of D deficiency include some of the most common types: cancers of the breast, bowel, ovary, and prostate.

**5.** The cost of diseases caused by vitamin D deficiency has been estimated to be \$50 billion per year in the United States and must be of the order of billions of pounds per year in the UK. According to government figures, falls and fractures alone cost some £2 billion per year in the UK. Scientific trials have shown that a substantial proportion of these falls and fractures can be prevented by vitamin D and calcium supplements. Diabetes costs £1.7 billion a year in the UK (official figures) and a substantial part of this could be saved if blood levels of vitamin D in the population were higher. The cost of other diseases such as cancer, heart disease, hypertension and schizophrenia must also run into billions per disease per year and a substantial number of people might be prevented from getting each of these diseases if vitamin D levels in the population were improved. Sunbathing is a popular and efficient way of increasing vitamin D levels in the population.

**6.** Deficiency of vitamin D during the final months of pregnancy and/or in the first months of life increases the risk of several chronic diseases e.g. schizophrenia, multiple sclerosis and diabetes type 1. The problem is most acute for babies that are breast fed because breast milk of women in the UK is generally deficient in vitamin D while artificial milk is supplemented with D. Pregnant and breast feeding mothers should be encouraged to sunbathe whenever possible, and should be actively encouraged to use NHS vitamin drops (which contain D) or other D supplements for themselves and their babies.

**7.** Casual exposure of the hands and face to the sun has in the past been thought to provide enough vitamin D for good health. But this exposure is now known to be insufficient in the UK climate. Active exposure of the skin to the sun by removing clothes and sunbathing is necessary to provide healthy levels of vitamin D that will provide a reserve for the autumn, winter and early spring (October to March or later) when the sun is not strong enough to induce synthesis of vitamin D. Vitamin D has a half life in the body of about six weeks and so high levels must be achieved in summer to provide levels in the body which remain sufficient at the end of winter.

**8.** A white skinned person in the UK needs at least three 20 minute sessions per week of sunbathing in bright midday sunlight with few clothes to obtain an optimum amount of vitamin D. Longer will be needed if sunbathing occurs at off-peak times for ultra-violet light (before 12am or after 3.00pm) or at the beginning or end of the summer (April or September) when the sun is lower in the sky for most of the day.

**9.** Dark skinned people take up to six times as long to make the same amount of vitamin D as white skinned people. So in the UK a person with dark skin would ideally need to sunbathe for three two hour sessions per week to achieve an optimum amount of vitamin D. It is impossible for most people in the UK to achieve this length of time in the sun in practice.

**10.** People with dark skin living in the UK have been found to be more vulnerable than white skinned people to a number of diseases associated with vitamin D deficiency including schizophrenia, diabetes, heart disease, hypertension and tuberculosis. Ways are needed of encouraging use of vitamin D supplements and/or artificial tanning by people with dark skins in order to improve their vitamin D levels and general health.

**11.** Deficiency of vitamin D may also be prevented in the population at large by taking supplements, especially in winter. However absorption of vitamin D, which is fat soluble, is not always efficient. In particular, old people and people who suffer from inflammatory bowel disease, which itself is caused in part by vitamin D deficiency, may have trouble absorbing D supplements. Sunlight, on the other hand, is free, enjoyable and widely available. Even though the sun cannot always be relied upon in the UK climate it is the natural source of vitamin D.

12. Excessive exposure to the sun may cause sunburn, skin aging, and skin cancer. Melanoma skin cancer causes some 1,750 deaths a year in the UK but the cause of melanoma is not clear and it is possible that less than half of these deaths may be attributed to sun exposure. Regular exposure to the sun seems to protect against melanoma while irregular exposure increases risk. This is probably because vitamin D protects against melanoma while excessive exposure to the sun causing sunburn may induce melanoma. A reduction in exposure to sunlight in the UK, as recommended by the government, might actually increase the incidence of melanoma rather than reduce it. Melanoma may occur on parts of the body such as the soles of the feet that are seldom exposed to the sun. Other types of skin cancer which are directly caused by excessive exposure to sunlight are very common. However they can generally be treated relatively easily and cause a few hundred deaths a year.

**13.** UK sunlight policy has been largely determined by dermatologists who bear the onerous task of treating skin cancer. Doctors concerned with the problems of osteoporosis and other bone disease do not appear to have been consulted despite an obvious interest. Now that vitamin D deficiency is known to be associated with a wide spectrum of disease there is a need for a new national sunlight policy that recognises the positive aspects of sunlight.

14. SunSmart, the UK's skin cancer awareness campaign, originated in Australia which has a much sunnier and hotter climate and an incidence of skin cancer about six times that of the UK. SunSmart advises the public to cover up, seek the shade, and wear sunscreen. It fails to provide any advice suggesting that people should sunbathe in order to obtain vitamin D. Anyone following the SunSmart advice in the UK risks becoming deficient in vitamin D and so risks chronic ill health. The campaign is totally unsuited to British needs and should be abandoned.

**15.** The public need constructive advice on how to sunbathe safely without burning. This should include advice on how to make best use of the intense midday sun, how long to spend in the sun at different times, how to recognise warning signs of burning, how to protect the eyes and the sensitive skin of the face when sunbathing, and how to make best use of the weak sunlight at the beginning and end of the summer.

**16.** The SunSmart programme has made extensive use of the slogan: 'There is no such thing as a healthy tan'. In fact there is no scientific basis for condemning tanning which is a natural side effect of sun exposure. Scientific evidence suggests that a deep tan actually protects against melanoma, although very rapid tanning on holiday in fierce sun conditions may not be wise because of the risk of burning. Nevertheless the public recognition of a tan as a sign of good health is almost certainly well founded. Further attempts by cancer campaigners to suggest that a tan is unhealthy or that a pale complexion is desirable should be dropped.

## Recommendations

1. The current skin cancer awareness campaign including the SunSmart programme should be abandoned as soon as possible.

2. The Chief Medical Officer's 'sixth tip' for good health should be re-written as follows: Take every opportunity to sunbathe wearing as few clothes as possible for up to half an hour or more per day depending on skin type, previous exposure and time of day. But take care – sensitive skin may burn after only a few minutes. Be ready to cover up or seek the shade to avoid baking or burning. Encourage children to undress in the sun but take care that they do not burn.

3. A campaign that encourages the public to expose themselves safely to the sun should be planned and begun in April/May 2005. Those people who cannot easily expose themselves to the sun or prefer not to do so should be advised to take a vitamin D supplement.

4. The importance of vitamin D for pregnant and nursing mothers and infants needs to be recognised as part of any government policy on sunlight. The policy should encourage mothers to sunbathe and to allow their children to play safely in the sun, while also encouraging mothers to make use of NHS vitamin drops. A special campaign is needed to promote the use of these vitamin drops.

5. Immigrants and their families and other people with dark skin need special advice on how to raise their levels of vitamin D. They should be encouraged to sunbathe, but they should also be recommended to take vitamin D supplements year round.

6. For six months of the year the sunlight reaching us in the British Isles is not strong enough to make any vitamin D in the skin so serious consideration needs to be given to permitting and encouraging increased fortification of food with vitamin D and in providing cheap supplements of vitamin D to people of all ages. In the United States, for example, almost all milk is fortified with vitamin D. In France milk fortified with vitamin D (at 1 microgram or 40iu/100ml\*) is available as a choice. Such milk is not available in the UK because all fortification stopped following a scare about the effects of D-fortified milk on babies in the 1950s. A market in D-fortified milk and other commodities should be encouraged in the UK. Expert consideration needs to be given to the optimum level of fortification and how to encourage this market.

\* iu = international units. 40iu = 1 microgram vitamin D

7. In the past artificial tanning using ultra-violet lamps has been condemned by the medical profession, but new evidence suggests that risks may have been exaggerated. The risks of ultra-violet lamps need to be carefully re-evaluated and balanced against the benefits that may be expected from increasing vitamin D levels and preventing or ameliorating a wide spectrum of disease. Consideration needs to be given to recommending ultra-violet lamps and tanning salons to the public as a means of maintaining vitamin D levels during winter. Recommendations and controls concerning use of sunbeds should be reviewed with the aim of encouraging use of sunbeds while maintaining safeguards.

8. Research in vitamin D is presently conducted by a few enthusiasts who understand its importance but they often have difficulty obtaining financial support because the subject is unfashionable. In recognition of the role of vitamin D in so many chronic diseases priority now needs to be given to all kinds of research involving vitamin D from epidemiology through cell biology to trials of prevention and treatment of disease.

9. An international conference of doctors, scientists and policy makers interested in vitamin D should be held in the UK as soon as possible to review what is known about the effects of vitamin D insufficiency and deficiency, the risks and benefits of sunlight, and best policy on supplementation with vitamin D and fortification of foods with vitamin D.

**10.** A committee should be formed in the UK to formulate a new sunlight policy based on what is now known about vitamin D deficiency/insufficiency and disease. The committee should be representative of all medical and scientific disciplines bearing on the subject, together with lay stakeholders from associations representing people with diseases, such as osteoporosis, multiple sclerosis, diabetes etc, that are associated with vitamin D insufficiency.

## Part 1: Sunlight and vitamin D deficiency

#### Sunlight policy in the UK

UK government health departments have spent £3.24 million over 10 years on promotion of public health programmes which advise the public to avoid or curtail exposure to the sun [3]. The government advice is based on a Consensus Statement of the UK Skin Cancer Working Party [4] which was originally agreed 10 years ago and has not been updated. The programme is currently promoted by Cancer Research UK, although it is still largely paid for by the government.

Cancer Research UK have adopted the name SunSmart for their programme which appears to be modelled on an Australian Public Health programme of the same name. In effect the UK SunSmart programme advises the public that exposure to the sun should generally be avoided and that they should cover up with clothes and sunscreen, and seek shade. In Australia the SunSmart programme claims to have reduced the incidence of sunburn by 50% and slowed the rise in skin cancer deaths [5].

The UK SunSmart programme has made people more aware of risks associated with UV exposure and persuaded people to be significantly less preoccupied with tanning according to monitoring studies [3]. But it has had no impact in reducing the incidence of melanoma, the most serious form of skin cancer, which has doubled in the UK over the last 20 years [6]. And most important of all the programme takes no account of the health benefits to be obtained from exposure to the sun.

The government policy on sunlight, like any public health policy, should be based on a calculation of risks and benefits. Advice should only be given to the public to reduce sun exposure if it can be shown that this is likely to do no harm and to provide substantial benefit. Any such calculation made 10 years or more ago, when the current policy was devised, was made without knowledge of the wide range of disease now known to be caused by vitamin D deficiency or insufficiency. Even now the extensive link between D deficiency or insufficiency and chronic disease is not well known in specialist scientific and medical circles.

Furthermore any calculation of risk and benefit leading to the current policy must have been made in the mistaken belief, expressed recently by the National Radiological Protection Board and accepted by government, that casual exposure of hands and face to the sun in the UK will provide sufficient vitamin D [7]. In other words the calculation of risk and benefit involved in our current policy on sun exposure is based on erroneous parameters and so has no secure foundation in scientific evidence.

It will be shown here that sufficient vitamin D for optimum health cannot generally be obtained in the UK simply from casual exposure to the sun. Planned exposure, including sunbathing and wearing clothing that exposes the skin, is necessary in the UK to provide optimum levels of vitamin D. Disease caused by vitamin D deficiency and exacerbated by present UK sunlight policy is much greater than was imagined when the policy was first devised. Furthermore any benefit of the government's present sunlight policy, such as a reduction in deaths from skin cancer, is substantially less than has been thought as will be shown in detail later in this report. A greater exposure of people in the UK to sunlight inducing higher levels of vitamin D in the population as a whole is likely to make a substantial improvement to general health of people in Britain while causing a very small, or even negligible, increase in risk of skin cancer.

This report provides the detailed evidence that was used by the author as the basis for an article in *The Independent* newspaper [8] and other articles in magazines [9, 10] drawing attention to the problems of vitamin D deficiency in the UK.

#### Sources of vitamin D

Vitamin D may be obtained in small amounts from the diet. Margarine, butter, some breakfast cereals, liver and other meats and eggs provide small amounts of vitamin D. Oily fish (including herring, mackerel, sardines, salmon, trout, fresh but not tinned tuna) provide more substantial amounts but are eaten by a minority of people. In the United States vitamin D is added to milk but this is not permitted in the UK and some other European countries. Sunlight is the most important source of vitamin D, providing around 90% of the supply for most people in the UK [11, 12]. People with darker skins require to spend up to six times as long in the sun as people with white skins to make the same amount of vitamin D and are more likely to have low levels of vitamin D [13, 14].

Deficiency of vitamin D is well known to cause rickets in children, and osteomalacia and osteoporosis in adults

[11, 12]. However these effects on bone are only the most extreme results of vitamin D deficiency. It has been discovered over the last ten years that vitamin D is not only made in the kidney (from pre-vitamin D synthesised by sunlight in the skin) but in some 30 other tissues of the body and that vitamin D plays an important role in the differentiation of cells and tissues [15].

Insufficient sunlight causing a deficiency of vitamin D has now been implicated as a contributory cause of some 16 cancers, of several nervous system diseases including schizophrenia and multiple sclerosis, of diabetes types 1 and 2, as well as being a contributory cause of heart disease, raised blood pressure (hypertension), inflammatory bowel diseases, polycystic ovary disease, menstrual problems and infertility, infections and dental decay. A full list of diseases caused by vitamin D deficiency and a summary of evidence is provided in Part 2.

Vitamin D deficiency may generally be prevented by taking supplements but these are not well absorbed by everyone, particularly old people and those with inflammatory bowel disease. Exposure to the sun or to artificial sunlight on sunbeds may be the best source of vitamin D for these people.

The lamps used in sunbeds produce a spectrum of ultra-violet light which is increasingly close to natural sunlight. There is no reason to believe that the effect of this artificial light on the skin is substantially different from the effects of sunlight. Research on the effects of sunbed use have produced conflicting results, but the most recent and most complete study [16] shows a small increase in risk of skin cancer only for those with the fairest skins. This small additional risk for some people has to be balanced against the very great gains in health that are associated with higher levels of vitamin D. Sunbeds may be a healthy option that can now be recommended, if used carefully, for maintaining winter levels of vitamin D in the UK and Europe generally where winter sun is not strong enough to support vitamin D synthesis.

Sunlight may have other benefits, not at present fully understood, apart from supporting vitamin D synthesis. For example, sunlight stimulates the production of alpha-melanocyte stimulating hormone ( alpha MSH ) [17, 18] which has an important role in regulating the action of insulin and energy balance [19]. So alpha MSH is important in controlling body weight and in preventing diabetes and obesity [20, 21]. In addition blue light reduces the production of melatonin by the pineal gland and so affects the sleep cycle. Winter depression (seasonal affective disorder or SAD) is widely believed to occur as a result of disturbance of this cycle. These are further reasons not to discourage exposure to the sun.

#### Vitamin D status of the UK population

Most children in the UK have less than optimum levels of vitamin D in both summer and winter. In winter, UK children have an average vitamin D level of 52 nmol/l (measured as serum 25(OH)D)[22] which means that one third to a half of the children have insufficient vitamin D for long term health (see Table 1). In summer the average increases to 80 nmol/l, a barely adequate level which does not allow the accumulation of any reserve to provide for the winter. Dark-skinned children in the UK have levels of vitamin D of 36-42 nmol/l (not differentiated by season)[23] – levels which lead to hyperparathyroidism and are not optimum for health [24].

A marked seasonal variation in vitamin D levels occurs in all European countries, typically with average winter levels that are deficient or insufficient for health [25]. Teenagers and young adults in Europe often have lower levels of vitamin D than children, presumably because they spend less time outdoors. Adolescents in France, for example, have been reported to have average levels of 71 nmol/l in summer and 21 nmol/l in winter [26], while young adults in Germany have average levels reported to be 70 nmol/l in summer and 30 nmol/l in winter [24].

The French teenagers and young adult Germans may safely be assumed to have a similar lifestyle to those of the same age in the UK. If anything they have a greater opportunity for exposure to the sun because of sunnier summer weather and summer holidays which begin earlier in late June or early July. So, extrapolating from the European figures, it seems likely that half of the teenagers and young adults in the UK have levels of vitamin D that are deficient in winter and likely to cause disease.

Elderly people in the UK have been found to have average levels of 35nmol/l vitamin D in summer and 23nmol/l in winter [27] – levels that are insufficient for good health in winter or summer and are associated with osteomalacia, muscle weakness, and a tendency to fall which inevitably causes bone fractures [28, 29].

In summary these figures show that most people in the UK have inadequate levels of vitamin D all year round. The average summer levels are in the 'hypovitaminosis D range' which means that the level is not high enough to ensure an adequate level during the following winter. This is borne out by the winter figures which show that the majority of people of all ages in Europe, and particularly in the UK, are at risk of serious vitamin D deficiency in winter.

It is not possible for most people in the UK with indoor jobs to obtain an optimum level of vitamin D from sunlight because weekends when they are able to go out in the middle of the day cannot be relied upon to be sunny. So it is advisable for most people in the UK to take a vitamin D supplement at least in winter and possibly year round. Yet this is not recognised in official advice in the UK which has not changed for many years. According to advice no supplements of vitamin D are necessary for adults (aged 19 to 50+) who are living a normal lifestyle. People over 65 and women who are pregnant or lactating are advised to take a supplement of 400iu vitamin D per day.

However 400iu per day is considered by expert opinion in the United States to be insufficient to maintain circulating levels of vitamin D at an optimum level [30-32]. A supplement providing at least 1000iu of vitamin D per day is needed to maintain optimum levels in the body. The UK Expert Group on Vitamins and Minerals regards 1000iu per day as safe, well tolerated long term, and necessary in some groups to avoid deficiency. So a supplement of 1000iu per day may be taken without any reservations. However, vitamin D will not reach the optimum level in the body with this amount of supplement unless substantial exposure to the sun is also obtained.

Experiments with lactating mothers undertaken by Bruce Hollis and Carol Wagner at the Medical University of South Carolina, Charleston, have established that a supplement of 4000iu vitamin D per day is needed to pass enough vitamin D on to the nursing infant to raise the level of the vitamin in the baby's blood [33]. While this level of supplementation appears to be safe, and arguably provides a blood level of the vitamin that would normally be achieved by our ancestors living close to nature, it does not yet have approval of the authorities in the UK.

To obtain as near optimum blood levels of vitamin D as possible, pregnant mothers in the UK should take a supplement of 1000iu vitamin D and sunbathe whenever possible (while taking care not to burn) – otherwise they will not obtain the optimum level suggested by the work of Hollis and Wagner. Mothers who are breast-feeding their babies should also sunbathe if possible to improve the amount of vitamin D in their milk. Breast fed babies should be given the standard infant vitamin drops which are recommended by the government. According to the official schedule these drops are intended to provide 340iu per day up to six months of age and 280iu per day from six months to three years. It is completely illogical to provide older babies with a smaller dose of supplement but these recommendations, which are based on the amount of vitamin D in a teaspoonful of cod liver oil, have remained unchanged for many years.

Vitamin D deficiency in the UK is greatly exacerbated by inappropriate advice on sun exposure given in the SunSmart programme and in other government guidance. Those who follow this official advice are likely to develop below average vitamin D levels which in the long term are hazardous to health. Encouragement to sunbathe could do much to improve vitamin D levels in people of all ages.

Deficiency of vitamin D may begin in the womb or shortly after birth and may continue throughout life, or it may begin at any age. For example, deficiency may begin with a change in lifestyle brought about by more intensive demands of work or as leisure habits change with age. Once D-deficiency has initiated disease, provision of supplements of the vitamin may or may not be able to reverse or limit damage. Bearing all this in mind the only safe policy is to obtain optimum amounts of vitamin D from the sun and/or supplements to ensure that deficiency or insufficiency never occurs. People who cannot obtain regular exposure to the sun in summer (two or three times a week) should take vitamin D supplements year round.

#### Table 1.

#### Vitamin D levels and risk of disease after Zittermann [25].

This table provides guidance on what levels of vitamin D are adequate and optimal. However the exact cut-off points are a matter of interpretation and by no means agreed by all scientists familiar with the research. Vitamin D is measured as nmol/l 25(OH)D in serum (2.5nmol/l = 1 microgram/l)

25(OH)D nmol∕l	Associated disease/State of health
Below 12.5	Frank deficiency – can result in rickets
Below 25	Deficiency – may lead to rickets or osteomalacia in the long run. Increased risk of heart attack
Below 50	Insufficiency – may lead to hyperparathyroidism. Reduced muscle strength. Danger of falls in elderly.
Between 50-100	Hypovitaminosis D – body stores are depleted. Parathyroid levels may be slightly elevated but remain in normal range
Between 100-150 or more	Adequate level for optimum health

#### What is the optimum exposure to the sun in the British Isles?

How much of the body needs to be exposed to the sun for how long in the British Isles to obtain optimum levels of vitamin D? Occasional exposure of the hands and face to sunlight is widely believed to provide sufficient vitamin D for good health. This view is repeated in the UK National Radiological Protection Board's report on Health Effects of Ultra-violet Radiation [7] which says, in its executive summary, that: "short periods outdoors, as normally occur in everyday life [in the UK], will produce sufficient vitamin D, and additional or intensive exposures will not confer further benefit."

The NRPB's conclusion is based primarily on observations of nine elderly patients aged 70-94 who lived in a hospital ward near Nottingham where they had access to a sunny terrace [34]. The patients were monitored over three months from 26 April to 26 July. All the patients began with levels of vitamin D (measured as 25-OHD) less than 50nmol/l, typical of elderly people in the UK, but nevertheless marginal and likely to lead to problems of falls, osteoporosis [28, 29] and possibly other diseases in the long term.

Over the three months of the experiment the patients made a small average gain in vitamin D but by the end of July with the summer almost over seven still had levels of vitamin D below 40nmol/l. A level greater than 40nmol/l is needed in summer to maintain plasma levels of vitamin D above the danger level of 20nmol/l during the following winter [35]. Three of the seven had levels of 22.5nmol/l or below and would be likely to run into problems of severe deficiency even before the arrival of winter.

Only one patient had a normal summer level of vitamin D (79.4nmol/l) and he had been discharged from the Nottingham hospital and 'spent many hours outside in the garden' and so had received relatively intensive exposure. This contradicts the NRPB's conclusion quoted above that 'intensive exposure' may be expected to confer no extra benefit. Even so despite intensive exposure and the 'normal' level of vitamin D achieved by this veteran gardener the level achieved was less than optimal [25] and would be likely to leave him with insufficient vitamin D during the following winter.

This experiment on just 10 patients exposed to the sun for three months during an uncertain English summer is hardly a sound basis for a national policy on sunlight. Yet it seems to have achieved that status. The experimenters record that there was an uneven distribution of warm sunny days over the three months of the study with most of these in May. Little time was spent outdoors by the patients in June and July because the days were not warm and sunny. It seems to have been an average English summer, a highly variable summer that cannot be relied upon to provide clear sunlight much of the time. Because of this variability a study such as this, made over one English summer, cannot be a satisfactory basis for making national policy.

The NRPB quote one further study of elderly people in Boston, Massachusetts, to support their assertion that normal exposure of hands and face will provide sufficient vitamin D [36]. Boston is located at latitude 42°, the same as Rome, and so has a climate substantially different from the British Isles with much longer and hotter summers. Furthermore the research was conducted in 1990 when much lower levels of vitamin D were accepted as optimal. The level of vitamin D produced by exposure to the sun in the Boston study was compared with a standard minimum blood level of 37.5nmol/l.

The 37.5nmol/l level of vitamin D in the blood was established by giving elderly volunteers, who had minimal outdoor activity, a daily multivitamin supplement containing 400iu vitamin D. A supplement of 400iu/day is now known to be inadequate because it induces hyperparathyroidism, an indication that the blood level of D is insufficient. And a summer level of 37.5nmol/l will certainly lead to vitamin D insufficiency in winter [24]. So even in Boston, where the hours of sunlight are so much better and the summer is substantially longer, exposure of only hands and face does not produce a sufficiently high level of vitamin D for optimum health.

The assertion made in the NRPB report that we obtain enough vitamin D from everyday exposure of hands and face to the sun is little more than a convenient *post hoc* rationalisation and is not securely founded in scientific evidence. Reinhold Vieth, an internationally recognised expert on vitamin D, has calculated that short exposures of hands and face to the sun provides as little as 200-400iu of vitamin D per day during summer months and will only do that if enough sunlight is available. But he calculates that such limited exposure would leave more than half the population with insufficient vitamin D [37]. It is now recognised by international experts that levels of vitamin D below 40nmol/l are dangerously low [25]. Many people in Britain have a level that falls below this in both summer and winter making them vulnerable to a wide spectrum of disease.

#### Sunlight, season, and vitamin D in the British Isles

Maximum synthesis of pre-vitamin D in the skin occurs after exposure to the sun for about 10 minutes in the Tropics [38]. Since one side of the body is always in the shade, it takes at least 20 minutes to expose the whole body to the sun and obtain the maximum synthesis of pre-vitamin D in the skin leading to maximal production of vitamin D in the body. Vitamin D synthesis in skin reaches this maximum after 10 minutes because the reaction comes to a chemical equilibrium when pre-vitamin D begins to be converted into other compounds as quickly as it is synthesised.

Holick, a well-recognised world authority on vitamin D states: "Sunlight itself seems to be the most important factor in regulating the total cutaneous production of cholecalciferol [vitamin D]. This is the likely explanation for why there are no reported cases of vitamin D intoxication from chronic excessive exposure to sunlight."

Outside the Tropics this process will certainly take longer to reach equilibrium. Holick has shown that there is a substantial difference in the amount of vitamin D synthesised at midday in Boston, Mass, and Edmonton, Canada. Boston (42°N) is on a latitude similar to Rome while Edmonton (52°N) is on a latitude similar to southern England. In Boston some 50% more pre-vitamin D can be synthesised in a given time around midday compared with in Edmonton [38].

Any public health policy concerned with exposure to sunlight in the UK must take account of its northerly location at latitude  $51-61^{\circ}N - a$  band of latitude equivalent to the southern half of Hudson's Bay in Canada – and the special weather conditions peculiar to the British Isles. At these latitudes the UVB content of sunlight is too weak for six or seven months of the year to promote any vitamin D synthesis [38].

People in England, like those of Edmonton, obtain virtually no vitamin D from exposure to sunlight between October and March (inclusive) [25, 38]. In Scotland (latitude 55-61°N) the winter season when UVB is negligible lasts four to six weeks longer - two to three weeks longer at each end in April and September [39]. Since the half life of vitamin D in the body is some four to six weeks [37] people in the British Isles and other northern European countries are in special danger of becoming deficient in vitamin D during the winter and early spring [25].

Babies who are born in winter or spring are particularly at risk. This is because babies need to increase the amount of calcium in their bodies very substantially during the two or three months before birth and in the first four months of life as their bones grow rapidly at this time [12]. Good vitamin D levels are required for the optimum absorption of calcium. A mother's body stores of vitamin D, accumulated during the previous summer, are not generally replenished until April or May. So winter and spring born babies have an increased risk of suffering from diseases linked with vitamin D deficiency including multiple sclerosis, schizophrenia, diabetes type 1, and tooth decay (reviewed in some detail in Part 2).

Babies that are breast fed and obtain no exposure to the sun are particularly at risk of vitamin D deficiency because breast milk of women in northern Europe is generally deficient in vitamin D [12, 33]. Since sunlight is our principle source of vitamin D it is important for pregnant and nursing mothers to seek exposure to the sun. These facts need to be born in mind when recommending a public health policy on sun exposure for the UK.

Vieth and others have recommended a daily vitamin D input into the body (by sunlight or supplements) of around 2,000 iu for optimum health [37, 40, 41], although 1000iu is a more widely accepted figure. A white-skinned adult who exposes his or her near naked body to bright midday sun for 15-20 minutes in Boston will create the equivalent of about 10,000iu of vitamin D which provides a good supply of vitamin D, enough to last two or three days. But in England, even at midday in midsummer, it might take a white skinned person more like 40 minutes to make the same amount of vitamin D, while a person with dark skin might take three hours or more to make the same amount of vitamin D in the same conditions.

So in England in midsummer a white person may obtain all the vitamin D he needs in three 20 minute sessions of sunbathing on three separate days around midday over the period of a week. But a person with dark skin would have to spend up to six one hour sessions over a week to achieve the same result. It is not easy to obtain this amount of clear sunlight in the British Isles, even in midsummer, as the experiment by NRPB quoted above illustrates [34].

In April and September, at the beginning and end of the English summer, longer exposures are needed to obtain an optimum level of vitamin D. In Scotland longer exposures are likely to be needed in early May and late August as well to obtain the same levels as in England. For most efficient results sunbathing sessions should be conducted in the two hours around solar noon – that is between 11.00am and 3.00pm. At other times of day the ultra-violet component of sunlight is weaker and so a longer period must be spent in the sun [7].

In Denmark the value of the midday sun is recognised by the public who do not hesitate to sunbathe naked in parks around midday. Apart from nudity being unacceptable to many in the UK there are other practical difficulties in obtaining 20 to 60 minutes in the sun (depending on skin colour) in the middle of the working day. Many people cannot get into the open air at this time and if they can the weather may not be suitable. The unreliability of summer weather in the British Isles is an important factor that needs to be considered in recommendations for a UK sunlight policy.

Our maritime climate often produces substantial cloud cover even at the height of summer. Heavy cloud often completely obscures the sun preventing any ultra-violet light from reaching earth, but light cloud will also reduce the level of ultra-violet light reaching earth on a sunny day. These considerations need to be taken into account in making recommendations for healthy sun exposure in the UK. Poor weather at crucial times of year, during holiday periods and summer weekends, may have a major effect in lowering population levels of vitamin D, causing ill health, particularly during the following winter and spring when vitamin D levels are lowest.

So the best advice that can be given to people living in the British Isles is to be opportunistic in obtaining sun exposure and not miss any opportunity to remove clothes and expose as large an area of skin as possible to the sun while taking care not to bake or burn. At midday in midsummer when the sky is clear a white skinned person will obtain maximum vitamin D from an exposure of about 20 minutes while a person with dark skin will require up to an hour and a half. If exposure to the sun begins in spring when the sun is weak such times may be well tolerated by most people except those with the most sensitive skin type. If sunbathing begins in midsummer, or on holiday abroad, then exposures should be shorter at first and special care must be taken not to burn.

## Part 2: Vitamin D and chronic disease

Sceptics may find it difficult to believe that the many diseases listed below can all be influenced by deficiency of just one 'vitamin'. In fact vitamin D is different from many other vitamins. Although vitamin D has become known as a vitamin through its discovery in food, most of our supply comes from sunlight rather than food. Actually vitamin D is better described as a steroid hormone. Vitamin D is now known to have many effects other than those on bone and is synthesised at numerous sites throughout the body in many tissues and organs [15].

Although many diseases have now been linked to deficiency or insufficiency of vitamin D or sunlight it is not yet known why one person may suffer from one particular D-deficiency disease rather than another. The timing, duration and extent of the deficiency in vitamin D are all likely to be important and may be expected to act in conjunction with other well known risk factors such as particular diets, virus infections, heredity and so forth. The evidence suggests that while vitamin D is a major risk factor for these diseases it is often not a unique risk factor. That is presumably because a particular combination of risk factors together with timing in the cycle of growth determines which disease develops as a result of D deficiency.

Although the involvement of vitamin D deficiency or insufficiency in many different types of chronic disease is now well established the facts are not well known to epidemiologists or to those who make public health policy. For example, the Wanless Report, published in early 2004, makes no mention of vitamin D deficiency, despite considering the problem of falls in older people which have been directly linked to D deficiency (see section on Muscle Weakness below) [42]. Furthermore, fractures, the most common serious injury associated with falls, are increased by osteoporosis which is caused by vitamin D deficiency/insufficiency (see Osteoporosis below) and these are scarcely mentioned.

The report of the National Radiological Protection Board on Health Effects from Ultra-violet Radiation (published in 2002) also betrays a lack of interest or awareness of the wide range of effects of vitamin D deficiency on health. It devotes only one page to the non-skeletal benefits of vitamin D [7, 43]. And a specialist report on Vitamin D undertaken by the Food Standards Agency which appeared in draft form in 2002 [44] also paid remarkably little attention to non-skeletal benefits of vitamin D. After a critical appraisal in peer review the draft FSA report is now being extensively re-written. It is hoped that a new version of this report may eventually fill some of these gaps in the accumulation of officially recognised knowledge.

Several reviews of the link between vitamin D deficiency and chronic disease are now available [25, 45-51]. However all are selective in some way. Information is now accumulating at an accelerating rate and deficiency of vitamin D or sunlight has been found to be associated with too wide a range of diseases to be easily encompassed in a single review. This section aims to summarise the evidence linking vitamin D deficiency/insufficiency with chronic disease in a way that is most directly relevant to a policy on sunlight.

#### Nervous system disease

Birth in winter or spring, when vitamin D reserves are at their lowest, is associated with a number of neurological diseases which include not only schizophrenia and multiple sclerosis but bipolar disorder, autism, Alzheimer's disease and amyotrophic lateral sclerosis [52, 53]. These observations, which are well established, may now be explained by strong experimental evidence showing that female rats deprived of vitamin D give birth to offspring with damaged brains [54]. The young rats have longer brains with larger ventricles. Cell growth in the brains of these rats is altered with increased proliferation, reduced apoptosis, and reduced levels of neurotropin receptor and nerve growth factor. In a separate experiment addition of vitamin D to laboratory cultures of nerve cells was shown to increase growth of neurites and production of nerve growth factor (Brown *et al* 2003).

With the rat model now before us it is no longer surprising to find that several neurological diseases may be caused by damage to the growing nervous system as a result of deficiency of vitamin D during pregnancy or early life. Substantial evidence from other sources supports the suggestion that MS and schizophrenia may be caused by vitamin D deficiency. This is presented below, but it should be born in mind that D-deficiency may reasonably be suspected to be a cause (at least in part) of other chronic neurological diseases such as autism or Alzheimer's which are not reviewed here. The suggestion that vitamin D is a cause of these diseases does not of course preclude other risk factors causing these diseases independently.

#### Multiple sclerosis (MS)

It has been known for many years that MS occurs more frequently at high latitudes and is less frequent in sunny countries such as South Africa. However the first suggestion that vitamin D deficiency might explain these observations was made in 1997 [55]. Since then a case/control study in Australia has found that risk of MS is reduced by exposure of children to sunlight, especially in winter [56, 57]. Other evidence from the Nurses' Health Study has shown that total ingested vitamin D may significantly reduce the risk of MS [58].

In the UK a record linkage study of people with non-melanoma skin cancer, an indication of extended sun exposure, has found that they have 50% of the risk of developing MS compared with healthy controls [59]. Observation of MS lesions in the brain has shown that they increase during the winter when MS symptoms are also known to worsen [60]. Furthermore it has been found that vitamin D supplements will reduce symptoms of MS [61].

People in Britain, Sweden, Denmark or Canada who develop MS are more likely to have been born in spring, particularly May, and less likely to be born in November suggesting that low vitamin D levels in the last three months of pregnancy are important in development of this disease [62]. And so it seems wise to advise pregnant women to sunbathe whenever possible, while taking care not to burn, and to take vitamin D supplements in winter. Any advice that may discourage pregnant women from sunbathing, such as the current government campaign against skin cancer, may have unfortunate consequences for the health of babies in later years.

In the opinion of one very senior British epidemiologist, expressed at a private meeting, all the evidence points to MS being caused by a deficiency of vitamin D. He declared that what is needed now is a trial to see whether vitamin D may reduce progression of the disease and/or prevent it.

#### Schizophrenia

Much research into causes of schizophrenia during the last 50 years or more has concentrated on genetics, with the conclusion that heredity plays a major role in the disease and that environment has rather a small role to play. However the methodology used in these genetic studies of schizophrenia (for example, twin studies) has not for the most part distinguished between effects of the environment acting during pregnancy and effects of heredity itself. Generally these effects have been lumped together and considered to be the result of heredity. Evidence showing that environment may have an important role has generally been ignored. Many studies have found a small but significantly increased risk of people with schizophrenia being born in winter or spring which is compelling evidence for an effect of season on pregnancy in mothers of schizophrenic people [53].

Based on this finding and other clues from epidemiology John McGrath has proposed that low vitamin D during pregnancy or early infancy alters normal brain development leading to an increased risk of schizophrenia [63]. He subsequently found further evidence to support this hypothesis from studies of variation in sunlight from year to year and correlation of this with the numbers of schizophrenic people born in those years [64]. Further analysis has shown that the size of the winter/spring increments in births of schizophrenics has been found to vary systematically with latitude, with the risk of a schizophrenic being born in winter/spring being greater in northern latitudes [65].

The importance of regular exposure to sunlight for prevention of schizophrenia is emphasised by other findings. Living in an urban environment in youth has been found to be a risk factor for schizophrenia\* and urban residence has been associated with reduced exposure to UVB \*\*. A higher than expected incidence of schizophrenia has been found in London compared with smaller cities such as Bristol and Nottingham [66] which may possibly be explained by greater air pollution and less penetration of UV light in the capital.

Immigrants and their families in the UK have also been found to have a higher than expected incidence of schizophrenia which cannot be accounted for by social factors despite many years of research [67]. The suggestion that this high incidence is at least in part the result of low vitamin D levels caused by dark skin needs to be taken seriously. It may take as much as six times longer for dark skin to make the same amount of vitamin D as white skin.

The evidence suggesting that deficiency or insufficiency of vitamin D causes or is a cause of schizophrenia is now considerable but it is still insufficient to compel sceptics to acknowledge its strength. However, it is consistent with other studies, e.g. those of MS, showing that deficiency of vitamin D causes damage to the nervous system. And so it supports the suggestion that all city dwellers and particularly those with a darker skin should be encouraged to sunbathe safely and use vitamin D supplements. Pregnant women should be encouraged to sunbathe and thought must be given to ways of enabling babies and infants to obtain safe exposure to the sun.

\* Pedersen, C. B., Motersen, P. B., 2001 Evidence of a dose – response relationship between urbanicity during upbringing and schizophrenia risk. Arch Gen Psychiatry, **58**, pp 1039-46.

\*\* Nesby O'Dell, S., et al , 2002, Hypovitaminosis and determinants. Am J Clin Nutr, 76, pp 187-92.

#### **Diabetes type 1**

It has often been observed that diabetes type 1 (type 1 DM) tends to be more common in northern than in southern countries although there are exceptions that until recently left doubts about interpretation [68, 69]. Now it has been shown that in Australia, where people have a common genetic and cultural background but live over a wide range of latitude, type 1 DM is three times more common in the temperate south of the continent than it is the tropical north of the country [70].

In certain countries more children who later develop diabetes have been found to be born in spring and summer (March to June or July) than at other times of year. This was first found in Scotland, England and Wales [71-73] and later in Sweden [74], Slovenia [75], Slovak Republic [76], and Sardinia [77]. It has also been found for boys but not girls in the Netherlands [78], and for Jews but not Arabs living in Israel [79]. On the other hand fewer than expected diabetic children are generally born in these countries at the end of the summer and particularly in October [74] when vitamin D levels are highest. Although these observations on seasonal distribution of birthdays show a lot of variation and have not been found in a number of countries they are consistent with other evidence referred to here which suggests that vitamin D levels in pregnancy and early life are important for prevention of type 1 DM.

Deficiency of vitamin D in pregnancy or infancy has been found to be closely linked to diabetes type I in three separate studies [81-83]. In the Finnish study diabetes risk was reduced by 80% in infants who had received vitamin D supplementation in the first year of life compared with others who had no supplementation. In the sub-group of infants who had received vitamin D supplementation regularly, the risk was reduced a further 80% if the child had received at least the recommended 2000iu of vitamin D per day compared to those receiving lower doses.

Up to 1975, infants in Finland were supplemented with 2000iu vitamin D per day. In 1975 this was reduced to 1000iu, and in 1992 to 400iu. Since this reduction in dosage of vitamin D supplement began, the incidence of type 1 diabetes in Finland has been increasing, which is consistent with vitamin D insufficiency being a risk factor for diabetes type 1. Further evidence of the ability of vitamin D to modulate the disease comes from two independent trials which have shown that progress of diabetes type 1 can be delayed by supplementation with vitamin D is a major risk factor for insulin dependent diabetes [86].

The evidence suggesting that vitamin D deficiency is a cause of diabetes type 1 is strong and on the whole consistent. There is every category of scientific evidence that might be asked for although the trial evidence showing that vitamin D has a therapeutic effect is still at an early stage. The theory that viruses or other environmental risk factors [87] may cause diabetes type 1 remains popular. The evidence linking diabetes type 1 to vitamin D has not yet compelled attention from those who prefer to think in terms of these theories but it cannot continue to be overlooked.

The observations of seasonal births of people with diabetes type 1 reinforce conclusions from studies of MS and schizophrenia which suggest that vitamin D levels during pregnancy and early life are vitally important for preventing disease. Officially endorsed advice presently discourages mothers from allowing their babies and infants to be exposed to sunlight. Thought needs to be given now on how best mothers should be advised to expose babies and infants to the sun or give them supplements of vitamin D. Encouragement also needs to be given to pregnant women and nursing mothers to sunbathe safely, taking care not to burn.

#### **Diabetes type 2**

Pioneering work by Dr Barbara Boucher at St Bartholomew's and the Royal London Hospitals has established an important link between vitamin D deficiency or insufficiency and diabetes type 2 [88]. Reduced levels of vitamin D induce insulin resistance and reduce insulin secretion and so increase the risk of diabetes, heart disease and syndrome X, a complex condition combining diabetes, hypertension and heart disease [88]. A four to five fold higher prevalence of diabetes type 2 occurs in dark skinned Asian immigrants in the UK compared with UK whites suggesting that low vitamin D status increases the risk of diabetes type 2 [89]. Some studies, but not all, have found that administration of vitamin D improves blood glucose and insulin levels [25].

#### **Obesity and weight control**

Obesity and overweight are associated with an increased risk of diabetes (see above), heart attacks, high blood pressure, stroke, and some forms of cancer, as well as increasing mortality generally [90]. These diseases may be prevented and sometimes reversed or ameliorated by loss of weight. Sunlight stimulates the production of another hormone, alpha-melanocyte stimulating hormone (alphaMSH) [19], which plays a critical role in controlling

energy metabolism. The way in which alphaMSH acts on the brain to control the effects of insulin and leptin on appetite and body weight has been worked out in extensive animal experiments [21].

The animal work has been confirmed in experiments in which alphaMSH has been given to human subjects over a period of four weeks in a placebo controlled experiment [20]. Subjects given alphaMSH lost about a kilogram of body weight over the period and more than one and a half kilograms of body fat. This is a very significant change in weight but further work is needed to see whether such a loss might continue with further administration of alphaMSH, how long such a loss might be maintained, and whether regular sunbathing could initiate weight loss by introducing alphaMSH naturally.

Evidence suggesting that there is a significant link between weight and sunlight exposure comes from the fourth Tromso study [91] which found that low vitamin D was an independent predictor of obesity in both men and women. Vitamin D and alphaMSH levels are likely to be closely linked because both are determined by exposure to sunlight, and so the association between obesity and vitamin D could be determined by alphaMSH and/or by vitamin D itself. Other evidence has shown that vitamin D and calcium exert a coordinated control over fat metabolism [92].

People who are obese are often deficient in vitamin D because the vitamin is preferentially deposited in body fat and is not as readily available for general use in other parts of the body as it is in normal weight people [93]. This is a very interesting area of research but the work needs to be repeated and extended before it will be possible to know if it is of clinical importance.

#### High blood pressure (hypertension)

Raised blood pressure is associated with low levels of vitamin D in the body and with living in northern countries such as the UK. A disturbance of calcium metabolism lies behind essential hypertension, the common form of the disease. People with essential hypertension have decreased extracellular calcium yet intracellular calcium concentrations are increased [94]. Diastolic blood pressure is inversely associated with vitamin D levels in population groups [95] and prevalence of hypertension increases with increasing latitude [96, 97]. Afro-Americans have a higher prevalence of diastolic hypertension and lower vitamin D levels in blood than white Americans [13, 97].

Exposure to six weeks of thrice-weekly UV radiation, which increases serum vitamin D, has been shown to lower blood pressure [98]. Supplementation with 800iu vitamin D and 1200mg of calcium per day has also been found to significantly reduce blood pressure [99]. What is known of the action of vitamin D on intracellular calcium is consistent with understanding of the therapeutic action of calcium blocking drugs in reducing blood pressure [25].

The evidence linking raised blood pressure to low or deficient levels of vitamin D in the body is substantial. Other risk factors such as salt are well known to be important. However, it is likely to be easier to persuade people to sunbathe as a means of reducing their blood pressure than it is to persuade them to reduce the amount of salt in their diet.

#### **Muscle weakness**

People with low levels of vitamin D are subject to muscle weakness, swaying and difficulty in balance [29, 100, 101]. This makes them vulnerable to falls and increases the likelihood that bones that have been weakened by osteoporosis will be broken.

Muscle strength is influenced by vitamin D in two ways [100, 102]: 1) vitamin D initiates specific gene transcription in muscle cells by means of direct action on a nuclear receptor. This results in synthesis of proteins which are essential for maturation and function of muscle cells. 2) vitamin D acts by means of a membrane bound receptor to maintain intracellular calcium uptake and metabolism.

Muscle strength has been found to be correlated with blood vitamin D levels in old people [103]. Serum levels of vitamin D below 50nmol/l are associated with sway and below 30nmol/l with decreased muscle strength [29, 100]. Supplementation with vitamin D for 1 to 2 months has been shown to normalise muscle strength in patients with myopathy [104, 105] and supplementation with vitamin D plus calcium has been shown to reduce falls in the elderly [106, 107].

These proven effects of vitamin D in reducing falls and in improving health and well-being, particularly of the elderly, are likely to be a good investment. Encouraging people to sunbathe safely and take a vitamin D supplement costs very little and provides a substantial immediate gain in muscle strength and ability to survive.

#### Heart failure

Low vitamin D status contributes to heart failure, a disease caused by weakness of the cardiac muscle (see section above on 'Muscle weakness'). A biochemical indicator of severity of heart failure has been found to correlate with blood vitamin D levels in adults [108], and so it has been suggested that low vitamin D status may be a contributing factor in the pathogenesis of congestive heart failure in adults. Heart failure in two Afro-British infants has been found to be associated with vitamin D deficiency [109] and low parathyroid hormone, suggesting that D deficiency may contribute to heart weakness in infants. A number of other reports of heart failure in infants linked to D deficiency exist in the literature [109]. The weakness of heart muscle associated with insufficient vitamin D may readily be explained in the same way as general muscle weakness caused by vitamin D deficiency [25].

#### Cardiovascular disease

A case/control study of myocardial infarction (heart attack) in New Zealand has shown that MI patients have lower blood levels of vitamin D than controls at all times of year [110]. The authors concluded that their results supported the hypothesis that increased exposure to sunlight is protective against coronary heart disease. As Zittermann [25] has pointed out the nadir of vitamin D levels in winter in the UK [111] is paralleled by an increased cardiovascular morbidity [112].

Relatively small differences in the number of hours of sunlight between one place and another in the UK may make a substantial difference because we have a relatively low number of hours overall. An inverse correlation between hours of sunlight and death rate from coronary disease has been shown for four towns in Lancashire: Burnley, Blackburn, Preston and Blackpool. Blackpool has 27% more hours of sunshine per year than Burnley and 9% fewer deaths [113]. Indeed gardening outdoors in summer is sufficient in the UK to increase vitamin D levels and reduce serum cholesterol [113].

High mortality from heart disease occurs in winter in countries such as the UK [114]. This can be compared with alpine areas which are much colder in winter yet have a low incidence of deaths from heart disease at that time of year. This may be explained by the increased exposure to UV that occurs at altitude. Evidence that benefits of sunlight may be replaced by vitamin D obtained from the diet come from studies of Inuit (Eskimo). The low risk of heart disease in Inuit despite low UV levels in the Arctic [115] can be explained by their diet which consists almost exclusively of fish and marine meat such as seal which are rich in vitamin D.

These epidemiological observations are backed up by comprehensive biochemical and physiological studies and animal work. Zittermann points out that there is now increasing evidence that arteriosclerosis is a low-grade systemic inflammatory disease characterised by an increase in C-reactive protein which is regulated by the cytokines IL-6 and TNF-alpha [25, 116]. Calcitriol, the active metabolite of vitamin D, suppresses the secretion of these cytokines in a dose dependent manner [117]. Furthermore an inverse association between TNF-alpha and vitamin D levels in human subjects has been observed by Zittermann [108]. Vitamin D also appears to have a role in arterial calcification which occurs in 90% of coronary artery lesions [118].

#### Cancer

Some 16 different types of cancer have been linked with lower intensity of UVB radiation in a US ecological study, suggesting that the risk of these cancers is increased by vitamin D deficiency or insufficiency [119, 120]. The ecological approach taken in this work examines the relationship between disease and risk factors in populations rather than individuals and is sometimes criticised for being potentially misleading. However Grant's analysis referred to above correctly identified cancers that have been linked in many other studies with smoking, alcohol, or Hispanic heritage so lending support to the efficacy of the method. This provides confidence that the method is likely to be correct in identifying cancers that are associated with and possibly caused by inadequate exposure to UVB light or low vitamin D status.

The 16 cancers linked by Grant to UVB are: breast, colon, endometrial, esophageal, ovarian, Hodgkin's and non-Hodgkin's lymphoma, bladder, gallbladder, gastric, pancreatic, prostate, rectal, renal, testicular, and vulvar. For several of these cancers the findings are backed up by independent case/control and cohort studies which use a different methodology. These studies confirm that incidence or mortality of cancers of the breast, colon, ovary, and prostate are associated with intensity of sunlight in the area of residence, variation in exposure to sunlight as a result of occupation, or serum vitamin D levels [121-125]. A critical review of the literature on colorectal cancer has found that vitamin D obtained from natural or artificial sunlight or supplements is necessary for protection against colorectal cancer and vitamin D from the diet is not sufficient [126].

Women who have the highest exposure to sunlight or the highest levels of vitamin D have 20-50% lower

incidence of breast cancer compared with those who have the lowest exposure [127, 128]. In Norway people who are diagnosed with breast, bowel or prostate cancer in the autumn have a 15% lower risk of death compared with those diagnosed in winter [129]. The authors suggest that UV radiation might be used effectively as a supplement to cancer treatment.

Important evidence supporting the association between prostate cancer and sunlight comes from Dr Christopher Luscombe and colleagues at Keele University and North Staffordshire Hospital [130]. They found that sunbathing, regular foreign holidays, and sunburn in childhood are associated with a lower risk of prostate cancer when men with prostate cancer were compared in a case/control study with men who had benign prostatic hypertrophy. Men who were sunburnt as children were most strongly protected against prostate cancer. This does not of course suggest that sunburn itself is protective but rather that intense exposure to the sun is likely to be protective.

Substantial knowledge has been gained in the last decade of ways in which vitamin D acts to prevent initiation and growth of tumours. Vitamin D induces cell differentiation, increases cell apoptosis, reduces metastasis and proliferation, and reduces angiogenesis [131-135]. In addition, vitamin D down-regulates parathyroid hormone which has been linked to growth of cancer cells [136-138]. Much research effort has been put into finding patentable analogues of vitamin D that have these beneficial actions and can be used at high pharmacological doses without disrupting calcium metabolism.

The cost of cancer caused by vitamin D deficiency is immense. The total cost of cancer caused by insufficient sunlight and/or vitamin D in the USA, including time lost from work and care-providing, has been calculated as some \$16 billion [139]. Mortality from breast cancer in the UK is 50% higher than in the USA and non-lung cancer mortality as a whole is 38% higher, while the population of the UK is 22% of that of the USA. With these figures in mind it can be seen that the cost of cancer caused by insufficient sunlight and or vitamin D in the UK is likely to be of the order of several billion pounds sterling.

#### **Psoriasis**

Psoriasis is a common skin condition affecting about 2% of the population of the UK. People with the disease have thickened inflamed skin which is uncomfortable and embarrassing. They commonly suffer from arthritis as well. The condition improves dramatically with exposure of skin to natural or artificial UV light or treatment with creams containing chemical analogs of vitamin D [140, 141]. Greater exposure of the population as a whole to sunlight would be expected to reduce the incidence of this disease.

#### Infections

Vitamin D has a profound effect on the immune system and deficiency of vitamin D increases vulnerability to infection. Vitamin D deficiency increases the risk of tuberculosis [142, 143] and possibly increases the risk of shingles [144]. Shingles is the reactivation of infection with the varicella-zoster virus that causes chickenpox. The reactivation occurs generally in old people whose immunity has declined and who are often deficient in vitamin D.

Patients with tuberculosis have lower vitamin D levels in blood than control subjects [145, 146]. Asian immigrants to the UK have a high incidence of TB probably because they bring dormant infection with them. The dormant infection is then activated by deficiency of vitamin D caused by low exposure to sunlight in the UK compared with their country of origin and low absorption of UV by dark skin [25, 142].

More cases of tuberculosis are notified in late spring or early summer than at other times of year in the UK whereas other respiratory diseases occur most frequently in winter with a peak of incidence in December to March [147]. It appears that this seasonal pattern is largely the result of cases of TB among people who have migrated from the Asian subcontinent, who are particularly subject to vitamin D deficiency in the UK, whereas the occurrence of the disease among whites occurs more evenly throughout the year [148].

Mycobacterium tuberculosis, the bacterium that causes TB, is an intracellular pathogen that resides predominantly within white blood cells called macrophages. Macrophages possess an enzyme which enables them to change the inactive form of vitamin D3 into the active form. Vitamin D also induces the transformation of other white cells called monocytes into macrophages. It stimulates the production of lysosomal enzymes in macrophages so enhancing phagocytosis [25]. These activities may all be presumed to be important in the defence against TB and other infections.

Historically tuberculosis was treated with sunlight in sanitoria built for the purpose, for example in mountain areas where sunlight is more intense. The sanitoria had rooms arranged so that beds could be moved into the sun [149]. In 1897 Niels Finsen, a Copenhagen doctor, published work describing how tuberculosis of the skin could be cured by directly irradiating it with UV from a carbon-arc lamp which became known as the Finsen lamp. Finsen

obtained the Nobel prize for this work in 1903 and later it was shown that tuberculosis of the skin could be cured by treatment with vitamin D itself [150].

Regrettably, the importance of sunlight for prevention of tuberculosis now seeems to have been forgotten.

#### Polycystic ovary disease, menstrual problems and fertility

About one in five women of child bearing age suffer from polycystic ovary disease which causes abnormal periods, unwanted body hair and infertility\*. Seven of 14 women treated with vitamin D and calcium by Dr Susan Thys-Jacobs at St Lukes-Roosevelt Hospital, Columbia University, New York, recovered normal periods and two became pregnant [151]. Dr Thys-Jacobs has also found that women with pre-menstrual syndrome including migraines are likely to be deficient in vitamin D and to have reduced bone mass [152] putting them at risk of osteoporosis. She has shown that premenstrual and menstrual symptoms may be alleviated by calcium or calcium plus vitamin D [153-155].

Increased exposure to sunlight may be expected to improve vitamin D levels in women generally and so do much to prevent these conditions from developing. At the same time increased exposure to sunlight may be expected to reduce the call for infertility treatment which is now very common. Women who suffer these symptoms may be expected to improve with greater exposure to sunlight.

\* Solomon, C. G., 1999, *The epidemiology of polycystic ovary syndrome. Prevalence and associated disease risks*. Endocrinol Metabol Clini, North America. **28**, pp 247-63.

#### Crohn's and other inflammatory bowel diseases

People with Crohn's or other inflammatory bowel disease generally have low vitamin D, even before any treatment [156]. These diseases are more common in northern states of USA [157, 158]. Patients with these diseases often have osteoporosis as well. Poor absorption of fat, a common complication of inflammatory bowel disease, may make it difficult for people with these diseases to absorb vitamin D. Sunlight could be the best answer.

#### Bone disease: Rickets, osteomalacia, osteoporosis

Rickets, a serious bone disease of children caused by vitamin D deficiency, was thought to have been conquered in the 1920s when vitamin D was discovered. In the 1930s margarine began to be supplemented with vitamin D and rickets ceased to be common in its grosser forms. But rickets has since reappeared in the UK and north America, particularly among children with dark skins.

In the UK the problem is particularly serious in Asian children aged 8-14 [159]. Professors Brian Wharton and Nick Bishop, writing in *The Lancet* [12], have identified three major reasons for the reappearance of the disease. They are: 1) promotion of exclusive breastfeeding for long periods without vitamin D supplementation, 2) reduced opportunity for production of the vitamin in skin because of fear of skin cancer, 3) vitamin D deficiency in immigrants which occurs because dark skin takes longer to synthesise vitamin D and full clothing worn by some immigrant women blocks out sunlight.

Wharton and Bishop recognise that official advice to avoid strong sunlight can have serious adverse effects. They say: "advice on prevention of skin cancer should also ensure that mothers and infants receive adequate solar radiation in summer". Regrettably current advice on prevention of skin cancer in the UK fails to provide any useful advice on vitamin D and ignores the special needs of people with dark or black skin.

Low levels of vitamin D in the body are associated with low absorption of calcium and increased bone turnover leading on to osteoporosis or osteomalacia [160, 161]. Osteomalacia has emerged in the UK as a particular problem in Asian immigrants. It emerges in a florid form during pregnancy when the future health of the foetus as well as the mother is at risk [162]. But it has also emerged as a problem among elderly women in Scotland [163]. The decrease in vitamin D in the blood that occurs during winter can lead to a transient loss of bone density in women [164]. This shows that advice to reduce sun exposure in summer could also lead to reduction in bone density in women whose vitamin D levels are marginal. More hip fractures occur in the US population in winter than in summer in both sexes [161]. Patients with hip fractures more often have blood levels of vitamin D lower than those of controls [165, 166].

The bone mineral content of children shortly after birth and nine years later has been shown to be related to blood levels of vitamin D during late pregnancy [167, 168]. Dr Jonathan Tobias of Bristol University and Dr Cyrus Cooper of Southampton University have argued on the basis of such evidence that bone development of adults is programmed by early life factors which are influenced by sunlight exposure and use of vitamin D supplements in pregnancy and early life [169].

Several studies have found that supplementation of adults with vitamin D increases bone mineral density but improvement may not occur if there is not enough calcium in the diet [25]. Two randomised controlled trials have shown that supplementation with either 800iu vitamin D and 1200mg calcium per day, or one capsule of 100,000iu vitamin D every four months for five years, will prevent fractures [170, 171]. The improvement shown in these trials may be the result not just of strengthening bones but of reduction in muscle weakness and body sway which are a contributory cause of falls (see section on muscle weakness, p17).

Osteoporosis cannot be dismissed as being in any sense a relatively trivial disease. It has a high morbidity and mortality. Some 40% of women and 13% of men suffer fractures of the spine, hip or wrist that may be largely attributed to osteoporosis. Between 10 and 20% of people die within six months of a hip fracture and 50% of those who have suffered a hip fracture are unable to walk again without assistance. The annual cost of osteoporosis in the USA has been estimated to be \$5-10 billion with a similar pro rata cost in other developed countries [172]. In the UK the cost of hip fractures alone has been put at £1.7 billion per year [173].

There is no scientific doubt that vitamin D deficiency is the cause of these bone diseases. The benefit from vitamin D in enabling growth of healthy bones is enough in itself to favour a public health policy that recommends safe sunbathing.

An eminent panel of doctors and scientists concerned with bone disease called for a review of current UK public health programmes on sunlight as long ago as 1998. Their report, Nutrition and Bone Health, [39] recommended that "the public health consequences of sunlight exposure should be reviewed to take account of both its beneficial and its adverse effects with a view to developing guidelines. The effect on vitamin D status of measures taken to reduce the risk of skin cancer, such as encouraging covering up with clothes and applying cosmetic creams which seek to prevent the UVR reaching the skin should be clarified."

This recommendation was never taken up and is now more urgent than ever.

#### Dental decay

The teeth are made from deposits of a dense calcium containing material. Since vitamin D is central to the body's control of calcium absorption and metabolism, it is not surprising to find that deficiency of vitamin D and sunlight have a role in dental decay (caries). The formation of dentine (the solid material in teeth) begins in the last two months of pregnancy and continues until eight or nine years of age, while the wisdom teeth (third molars) continue to form during the following 10 years [12]. So exposure to sunlight during pregnancy and throughout childhood, together with vitamin D in the diet or in supplements, may be expected to be important for the formation of strong teeth.

The teeth are completed with the laying down of enamel, an insoluble calcium phosphate compound. Delayed eruption of teeth and hypoplasia (faulty development) of the dental enamel are recognised signs of rickets [12]. Supplementation of mothers with vitamin D during pregnancy has been shown to prevent hypoplasia of the enamel in infants which may occur without other symptoms of rickets [174, 175]. Hypoplasia of the dental enamel has been found to occur more frequently in low birthweight babies born in winter or early spring months [176] which is consistent with sunlight and vitamin D being important for sound development of teeth.

Enamel hypoplasia allows carious lesions to become established more easily making people vulnerable to other dietary factors which increase the risk of caries. The importance of calcium in the diet, vitamin D and sunlight for good development of teeth appears to have been generally overlooked. Emphasis has been given in public health campaigns to the effects of diet, fluoride, and tooth brushing in preventing tooth decay. Sunlight and vitamin D supplements are also important and need to be recognised alongside these well known risk factors.

The relationship between sunlight and prevalence of tooth decay was first established as early as 1939 in a study of data obtained by the US Public Health Service. A very clear relationship was shown between prevalence of tooth decay and hours of sunshine in an analysis of the teeth of white boys living in communities with less than 5,000 people [177]. Boys living in the sunny southern states had an average of about three cavities each compared with five cavities each for boys living in the least sunny northern states.

Evidence for the importance of sunlight for healthy teeth in Britain comes from studies made by the British Association for the Study of Community Dentistry in the early 1990s [178]. These studies found a consistent north/south variation in the prevalence of caries with more caries always in children from Scotland, the Northwest, Wales and Mersey regions – areas with less average sunlight. The proportion of 12-year-old children with untreated dentinal caries was three times greater in Scotland than in the South West Thames region.

The importance of vitamin D in preventing tooth loss in old people has been shown in a randomised, placebo controlled trial [179]. Supplements of vitamin D (700iu) and calcium (500mg) were given to 145 people over 65 years of age. Those taking supplements were found to have lost half as many teeth as those taking placebo when assessed

18 months after the start of the trial. It appears that deficiency of vitamin D allows softening of the bone in the jaw and loosening and consequent loss of teeth.

There can be little doubt that an adequate level of vitamin D is necessary for strong healthy teeth. This needs to be taken into account in making public health policy.

#### Conclusion: vitamin D deficiency/insufficiency and chronic disease

The evidence reviewed in this section shows a strong association between vitamin D deficiency/insufficiency and a wide spectrum of disease. It is suggested that these diseases are caused at least in part by D deficiency/insufficiency. The link between vitamin D and these diseases is new and naturally arouses scepticism among those not familiar with the evidence. Nevertheless it seems extraordinary that such an important cause of so much disease could have been overlooked for so long.

The link between vitamin D and disease is most obvious in the seasonal occurrence of certain diseases. However, until now seasonal virus infections have generally been preferred as an explanation of seasonal disease. This is because until recently the important role of vitamin D in controlling development and metabolism of 30 or more body tissues [180] was not understood and still remains largely overlooked outside a small circle of expertise. While north/south gradients occur for many of the diseases discussed here exceptions (which can often be explained) have prevented easy acceptance of explanations based on vitamin D deficiency. And so the link between D deficiency and a wide spectrum of disease has remained largely unrecognised until recently.

However, recent advances in biochemistry make it no longer surprising to find so many diseases linked to vitamin D insufficiency. Vitamin D not only controls absorption of calcium in the bowel but also controls access of calcium to cells and regulates the action of a number of genes [25].

We cannot always explain why one individual suffers from one particular D deficiency/insuffiency disease while another does not, although answers may be within sight for some diseases. In the case of breast cancer, for example, a diet high in calories (often leading to overweight), together with little exercise, encourages proliferation of cells many of which may be poorly differentiated. Vitamin D promotes differentiation of cells and apoptosis of redundant cell lines. And so the combination of high calorie diet, relatively little exercise and insufficient vitamin D increases the risk of redundant cell lines persisting in the breast where they may accumulate mutations that transform them into cancer cells.

The risk of these diseases, as in the example of breast cancer, may be reduced by increasing individual or population levels of vitamin D. However other risk factors will continue to operate and so increasing vitamin D, while reducing the risk, cannot be expected to eliminate these diseases. Best protection against disease will be obtained by attention to diet and exercise as well as increasing vitamin D intake.

Increase in vitamin D levels may be achieved by sunbathing in summer and/or taking a vitamin D supplement. The oral intake of vitamin D needed to maintain levels circulating in the blood in the absence of sun exposure may be as high as 4,000iu/day [181]. Only a very small minority of people who eat oily fish everyday are likely to come anywhere near such a high level of vitamin D without regular exposure to the sun.

UK government experts advise that vitamin D taken in the form of a supplement should not exceed 1000iu per day [182]. Very little is obtained in the average diet and so the remaining intake that is needed to make 4,000iu or thereabouts must be obtained by exposure to the sun. This is only likely to be possible in the UK if every opportunity is taken to expose as much of the body as practicable to the sun in the middle of the day while taking care not to burn (see detailed discussion in Part 1).

## Part 3: Risks and benefits of sunlight

#### Melanoma

Melanoma is the most serious form of skin cancer. There are some 7,000 cases a year in the UK and about 1750 deaths. Other types of skin cancer cause only a few hundred deaths annually, making altogether around 2,000 deaths annually from skin cancer in the UK. Melanoma is some six times more common in northern tropical parts of Australia than in the colder southern parts [183]. These and similar observations have given rise to the widely held belief that melanoma is caused by sunlight.

However, there are real doubts about the way in which sun exposure causes melanoma [17]. Adults who work outdoors and children who play outdoors are regularly exposed to the sun and are less likely to develop melanoma than those who work or play more indoors [184, 185]. While people who have irregular exposure to the sun and those who recall being sunburnt have a higher risk of melanoma, especially if they have a fair skin type [186]. Occasional exposure of skin to sunlight appears to carry the greatest risk of melanoma, while regular exposure of skin to sunlight appears to protect against melanoma, probably because it provides higher levels of vitamin D which are protective against cancer in general.

Furthermore, melanomas occur most commonly on the backs of men and the upper legs of women, areas which do not get so much exposure to the sun as face or hands [187]. In people under 50 melanoma is most frequent on sites which are exposed irregularly to the sun [188]. In black people melanomas occur predominantly on the lower legs and commonly on the sole of the foot, an area which gets virtually no sun at all [189]. This evidence shows that the relationship between melanoma and sunlight is not simple.

It is widely accepted that as many as two-thirds of melanomas are caused by excessive exposure to the sun [190]. However, other methods of analysis challenge this figure. A person who has had melanoma may develop a second primary melanoma which occurs completely independently of the first tumour. Analysis of data on second primary melanomas has enabled the importance of risk factors such as sun exposure, skin and eye colour, and skin type to be calculated. Using this method it has been found that these known risk factors account for only about 23% of variation of melanoma risk [191]. Since skin type is a very important variable it leaves sun exposure accounting for perhaps 10-15% of the overall risk of melanoma according to this method.

Other risk factors that increase the risk of melanoma include increased body weight (obesity), lack of exercise [192], and diet [193]. Indeed the steady increase in incidence of melanoma over the last 10 to 20 years (24% increase in the last five years) parallels the increase in other cancers such as breast, colorectal, prostate, testis, leukaemia and lymphoma [194].

The increase in obesity and decrease in regular exercise in the UK over this period may account for the increase in melanoma [195, 196]. Much of this epidemic of obesity appears to be the result of increased consumption of fast foods and snacks with a high energy density [197] and these should be identified as a likely cause of melanoma.

These considerations are enough to explain why the SunSmart programme has not been successful in reducing deaths from melanoma in the UK where the average intensity of sunlight is much less than in Australia. Indeed it is possible that reduction of exposure to the sun in the UK actually increases the incidence of melanoma rather than decreases it, and that regular careful exposure of skin to the sun in the UK would actually reduce the incidence of melanoma. The evidence certainly does not provide adequate support for a policy favouring reduction of sun exposure.

The risk of a person suffering from melanoma is about 10 in 100,000 which is described as 'very low' in Professor Sir Kenneth Calman's 'language of risk' [17]. Only part of this very low risk, perhaps 10-15%, may be attributable to sunlight. So the risk of contracting melanoma as a result of exposure to sunlight could be as low as one in a 100,000. So even the most forceful campaign advocating sun avoidance could be expected to prevent only a few hundred deaths [198].

For practical purposes the risk of death from any kind of skin cancer caused by exposure to sunlight is negligible when compared with the high risk of other diseases of many different types which are caused, at least in part, by D deficiency.

#### Non-melanoma skin cancer

More than 60,000 cases of non-melanoma skin cancer occur every year in the UK. In the vast majority of cases the lesions are removed without problems as a simple out-patient procedure.

But problems associated with these cancers should not be underestimated. These cancers cause a few hundred deaths each year in the UK. In a relatively small proportion of people the lesions are in an awkward position that requires delicate surgery or the lesion may be extensive and require a more difficult procedure. Some of these cancers require surgery which leaves a disfiguring scar or causes disfiguring removal of tissue.

Basal cell carcinoma, which generally grows quite slowly, is the most common type of non-melanoma skin cancer. Squamous cell carcinoma which can spread to other parts of the body if untreated is the second commonest type. Both types occur most commonly in old people. Regular exposure to sunlight during work outdoors is a risk factor for squamous cell carcinoma but probably not for basal cell carcinoma. While squamous cell carcinoma is clearly caused by sun exposure the relationship between basal cell carcinoma and sunlight, like that of melanoma, is more complicated.

Basal cell carcinoma had an incidence of 114 per 100,000 population in South Wales in 1998 compared with 726 per 100,000 in Australia, suggesting an association with sunlight. In the United States it has recently increased in incidence at a rate of 10% a year. Exposure to sunlight is widely accepted to be a cause of basal cell carcinoma but it does not explain why particular people get these tumours and others do not, or the fact that these tumours often occur on the body in clusters, and are found mainly on the trunk rather than on areas such as the head that are exposed for longer periods [199].

Diet appears to have an important effect on susceptibility to skin cancer and actinic keratosis, a form of skin aging [200, 201] that may lead on to squamous cell carcinoma. A trial at Baylor College of Medicine in Houston, Texas, has shown that a low fat diet can reduce recurrence of skin cancer and actinic keratosis over a period of two years. Patients who had suffered skin cancer (basal cell or squamous cell carcinoma) were randomised to one of two groups at the beginning of the study. One group continued with their normal diet which contained 36% fat. The second group were given dietary advice and reduced fat to under 21%, while also losing 2-4kgs of body weight. The number of skin cancers in the diet group declined from eight to one over 16 months of the study compared with a steady six in the first eight months and six in the second eight months for the control group. The reduced risk of skin cancer (both basal cell and squamous cell) in the diet group may be the result of loss of body weight or the change to a low fat diet.

Other types of study have produced equivocal results. The view that excess energy consumption is a cause of basal cell carcinoma is supported by a cohort study of 73,366 women in the Nurses' Health Study [202]. But the Health Professionals Follow-up Study of some 43,000 men has failed to confirm a link between basal cell cancer and fat consumption [203].

Nevertheless the randomised studies suggest that there is an effect of diet on recurrence of skin cancer, and that much more is involved in the initiation of these cancers than simply exposure to the sun. It may be that only certain people who have a relatively rich diet, those who are relatively overweight, or, have a high calorie consumption compared with energy output, are at high risk of developing these skin cancers. More research is needed in this area but it is a mistake to assume that sunlight is necessarily the most important risk factor for these two cancers.

#### Risks to children and young people

Current skin cancer prevention programmes warn of a special risk to children and young people from exposure to the sun. Official literature asserts that exposure to the sun in childhood may disproportionately increase the risk of skin cancer many years later [204, 205]. In fact this idea is controversial and has been challenged by several authors.

Whiteman *et al* [206] undertook a systematic review of the literature and found that the way in which sun exposure was measured made a striking difference to the association between melanoma and age at which exposure occurred. Case/control studies produced no consistent associations between melanoma and childhood sun exposure. On the other hand ecological studies (which measure sunlight exposure of geographical areas rather than of individuals) did show a relationship between early exposure and melanoma risk. However it is unwise to come to firm conclusions when these two types of study produce widely differing results.

A recent study of 603 melanoma cases and 627 controls in seven European countries concludes that there is no evidence for a critical period of high susceptibility in childhood when solar radiation is more likely to induce melanoma [207]. The study concluded that more than five different sunburns doubled the risk of

melanoma regardless of their timing in life. Another study has found that outdoor activities in childhood are associated with a lower risk of melanoma [185].

Basal cell carcinoma has also been reported to be more common after sunburn in childhood [208, 209] but the research findings are not clear. One study found that living in a region of high solar radiation in childhood does not increase risk of basal cell carcinoma whereas living in such a region as an adult does increase the risk [210]. This study found that risk of basal cell carcinoma was proportional to lifetime accumulation of blistering sunburns [210]. Other studies have found no clear link between sunburn in adulthood and basal cell carcinoma [209]. On the other hand an Italian study found that an average summer holiday exposure of eight weeks per year throughout childhood increased the risk of basal cell carcinoma almost fivefold [208]. The research results are conflicting and so it is by no means certain that childhood and adolescence are critical periods for this cancer.

#### A skin cancer policy for children and young people

Summarising the scientific evidence reviewed above: sunburn or sunlight exposure in childhood may possibly increase the risk of basal cell carcinoma, the commonest form of skin cancer, but this is not firmly established. On the other hand sunburn/sunlight exposure in childhood does not seem to increase the risk of the most serious form of skin cancer, melanoma. Relevant evidence appears to be lacking for the third type of skin cancer, squamous cell carcinoma.

Children should obviously be protected against sunburn but they also need exposure to the sun so that they can synthesise vitamin D. This may mean accepting an uncertain risk of causing basal cell carcinoma in later life. The vast majority of basal cell carcinomas are readily treated and so the risk of serious consequences is small. This small, and possibly non-existent, risk may be further reduced by avoiding sunburn.

In conclusion, children can safely be allowed to run about in strong sun wearing brief clothing without suncreams for limited periods of time, so long as care is taken to avoid burning. This will enable children to benefit from vitamin D production in the skin. Suncreams cannot be relied upon to prevent cancer (see discussion in Part 4, section 8), so burning is best avoided by encouraging children to seek the shade after a suitable time in the sun. Time that may safely be spent in the sun depends upon skin type, previous exposure to the sun, time of day, season (early, middle or late summer), latitude, and whether or not the sky is at all overcast. Suncreams can be used when extended exposure cannot easily be avoided e.g. when playing sports.

#### Skin wrinkling and aging

Warnings that exposure to the sun may cause wrinkling and aging of skin are frequently made at the same time as warnings about skin cancer. Although sunlight can cause wrinkling this does not seem to be common in the UK.

Studies in Japan have found that the average 40-year-old woman from Kagoshima (32°N) in the south of the country has facial wrinkling equivalent to that of a 48-year-old woman living further north in Akita (40°N) [211], suggesting that sunlight induces wrinkles. However, a study of 792 people over 60 in South Glamorgan, UK, found no association between sun exposure and wrinkling of skin on the face, neck or back of the hand [212]. This is probably because the average person in Glamorgan gets relatively little intense exposure to the sun compared with people in Japan. Glamorgan is located at latitude 51°N, a great deal further north than either of the Japanese locations.

On the other hand, daily cigarette smoking has been found to be closely associated with the development of wrinkles in people in Glamorgan, as in other parts of the world. Smoking 20 cigarettes a day in Glamorgan increased wrinkles sufficient to give a person the appearance of someone 10 years older. Strangely, people with wrinkles have been found to be less likely to develop basal cell carcinoma, one of the common types of skin cancer, showing that other factors, and not just sunlight, must be involved in these skin changes [213]. Another study has found that a diet with a high intake of vegetables, legumes (beans and peas), olive oil, apples, prunes or tea is associated with fewer wrinkles [214].

In summary, sun exposure is only one factor influencing wrinkling of skin and not necessarily the most important one. In the UK sunlight does not seem to be a significant cause of wrinkling for most people. Nevertheless regular sunbathing in the UK could cause wrinkling. A healthy 'five a day' fruit and vegetable diet recommended for prevention of cancer and heart disease may reduce or prevent wrinkling.

While wrinkling is obviously undesirable it seems a small, perhaps even insignificant risk to take, in return for the benefits of increased vitamin D levels that follow from sunbathing. Anyone choosing to avoid sun exposure for fear of wrinkling should take a vitamin D supplement all year round.

#### Cost of disease caused by vitamin D deficiency

The large number of chronic diseases caused at least in part by vitamin D deficiency make a formal estimate of the total cost very difficult to make. Nevertheless the cost of vitamin D deficiency diseases in the UK or USA has been put at billions of pounds or dollars per year [215].

There is no doubt that the cost of disease caused by D deficiency is much greater than the cost of disease caused by excessive exposure to sunlight. This is clear from the fact that the 2,000 deaths per year from skin cancer in the UK are a tenth of the deaths from other types of cancer that are attributable to D-deficiency [139, 216]. Sunlight is our primary source of vitamin D. So it must be concluded that any public health policy regarding sunlight should favour exposure to sunlight rather than avoidance of it. This conclusion is reinforced when it is considered that a substantial proportion of skin cancer deaths are not caused by sunlight and that many other chronic diseases apart from cancer are caused at least in part by D deficiency.

The two examples below illustrate the immense cost of disease caused by D deficiency in the UK.

#### **Cost: Diabetes**

Approximately 1.3 million people in England have been diagnosed as having diabetes and about 85% of these have diabetes type 2. Another one million people probably have undiagnosed diabetes type 2 which must seriously affect their lives and reduce their capacity to work. The total cost to the National Health Service of diabetes types 1 and 2 is £1.3 billion annually but the cost to society as a whole is much larger.

These facts about the incidence and costs of diabetes are given as an example in the government's consultation exercise on improving people's health [217]. According to this government analysis, obesity and physical inactivity together cause some 60% of diabetes type 2. Onset of the disease can be delayed and possibly prevented by modifying lifestyle to reduce obesity and physical inactivity. Vitamin D deficiency is another risk factor which may delay and prevent onset of diabetes 1 or 2 although it is not considered in the government analysis.

The financial saving that could be obtained in preventing or ameliorating diabetes type 2 by increased exposure to sunlight or increased consumption of vitamin D supplements can not be determined at present. But advice to increase exposure to the sun can be expected to be popular, easy to implement at little expense, and have multiple health benefits.

Some 200,000 people in the UK suffer from diabetes type 1 which probably costs about £500 million a year to treat – more per head than type 2 diabetes because people with type 1 always require insulin, live longer with the disease, and require more treatment of all kinds [218]. It is possible that the disease might be largely prevented if mothers and babies had higher vitamin D levels. Advice to pregnant and nursing mothers to sunbathe and take supplements might thus make a substantial improvement in the nation's health.

Vitamin D supplements are not expensive and are easily distributed through mother and baby clinics [10]. How much illness might be prevented and how much money saved will depend on efficiency in distributing the supplements and in communicating the advice. It will also depend on the efficacy of the preventive measures. At present none of these are known and so an estimate of savings cannot be made although potentially they are very substantial.

#### Cost: Falls

Falls are a major cause of disability and mortality in the UK. One third to one half of people aged over 65 fall each year resulting in more than 400,000 attendances at accident and emergency departments [42]. Hip fractures alone cost £1.7 billion per annum in England [173]. Each year some 14,000 people die as a direct result of hip fracture and 50 per cent of those who suffer hip fracture can no longer live independently afterwards. The cost of dealing with all fractures and other consequences of falls in the whole of the UK must exceed £2 billion.

Government reports have looked for urgent solutions to the problem [42, 219] but the role of vitamin D in causing muscle weakness leading to falls has not been considered as a cause of the problem despite being well recognised in the scientific medical literature. The Department of Health report, '*Preventing Accidental Injury – Priorities for Action*,' refers to the need for prevention and treatment of osteoporosis but makes no reference to vitamin D deficiency as a cause of falls. A substantial reduction in falls and consequent fractures could be expected if vitamin D and calcium supplements were provided to old people and careful sunbathing was encouraged. Immediate savings on treatment of falls cannot be determined from presently available information but could run to many millions of pounds annually.

## Part 4: Review of public health policy on sunlight in the UK

#### The SunSmart campaign

SunSmart is described as the UK's skin cancer awareness campaign but it was originally designed for Australia [5] which has considerably greater intensity of sunlight than the UK and a much higher incidence of skin cancer. The government's NHS sun-safety programme '*Sun Safety for Children*' [220, 221] seems to be based on the same precepts.

A comparison of sun exposure in England and Australia [222] using UV-sensitive film badges found that Australian children in Queensland were exposed on average to twice as much sun as English children. The English children rarely received enough sun to cause any reddening of the skin whereas children in Queensland received enough sunlight to cause reddening in one out of three days. These observations suggest that a sunlight programme designed for Australia is likely to be unsuitable as a model for the UK. They also show that reddening of the skin, a preliminary to sunburn, is relatively rare in English children in England.

A comparison of latitudes of Australia and the UK also shows that a direct transfer of sun policy from Australia to the UK cannot be wise. London has a latitude of 51°N, Birmingham 52°N and Aberdeen 57°N whereas Sydney lies at 34°S and Melbourne at 35°S while Brisbane lies just outside the Tropic of Capricorn on the edge of the Coral sea. Furthermore the southern hemisphere is closer to the sun during the summer than is the northern hemisphere during its summer. And so most of Australia has a climate similar to North Africa. Only Tasmania has a climate that might be called temperate and approaching that of Europe.

Key quotes from the SunSmart UK website [223] are shown in the box below. These quotes are followed with an item by item criticism of the SunSmart message. References supporting comments made here will be found in previous sections of this document:

"Stay in the shade 11-3pm - the sun is most dangerous in the middle of the day – find shade under umbrellas, trees, canopies, indoors"

"Always cover up – sunscreen is not enough – wear a t-shirt, wide brimmed hat and wraparound sunglasses (eyes get sun-damaged too)"

> "Remember to take extra care with children – young skin is delicate – keep babies out of the sun completely"

"Then use factor 15+ suncream – apply sunscreen 15-30 minutes before you go outside – it does not work immediately"

Quotes from the SunSmart UK website

#### 1) 'Covering up' risks D deficiency

The SunSmart campaign advises covering up with 15+ factor sunscreen, wearing a shirt, wide brimmed hat and seeking the shade. Most people in the British Isles have low or marginal levels of vitamin D in summer and low or deficient levels of vitamin D in winter. Telling people to cover up and, in effect, avoid the sun can only make their level of vitamin D lower and increase the risk of cancer and other diseases. Anybody implementing these draconian instructions in the UK puts themselves at serious risk of vitamin D deficiency and consequent disease.

#### 2) Regular sunbathing needed for optimum health

The SunSmart campaign fails to explain that regular sun exposure is needed in summer in the British Isles so that vitamin D stores may be replenished after the winter. Vitamin D levels in people living in the British Isles are generally low and sun exposure is of vital importance to maintain these levels. Some 80-90% of our vitamin D comes from the sun.

Exposure of the naked body to the sun for 20 minutes (10 minutes each side) at midday in midsummer in the UK will provide maximum vitamin D synthesis. Less time should be spent in the sun if there is any baking or burning sensation. People with very fair skins may be able to tolerate only five minutes (or less on first exposure) in the

midday sun in the UK. Nevertheless such brief exposure is important for them and will generate a substantial amount of vitamin D. Under optimum conditions longer than 20 minutes exposure will not produce any more vitamin D.

In fact optimum conditions for vitamin D synthesis are seldom achieved in the UK because of cloud cover. To obtain good exposure of the skin to UV light and obtain good vitamin D synthesis (while taking care to avoid any baking or burning sensation) longer than half an hour will often need to be spent in the sun in the UK. These facts should be addressed in a public health programme concerned with sunlight.

#### 3) If cover-up is unavoidable take a D supplement

The SunSmart campaign does not make it clear that avoidance of exposure to the sun may have serious health consequences if a vitamin D supplement is not taken. Some individuals who have an increased risk of melanoma because they have an unusually high number of melanocytic naevi (moles) on their skin may be given such advice by a doctor [224]. Advice to curtail sun exposure should always be accompanied by additional advice to take an appropriate dose of vitamin D (at least 1000iu/day) year round.

Individuals with fair skin and a tendency to sunburn may also be at increased risk of melanoma [225] but the reasons for this are not clear. Fair-skinned people often avoid the sun and so may be more prone to vitamin D deficiency and to cancer. It is not clear at present that they should necessarily be advised to avoid the sun rather than to be specially careful to avoid burning. But any advice to avoid or seriously curtail sun exposure should be accompanied by advice to take a vitamin D supplement year round.

#### 4) Englishmen should go out in the midday sun.

In the words of the Noel Coward song: "Mad dogs and Englishmen go out in the midday sun". There is a reason for this madness. The English sun, even at midday, is often rather weak. Yet the SunSmart campaign advises: "Stay in the shade between 11am and 3pm – the sun is most dangerous in the middle of the day".

It is true that the sun will take a shorter time to inflict a burn between 11am and 3pm (two hours each side of solar noon) but this is also the time when exposure of the skin to the sun produces most vitamin D. Exposure only at 'off-peak' times may not provide enough vitamin D in the UK. Responsible advice should make it clear that the skin may be exposed to advantage for short periods in the middle of the day to obtain good synthesis of vitamin D while care must always be taken not to bake or burn. Extra care must be taken if sunbathing around midday in midsummer in England when the sky is clear of cloud, and special care must also be taken when abroad in more southerly countries. The lunch break, around midday, is a time when many people are able to sunbathe for a short time and it is folly to discourage this.

#### 5) Autumn and spring in the British Isles

Synthesis of vitamin D in autumn and spring are particularly important to prevent vitamin D in the body remaining at a critically low level for a long winter period. Several diseases including schizophrenia, multiple sclerosis, and diabetes type 1 have been linked with deficiency of vitamin D in babies born in winter or spring (see Part 2). This should be taken into consideration in a responsible sun for health policy.

In England sunlight (measured as UV irradiance) is relatively weak in early spring (March and April) and autumn (September and October) [39]. In Scotland (and probably the north of England although figures are not available) sunlight is relatively weak in every month except June [39]. At these times of year the best, or only, time of day to obtain good exposure of skin to sun and satisfactory synthesis of vitamin D is midday. The instruction given by SunSmart to avoid exposure of the skin at midday fails to take into consideration seasonal changes in intensity of sunlight at midday.

#### 6) People with dark skins

The SunSmart campaign provides no information for people with darker skins, probably because it is based on the Australian programme and there are few people in Australia with really dark skins other than aborigines. People with dark skins need to spend up to six times longer in the sun to obtain the same amount of vitamin D as a white person [226]. People with dark skins living in the UK need to take every opportunity possible to expose their skin to the sun if they are to obtain a reasonable level of vitamin D. They should be advised to do this while taking care not to bake or burn. The immigrant population of Britain is at greater risk of diseases such as rickets, schizophrenia, diabetes and multiple sclerosis which are caused at least in part by D deficiency and this should be taken into account in any public health programme.

#### 7) Babies need sunlight or vitamin D supplements

The SunSmart campaign advises: "Young skin is delicate – keep babies out of the sun completely". This advice is likely to cause serious D-deficiency or insufficiency in breast fed babies because breast milk generally contains little vitamin D and few mothers give their babies vitamin D supplements. Bottle milk is supplemented with vitamin D and so bottle fed babies may probably be safely kept out of the sun. Complete avoidance of the sun, as recommended by SunSmart, is also likely to cause serious D-deficiency in toddlers, often called babies in common parlance, unless they continue to drink a substantial quantity of artificial milk supplemented with vitamin D. D-deficiency in early life is now being linked to several chronic diseases which may emerge in subsequent years including schizophrenia, multiple sclerosis and diabetes type 1 (see above). Urgent consideration needs to be given to finding the best advice for mothers and babies.

#### 8) Sunscreens and SunSmart

SunSmart recommends covering up with sunscreen as a normal routine. But benefits of sunscreens are not scientifically established. While sunscreens protect against burning, they do not adequately protect against the whole of the UV spectrum and may encourage people to spend longer in the sun and so increase the risk of skin cancer [227, 228]. So sunscreens should not be endorsed for everyday use by all people in Britain.

Furthermore, sunscreens block synthesis of vitamin D with the result that chronic users have lower levels of vitamin D in blood [229] which the majority of people in the UK can ill afford. People who use sunscreens regularly have been found to have half as much vitamin D in their blood as people who do not use sunscreens [229]. Sunscreens are now included in many cosmetic preparations used on the face further blocking the possibility of casual vitamin D synthesis. Women who use these cosmetics put themselves in danger of severe vitamin D deficiency and ill health unless they take vitamin D supplements or expose other parts of the body to the sun regularly. A public health policy on sunlight should advise against use of such cosmetics in the UK because opportunities for exposure to the sun are so limited here.

Professor Brian Diffey has questioned the wisdom of regular use of sunscreen creams in the UK [228]. He wrote: "while regular sunscreen application can prevent the development of cutaneous dysplasias in sunny countries like Australia without compromising vitamin D levels, daily use of skin products containing UV filters in the UK, which is not known for its sunny climate, is unnecessary and may be potentially harmful."

The SunSmart campaign suggests that sunscreen should be applied 15-30 minutes before going outside. This advice is completely unsuited to the British climate. More appropriate advice for the British Isles would be to expose the body to full sun (so long as there is no baking or burning) for up to half an hour before applying any suncream. In the British Isles the sun often comes and goes behind fast moving clouds and under these circumstances a person may need to be fully exposed to the sun without sunscreen for an hour or more to get optimum vitamin D synthesis.

Sunscreens do protect against burning and so are useful when excessive exposure cannot be avoided and a suitable hat is not entirely effective or cannot be used, for example, when involved in sports such as skiing, sailing, tennis etc. But covering up or seeking shade is generally preferable to sunscreen.

#### The Consensus Statement by The UK Skin Cancer Prevention Working Party

"There is no such thing as a healthy tan. A tan is a sign that already damaged skin is trying to protect itself from further damage. The protecting power of a tan is weaker than that of a mild sunscreen of SPF2-4." from the Consensus Statement of The UK Skin Cancer Prevention Working Party

Government policy on sunlight is based on the UK Consensus Statement on skin cancer [4]. This Consensus Statement has the backing of the British Association of Dermatologists, the Department of Health and some 14 other organisations including the major cancer charities. The Working Party co-ordinates Sun Awareness Week each year at the beginning of June when the SunSmart campaign issues advice to all media emphasising sun avoidance. The Consensus Statement was agreed 10 years ago in 1994 when it reflected current thinking of dermatologists [230]. Since it was written much new evidence on the importance of vitamin D for many other body systems and for prevention of chronic disease has emerged (reviewed above in Part 2).

The Consensus Statement contains several errors and makes omissions when judged by the knowledge we have today. These errors and omissions have been discussed above in relation to the SunSmart campaign. The

Consensus Statement also makes a much quoted, but misleading, claim suggesting that tanning is dangerous. It says: "There is no such thing as a healthy tan". This mantra has been repeated year after year as part of the campaign to prevent skin cancer even though it was considered "debatable" by at least one of those responsible for the Consensus Statement [230]. Constant repetition of the assertion that tanning is unhealthy has given it some spurious authority although there is no more evidence today than there was 10 years ago that tanning is in any way bad for health.

Experts in vitamin D and health, both here and in the United States, who have been consulted have been unable to provide any epidemiological evidence suggesting that tanning is a direct risk for disease or even that it is a marker indicating a high risk of disease. A search of Pub Med, the database of scientific medical literature held by the US National Library of Medicine, has been unable to find any research articles linking tanning with ill health.

The only relevant evidence that can be found suggests that tanning is associated with beneficial effects on health. A deep tan, particularly in childhood and the adolescent years, has actually been found to be associated with protection against melanoma [186, 231], while sunburn in childhood is associated with a reduced risk of prostate cancer [130] and multiple sclerosis [57] later in life. People who develop non-melanoma skin cancers actually have a reduced risk of multiple sclerosis [59]. Tanning, sunburn and non-melanoma skin cancer are all indications of intense exposure to the sun. The finding that such exposure protects against several diseases is entirely consistent with what is known about vitamin D deficiency causing these diseases.

Sunburn should be avoided for obvious reasons but there is no good reason to avoid tanning. On the contrary intense exposure to the sun sufficient to produce a tan is likely to provide lasting benefit as shown by the studies quoted above. A small risk of non-melanoma skin cancer, which is generally a self-limiting and easily treated disease, should be accepted since it is associated with a reduced risk of much more serious disease.

The lay perception that a good tan is a sign of good health has been severely criticised by those who promote sun avoidance. In fact, scientific evidence suggests that the lay perception is correct and the public has been ill-served by the sophistry involved in suggesting that a tan is unhealthy. The phrase "there is no such thing as a healthy tan" is no more than a clever slogan devised by propagandists to discourage people from seeking sun exposure. Controlled exposure to the sun is beneficial and a tan, which provides some protection against sunburn, will naturally be acquired in the course of such exposure. It is quite wrong to make people anxious about a tan when there is no sound basis for doing so.

#### The NHS leaflet 'SunSafety for Children'

This leaflet [221] and supporting material sent out to schools [220] has the same faults as the SunSmart programme. It says nothing about the need to expose the body to the sun in order to obtain vitamin D which is essential for healthy bones and for preventing disease. It says that children should wear a hat whenever outdoors which is quite unnecessary in the British Isles except for particularly fair-skinned children around midday in midsummer if the sky is clear. It advises playing in shaded areas between 11am and 3pm and use of a sunscreen when exposure is unavoidable. These measures will induce vitamin D deficiency/insufficiency in any child and particularly in children with darker skins. The advice is dangerous and should be withdrawn as soon as possible.

#### The chief medical officer's 'sixth tip' for better health

"Protect yourself from the sun – cover up, keep in the shade, never burn and use factor 15 plus sunscreen. Take extra care to protect children."

Sixth tip for healthy living offered by the chief medical officer as part of the public consultation on health [217]

The chief medical officer's advice appears to suggest that exposure to sunlight should always be avoided. While such advice might be appropriate for tropical regions of Australia it is totally unsuited to Great Britain as evidence accumulated in this report shows. The British Isles are so far north that our summers are very short, and even summer sun is often severely restricted by cloud. Therefore advice should be focused on telling the public to take every advantage of the sun in order to remedy the almost universal insufficiency of vitamin D in the UK population.

Appropriate advice from the chief medical officer would be to expose the body as often as possible while taking care to do so safely. It remains to suggest how the chief medical officer's health tip may be re-drafted so that

the public is best advised on how to benefit from the sun with the minimum of risk. A possible form of words is: "Take every opportunity to sunbathe wearing as few clothes as possible for up to half an hour or more per day depending on skin type, previous exposure and time of day. But take care – sensitive skin may burn after only a few minutes. Be ready to cover up or seek the shade to avoid baking or burning. Encourage children to undress in the sun but take care that they do not burn."

Advice of this kind could do much to increase levels of vitamin D in the population with a consequent reduction of the burden of D-deficiency disease, while at the same time doing all that is possible to reduce the risk of skin cancer. The redrafted tip may be more effective in preventing melanoma for two reasons: 1) it will encourage people to sunbathe regularly which will raise their vitamin D level so providing protection against melanoma. 2) advice to sunbathe will be better accepted than advice to cover up and so the advice to avoid burning (which seems to be associated with melanoma) may be better received.

A survey of UK students found that most emphasised the positive benefits of sun exposure, enjoyed sunbathing and did not intend to change this behaviour [232] showing that they are more likely to respond positively to a message that encourages sunbathing. The chief medical officer's advice to use sunscreens would be better avoided for the reasons already given (p29).

## Part 5: A note on methodology

Information for this report has been gathered from many sources. Pub Med, the National Library of Medicine's database, has been searched with hundreds of questions looking for keywords in titles and abstracts of papers. Keywords chosen have included sun, sunlight, light, ultra-violet, season, vitamin D, altitude, latitude, geography, parathyroid, fish, calcium, birth month, month of birth, migrant, immigrant etc. These words have been used on their own or in combination with various diseases known or suspected to be linked with vitamin D deficiency.

The aim has generally been to use open-ended searches. Narrowly defined searches have on the whole been avoided because they exclude unexpected ideas and observations and so may provide unwarranted support for pre-existing concepts. Searches have looked for papers going back at least 10 years. As I accumulated more information I traced the literature back further and developed contacts with many researchers in the various fields who have very kindly supplied me with help of all kinds including useful comments and copies of papers.

The searches began with an interest in schizophrenia and season and have been continued over a period of three years. I started with the very well-established observation, many times repeated, that schizophrenic people are more likely to be born in winter or spring. It seemed possible that this observation might give an alternative view of causes of schizophrenia to that provided by those primarily concerned with genetics of the disease. I studied other diseases with a similar seasonal pattern of births on the grounds that these diseases might have a common cause with schizophrenia. I also studied other seasonal disease patterns.

Explanations of the seasonal occurrence of these diseases have in the past mostly centred on seasonal viral disease. However, despite very strenuous research efforts these diseases have not been consistently linked to virus epidemics. I have reviewed this evidence but have not attempted to summarise it here because it is beyond the scope of this work. Much of the work on seasonal occurrence of disease made no mention of vitamin D even as a hypothesis because the effects of vitamin D were believed by most scientists to be limited to bone formation.

At first I thought that folic acid deficiency might explain the seasonal births of schizophrenics because folic acid deficiency is known to affect the nervous system and the amount of folic acid in the diet varies with season. However, I was unable to find any evidence to support this idea although it remains a theoretical possibility. Vitamin D deficiency/insufficiency was another possibility. As I began to look into it I found a trail of information linking D-deficiency to several chronic diseases several of which had a seasonal pattern of some kind.

Variation in the amount of vitamin D synthesised in the skin depends on the amount of ultra-violet radiation penetrating the skin which in turn depends on latitude, climate, season, altitude, skin colour, and length of exposure. While the amount of vitamin D in the diet varies most notably with consumption of fish. Searches were made for links between these variables and the diseases under study. I was then able to find from the literature whether incidence of disease (or other variables such as prevalence or mortality) consistently increased with various surrogate measures of UV radiation or vitamin D "intake" such as latitude, altitude, skin colour, city or rural residence, air pollution (filters out UV), or diet. With very few exceptions incidence (or other measures) of the chronic diseases under study increased with decreased exposure to UV radiation or lesser amounts of fish in the diet.

There were exceptions, most notably between skin colour and one or two diseases and between latitude and incidence of disease considered at the country level. Probable explanations of such exceptions can be found in cultural differences between black and white people leading, for example, to lower average birth weight of black babies in the United States[233], or in differences of fish consumption, for example between Iceland, Norway and other countries. The exceptions could generally be explained by the fundamental hypothesis (that D-deficiency causes the disease in question) without difficulty – a reassuring test that the hypothesis is likely to be correct. So the overall consistency of these findings using different surrogate measures of D 'intake' suggests a robust association between vitamin D deficiency and a number of chronic diseases.

The power of the vitamin D hypothesis to explain facts and the strength of the association between vitamin D and chronic disease makes it difficult to think of D deficiency as other than causative of these chronic diseases. However, D-deficiency is not likely to be a unique cause of these diseases. Generally D deficiency may be expected to act in concert with other significant risk factors.

In reviewing the literature Sir Austin Bradford Hill's criteria of causation have been born in mind [234]. As well as consistency, these include: strength, specificity, temporality, biological gradient, plausibility, coherence and experiment. All these criteria with the exception of specificity are fulfilled in various ways for many of the chronic diseases considered.

Hill warns against over-emphasising the criterion of specificity. Often specificity is absent as in the large

number of diseases caused by smoking, or, in the example given by Hill: milk may act as a carrier of some eight different infectious diseases. So the association between vitamin D levels (or its surrogates) and two dozen or more different chronic diseases is not a reason in itself to reject the hypothesis of causation. Indeed from what is now known about the presence of vitamin D receptors in some 30 or more different tissues of the body a wide spectrum of disease might be predicted to occur as a result of vitamin D deficiency/insufficiency [25, 180]. Why an individual may suffer one particular disease caused by vitamin D deficiency/insufficiency and not others is presumably determined by their experience of infections, heredity, and their lifestyle, particularly diet.

#### The method of investigative review

The report has set out to answer questions about vitamin D, sunlight, disease and tanning in a systematic way. But the questions that have been asked have changed as the inquiry progressed. This method of inquiry has developed from my own experience in both scientific and journalistic investigations. I call this method of research 'investigative review' to distinguish it from narrative review, which may have a marked individual slant, and systematic review, which may restrict itself to answering certain predetermined questions.

Investigative review, on the other hand, is based on the idea that we do not always know the best questions to ask at the beginning of an inquiry, and that certain assumptions may need to be tested against the literature before we know the best questions to put. An important part of the process is to highlight incorrect assumptions that are widely held about a body of data, and to gather scattered evidence that question existing ideas and may support a new interpretation. To achieve this questions are put in the most general form possible to retrieve the maximum information. Questions take the form: what is the relationship between disease A and factor B, what is the relationship between disease A and related subjects.

In this way I have been able to gather together a large but widely scattered body of evidence on vitamin D. Despite its importance, research on vitamin D has not been fashionable and so its wider relevance has been largely overlooked. An investigative review aims to gather together such scattered evidence and, in this case, consider its relevance to national policy. The narrative in an investigative review consists then of a theme giving an account of what has been discovered and the evidence for it and may present a prime facie case for consideration of a new interpretation of evidence or a change of policy.

## References

- 1. Oppenheimer, S., *Out of Eden. The peopling of the world.* 2003, London: Constable. 440.
- 2. Jablonski, N. and Chaplin, G., *The evolution of human skin coloration*. Journal of Human Evolution, 2000. **39**: p57-106.
- 3. Lapsley, P., *Preventing Skin Cancer Rapid Response to Alison Fry and Julia Verne,* British Medical Journal 2003; 326: 114-115.
- 4. Consensus Statement on sunlight and skin cancer. 1992, UK Skin Cancer Working Party, British Association of Dermatologists.
- 5. SunSmart Evaluation Studies No. 6. 2004, Anti-Cancer Council of Victoria.
- 6. *Cancer trends in England and Wales 1950-1999: studies on medical and population subjects.* 2001, Office for National Statistics: London.
- 7. *Health effects of ultra-violet radiation, Report of an Advisory Group on Non-ionising Radiation.* 2002, National Radiological Protection Board: London. p18-20.
- 8. Gillie, O., Sunny D. The Independent on Sunday, Sunday Review, 2004: p8-12.
- 9. Gillie, O., Ray of Light. pH7, 2003 (autumn 2003): p22-23.
- 10. Gillie, O., When nanny did know best. Parliamentary Monitor, 2004. 113: p58-61.
- **11.** *Nutrition and Bone Health.* 1998, Committee on Medical Aspects of Food Policy (COMA), Department of Health, UK: London. p41-43.
- 12. Wharton, B. and Bishop, N., *Rickets*. Lancet, 2003. 362: p1389-1400.
- 13. Harris, S. and B. Dawson-Hughes, *Seasonal changes in plasma 25-hydroxyvitamin D concentrations of young American black and white women.* American J Clinical Nutrition, 1998. **67**: p1232-1236.
- Serhan, E., et al., Prevalence of hypovitaminosis D in Indo-Asian patients attending a rheumatology clinic. Bone, 1999.
   25: p609-11.
- **15.** Hewison, M., et al., Vitamin D and barrier function: a novel role for extra-renal 1 alpha-hydroxylase. Molecular and Cellular Endocrinology, 2003. in press.
- **16.** Bataille, V., *et al.*, *Exposure to the sun and sunbeds and the risk of cutaneous melanoma in the UK: a case-control study.* Eur J Cancer, 2004. **40**(3): p429-35.
- 17. Ness, A., et al., Are we still dying for a tan? J Cosmetic Dermatology, 2002. 1: p43-46.
- **18.** Suzuki, I., *et al.*, *Participation of the melanocortin-1 receptor in the UV control of pigmentation.* J Investig Dermatol Symp Proc, 1999. **4**(1): p29-34.
- 19. Altmeyer, P., Stohr, L., and Holzman H., *Seasonal rhythm of the plasma level of alpha-melanocyte stimulating hormone.* J Invest Dermatol, 1986. **86**: p454-6.
- 20. Fehm, H., et al., The Melanocortin Melanocyte-Stimulating Hormone/Adrenocorticotropin(4-10) decreases body fat in *humans*. The Journal of Clinical Endocrinology and Metabolism, 2001. **86**(3): p1144-7.
- **21.** Schwartz, M., Progress in the search for neuronal mechanisms coupling type 2 diabetes to obesity. J Clinical Investigation, 2001. **108**: p963-4.
- 22. Davies, P. S., et al., Vitamin D: seasonal and regional differences in preschool children in Great Britain. Eur J Clin Nutr, 1999. 53(3): p195-8.
- 23. Lawson, M. L., Thomas, M., and Hardiman, A., *Dietary and lifestyle factors affecting plasma vitamin D levels in Asian children living in England.* European Journal of Clinical Nutrition, 1999. 53: p268-272.
- 24. Zittermann, A., Scheld, K., and Stehle, P., *Seasonal variations in vitamin D status and calcium absorption do not influence bone turnover in young women*. European Journal of Clinical Nutrition, 1998. **52**: p501-506.
- 25. Zittermann, A., Vitamin D in preventive medicine: are we ignoring the evidence? Br J Nutr, 2003. 89(5): p. 552-72.
- **26.** Guillemant, J., et al., Wintertime vitamin D deficiency in male adolescents: effect on parathyroid function and response to vitamin D supplements,. Osteoporosis International, 2001. **12**: p875-879.
- 27. Hegraty, V., Woodhouse, P., and Khaw, K., *Seasonal variation in 25-hydroxyvitamin D and parathyorid hormone concentrations in healthy elderly people.* Age and Ageing, 1994. 23: p478-482.
- **28.** Basha, B., et al., Osteomalacia due to vitamin D depletion: a neglected consequence of intestinal malabsorption. American Journal of Medicine, 2000. **108**: p296-300.
- 29. Janssen, H., Samson, M., and Verhaar, H., *Vitamin D deficiency, muscle function, and falls in elderly people.* Am J Clin Nutr, 2002. **76**(6): p1454-5 author reply 1455-6.
- 30. Hollis, B. and Wagner, C., Assessment of dietary vitamin D requirements during pregnancy and lactation. American

Journal of Clinical Nutrition, 2004. **79**(5): p717-726.

- **31.** Heaney, R., et al., Human serum 25-hydroxycholecalciferol response to extended oral dosing with cholecalciferol. Am J Clin Nutr, 2003. **77**(1): p204-210.
- 32. Vieth, R., Chan, P., and MacFarlane, G., *Efficiency and safety of vitamin D3 intake exceeding the lowest observed adverse effect level.* Amer J Clinm Nutr, 2001. **73**: p288-294.
- **33.** Hollis, B. and Wagner, C., Vitamin D requirements during lactation: High dose maternal supplementation as therapy to prevent hypovitaminosis D in both mother and nursing infant. American Journal of Clinical Nutrition, 2004. in press.
- 34. Webb, A., et al., Correction of vitamin D deficiency in elderly long-stay patients by sunlight exposure. J Nutritional Medicine, 1990. 1: p201-7.
- **35.** Lawson, D., *et al.*, *Relative contributions of diet and sunlight to vitamin D state in the elderly*. British Medical Journal, 1979. **2**: p303-8.
- **36.** Webb, A. R., et al., An evaluation of the relative contributions of exposure to sunlight and of diet to the circulating concentrations of 25-hydroxyvitamin D in an elderly nursing home population in Boston. Am J Clin Nutr, 1990. **51**(6): p1075-81.
- **37.** Vieth, R., Vitamin D supplementation, 25-hydroxyvitamin D concentrations, and safety. Am J Clin Nutr, 1999. **69**(5): p842-56.
- Holick, M. F., McCollum Award Lecture, 1994: Vitamin D new horizons for the 21st Century. Am J Clin Nutr, 1994.
   60: p619-30.
- **39.** *Nutrition and Bone Health* Committee on Medical Aspects of Food Policy (COMA). 1998, Department of Health, UK: London. p41-43.
- **40.** Vieth, R., Ladak, Y., and Walfish, P. G., *Age-related changes in the 25-hydroxyvitamin D versus parathyroid hormone relationship suggest a different reason why older adults require more vitamin D.* J Clin Endocrinol Metab, 2003. **88**(1): p185-91.
- **41.** Vieth, R., *Effects of vitamin D on bone and natural selection of skin color: How much vitamin D nutrition are we talking about? Bone loss and osteoporosis: An anthropological perspective.*, ed. S. C. Agarwal and S. D. Stout. 2003, New York: Kluwer Academic/Plenum Publishers.
- 42. Wanless, D., Securing Good Health for the Whole Population. 2004, HM Treasury: London. p103-5.
- **43.** Acceptance of new criteria for diagnosis of diabetes mellitus and related conditions by the Canadian Diabetes Association. Can Med Assoc J, 1982. 126(5): p473-6.
- 44. *Review of Vitamin D (draft). 2001,* Food Standards Agency: Expert Group on Vitamins and Minerals: London.
- **45.** Ponsonby, A .L., McMichael, A., and van der Mei, I., *Ultra-violet radiation and autoimmune disease: insights from epidemiological research.* Toxicology, 2002. **181-182**: p71-8.
- **46.** Calvo, M. and Whiting, S., Prevalence of vitamin D insufficiency in Canada and the United States: importance to health status and efficacy of current food fortification and dietary supplement use. Nutr Rev, 2003. **61**(3): p107-13.
- **47.** Grant, W. and de Gruijl, F., *Health benefits of solar UV-B radiation through production of vitamin D, Comment and response*. Photochem Photobiol Sci, 2003. **2**: p1307-10.
- Hayes, C., et al., The immunological functions of the vitamin D endocrine system. Cell Mol Biol (Noisy-le-grand), 2003.
  49: p277-300.
- 49. Heaney, R., Long-latency deficiency disease: insights from calcium and vitamin D. Am J Clin Nutr, 2003. 78: p912-19.
- 50. Holick, M. F., Vitamin D: A millenium perspective. J Cell Biochem, 2003. 88(2): p296-307.
- **51.** Grant, W. and Holick, M., *A review of evidence supporting the role of vitamin D in reducing the risk of non-skeletal diseases.* American Journal of Clinical Nutrition, 2004. for submission.
- 52. Torrey, E. F., et al., Seasonal birth patterns of neurological disorders. Neuroepidemiology, 2000. 19: p177-185.
- 53. Torrey, E. F., et al., Seasonality of births in schizophrenia and bipolar disorder: a review of the literature. Schizophrenia Research., 1997. 28: p1-38.
- 54. Eyles, D., et al., Vitamin D3 and brain development. Neuroscience, 2003. 118: p641-53.
- 55. Hayes, C. E., Cantorna, M. T., and DeLuca, H. F., *Vitamin D and multiple sclerosis*. Proc Soc Exp Biol Med, 1997. 216(1): p21-7.
- **56.** van der Mei, I. A., et al., Regional variation in multiple sclerosis prevalence in Australia and its association with ambient ultra-violet radiation. Neuroepidemiology, 2001. **20**(3): p168-74.
- van der Mei, I. A., et al., Past exposure to sun, skin phenotype, and risk of multiple sclerosis: case-control study. BMJ, 2003.
   327(7410): p316.
- 58. Munger, K., et al., Vitamin D intake and incidence of multiple sclerosis. Neurology, 2004. 62: p. 60-5.

- **59.** Goldacre, M., *et al., Skin cancer in people with multiple sclerosis: a record linkage study.* J Epidemiology Community Health, 2004. **58**: p. 142-4.
- **60.** Embry, A., Snowdon, L., and Vieth, R., Vitamin D and seasonal fluctuations of gadolinium-enhancing magnetic resonance imaging lesions in multiple sclerosis. Ann Neurol, 2000. **48**: p271-2.
- 61. Embry, A., Vitamin D supplementation in the fight against multiple sclerosis. J Orthomolec Med, 2004. in press.
- **62.** Willer, C., et al., Timing of birth influences multiple sclerosis susceptibility: the Canadian Collaborative Study Group. manuscript, 2004.
- **63.** McGrath, J., *Hypothesis: Is low prenatal vitamin D a risk modifying factor for schizophrenia*? Schizophrenia Research., 1999. **40**: p. 173-177.
- **64.** McGrath, J., Selten, J.-P., and Chant, D., Long term trends in sunshine duration and its association with schizophrenia birth rates and age at first registration data from Australia and the Netherlands. Schizophrenia Research., 2002. **54**: p199-212.
- **65.** Davies, G., et al., A systematic review and meta-analysis of Northern Hemisphere season of birth studies in schizophrenia. Schizophr Bull, 2003. **29**: p587-93.
- 66. Fearon, P., et al., Raised incidence of psychosis in all migrant groups in south London, Nottingham and Bristol: The AESOP study. Schizophrenia Research, 2004. 67, Suppl. 15.
- **67.** McGrath, J., et al., A systematic review of the incidence of schizophrenia: the distribution of rates and the influence of sex, urbanicity, migrant status and methodology. BMC Psychiatry, 2004. in press.
- **68.** Levy-Marchal, C., Patterson, C. C., and Green, A., *Geographical variation of presentation at diagnosis of type I diabetes in children: the EURODIAB study.* Diabetologia, 2001. 44 Suppl 3: pB75-80.
- **69.** Patterson, C.C., *et al.*, *Variation and trends in incidence of childhood diabetes in Europe*. EURODIAB ACE Study Group. Lancet, 2000. **355**(9207): p873-6.
- **70.** Staples, J. A., et al., Ecologic analysis of some immune-related disorders, including type 1 diabetes, in Australia: latitude, regional ultra-violet radiation, and disease prevalence. Environ Health Perspect, 2003. **111**(4): p518-23.
- **71.** Rothwell, P., *et al., Seasonality of birth of patients with childhood diabetes in Britain*. British Medical Journal, 1996. **312**: p1456-7.
- 72. McKinney, P. and Eurodiab Group, *Seasonality of birth in patients with childhood Type 1 diabetes in 19 European regions*. Diabetologia, 2001. 44 [Suppl 3]: pB67-B74.
- **73.** Rothwell, P., *et al., Seasonality of birth in children with diabetes in Europe: multicentre cohort study.* British Medical Journal, 1999. **319**: p887-8.
- 74. Samuelson, U., Johanson, C., and Ludvigsson, J., *Month of birth and risk of developing insulin diabetes in southeast Sweden*. Archives of Disease in Childhood, 1999. **81**: p143-146.
- **75.** Ursic-Bratina, N., *et al., Seasonality of birth in children (0-14 years) with type 1 diabetes mellitus in Slovenia.* Journal of Pediatric Endocrinology & Metabolism, 2001. **14:** p47-52.
- **76.** Mikulecky, M., Michalkova, D., and Petrovicova, A., *Coxsackie infection and births of future diabetic children: year, seasonality and secularity.* Journal of Pediatric Endocrinology & Metabolism, 2000. **13**: p523-527.
- Songini, M., et al., Seasonality of birth in children (0-14 years) and young adults (15-29 years) with type 1 diabetes mellitus in Sardinia differs from that in the general population. Journal of Pediatric Endocrinology & Metabolism, 2001.
   14: p781-783.
- **78.** Jongbloet, P. H., *et al.*, *Seasonality of birth in patients with childhood diabetes in the Netherlands*. Diabetes Care, 1998. **21**(1): p190-191.
- **79.** Laron, Z., et al., Month of birth and subsequent development of type 1 diabetes (IDDM). Journal of Pediatric Endocrinology & Metabolism, 1999. **12**: p397-402.
- **80.** Vitamin C. New clinical applications in immunology, lipid metabolism and cancer. Int J Vitam Nutr Res Suppl, 1982. **23**: p1-294.
- **81.** EURODIAB, Vitamin D supplement in early childhood and risk of Type I (insulin-dependent) diabetes mellitus. Diabetologia, 1999. **42**: p51-54.
- 82. Stene, L. C., et al., Use of cod liver oil during pregnancy associated with lower risk of Type I diabetes in the offspring. Diabetologia, 2000. 43(9): p1093-8.
- **83.** Hypponen, E., et al., Intake of vitamin D and risk of type 1 diabetes: a birth-cohort study. Lancet, 2001. **358**(9292): p1500-3.
- **84.** Ziegler, A., *et al. Prediction and prevention of type 1 diabetes.* Poster display. In 18th International Diabetes Federation Congress. 2003. Paris, France.

- **85.** Pozzilli, P., et al. Vitamin D supplementation in patients with recent onset type 1 diabetes: IMDIAB XI Trial. In American Diabetes Association 63rd Scientific Sessions. 2003. New Orleans.
- 86. Zella, J. B. and DeLuca, H. F., Vitamin D and autoimmune diabetes. J Cell Biochem, 2003. 88(2): p216-22.
- 87. Devendra, D., Liu, E., and Eisenbarth, G., *Type 1 diabetes: recent developments.* British Medical Journal, 2004. 328: p750-754.
- **88.** Boucher, B. J., *Inadequate vitamin D status: does it contribute to disorders comprising syndrome 'X'?* British Journal of Nutrition, 1998. **79**: p315-327.
- **89.** McKeigue, P., et al., Relationship of glucose intolerance and hyperinsulinaemia to body fat pattern in south Asians and *Europeans*. Diabetologia, 1992. **35**: p785-791.
- **90.** WHO, Obesity: preventing and managing the global epidemic. 1997: Geneva.
- **91.** Kamycheva, E., Joakimsen, R., and Jorde, R., *Intakes of calcium and vitamin D predict body mass index in the population of northern Norway*. Journal Nutrition, 2002. **132**: p102-6.
- 92. Hang, S., et al., 1-alpha, 25-dihydroxyvitamin D3 modulates human adipocyte metabolism via nongenomic action. The FASEB Journal, 2001. 15: p2751-3.
- 93. Wortsman, J., et al., Decreased bioavailability of vitamin D in obesity. Am J Clin Nutr, 2000. 72: p690-3.
- 94. McCarron, D. A., Calcium metabolism in hypertension. Keio J Med, 1995. 44(4): p105-14.
- **95.** Scragg, R., et al., Plasma 25-hydroxyvitamin D3 and its relation to physical activity and other heart disease risk factors in the general population. Annals of Epidemiology, 1992. **2**: p697-703.
- **96.** Rostand, S., Ultra-violet light may contribute to geographic and racial blood pressure differences. Hypertension, 1997. **30**: p150-6.
- 97. Dustan, H., *Obesity and hypertension in blacks.* Cardiovascular Drugs and Therapy, 1990. 4 Suppl 2: p395-402.
- 98. Krause, R., et al., Ultra-violet B and blood pressure. Lancet, 1998. 352: p709-10.
- **99.** Pfeiffer, M., et al., Effects of a short-term vitamin D3 and calcium supplementation on blood pressure and parathyroid hormone levels in elderly women. Journal of Clinical Endocrinology and Metabolism, 2001. **86**: p1633-1667.
- **100.** Pfeiffer, M., Begerow, B., and Minne, H., *Vitamin D and muscle function.* Osteoporosis International, 2002. **13**(3): p187-94.
- **101.** Plotnikoff, G. and Quigley, B., *Prevalence of severe hypovitaminosis D in patients with persistent, nonspecific musculoskeletal pain.* Mayo Clin Proc, 2003. **78**: p1463-1470.
- **102.** Curry, O., et al., Calcium uptake by sarcoplasmic reticulum of muscle from vitamin D deficient rabbits. Nature, 1983. **249**: p83-4.
- **103.** Bischoff, H. A., et al., Relationship between muscle strength and vitamin D metabolites: are there therapeutic possibilities in the elderly? Z Rheumatol, 2000. 59 Suppl 1: p39-41.
- 104. Ritz, E., R. Boland, and Kreusser, W., *Effects of vitamin D and parathyroid hormone on muscle: potential role in uraemic myopathy.* American Journal of Clinical Nutrition, 1980. 33: p1522-29.
- **105.** Rimaniol, J., Authier, F., and Chariot, P., *Muscle weakness in intensive care patients' initial manifestation of vitamin D deficiency.* Intensive Care Medicine, 1994. **20**: p591-2.
- **106.** Bischoff, H. A., et al., Effects of vitamin D and calcium supplementation on falls: a randomized controlled trial. J Bone Miner Res, 2003. **18**(2): p343-51.
- **107.** Pfeiffer, M., et al., Effects of a short term vitamin D and calcium supplementation on body sway and secondary hyperparathyoridism in elderly women. Journal of Bone and Mineral Research, 2000. **15**: p1113-8.
- **108.** Zittermann, A., *et al., Low vitamin D status: a contributing factor in the pathogenesis of congestive heart failure.* Journal of the American College of Cardiology, 2003. **43**: p105-12.
- **109.** Carlton-Conway, D., *et al., Vitamin D deficiency and heart failure in infancy.* Journal of the Royal Society of Medicine, 2004. **97**(May): p238-239.
- **110.** Scragg, R., *et al.*, *Myocardial infraction is inversely associated with plasma 25-hydroxyvitamin D3 levels: a community based study.* International Journal of Epidemiology, 1990. **19**(3): p559-563.
- 111. Hegarty, V., Woodhouse, P., and Khaw, K., *Seasonal variation in 25-hydroxyvitamin D and parahayrorid hormone concentrations in elderly people.* Age and Ageing, 1994. **23**: p478-82.
- 112. Douglas, A., et al., Winter pressure on hospital medical beds. British Medical Journal, 1991. 303: p508-9.
- 113. Grimes, D., et al., Sunlight, cholesterol and coronary heart disease. Quarterly Journal of Medicine, 1996. 89: p579-589.
- **114.** Scragg, R., Seasonality of cardiovascular disease mortality and the possible protective effect of ultra-violet radiation. International Journal of Epidemiology, 1981. **10**: p337-341.
- 115. Feskens, E. J. and Kromhout, D., *Epidemiologic studies on Eskimos and fish intake*. Ann N Y Acad Sci, 1993.
  683: p9-15.

- **116.** Timms, P. M., et al., Circulating MMP9, vitamin D and variation in the TIMP-1 response with VDR genotype: mechanisms for inflammatory damage in chronic disorders? Quarterly Journal of Medicine, 2002. **95**(12): p787-96.
- **117.** Muller, K., et al., 1,25 hydroxyvitamin D3 inhibits cytokine production by human blood monocytes at the post-transcription level. Cytokine, 1992. **4**: p506-12.
- **118.** Watson, K., *et al.*, *Active serum vitamin D levels are inversely correlated with coronary calcification*. Circulation, 1997. **96**(6): p1755-60.
- **119.** Grant, W., An estimate of premature cancer mortality in the United States due to inadequate doses of solar ultra-violet-B radiation. Cancer, 2002. **94**: p1867-75.
- **120.** Grant, W., A multifactor ecologic analysis of the geographic variation in mortality rates in the U.S.A. manuscript.
- 121. Pritchard, R. S., Baron, J. A., and Gerhardsson de Verdier, M., *Dietary calcium, vitamin D, and the risk of colorectal cancer in Stockholm, Sweden*. Cancer Epidemiol Biomarkers Prev, 1996. 5(11): p897-900.
- 122. White, E., Shannon, J. S., and Patterson, R. E., *Relationship between vitamin and calcium supplement use and colon cancer.* Cancer Epidemiol Biomarkers Prev, 1997. **6**(10): p769-74.
- **123.** Grau, M., et al., Vitamin D, calcium supplementation, and colorectal adenomas: results of a randomised trial. J Natl Cancer Inst, 2003. **95**: p1765-71.
- 124. Peters, U., et al., Vitamin D, calcium, and vitamin D receptor polymorphism in colorectal adenomas. Cancer Epidemiol Biomarkers Prev, 2001. 95: p1267-71.
- 125. Emerson, J. and Weiss. N., *Colorectal cancer and solar radiation*. Cancer Causes Control, 1992. **3**: p95-9.
- **126.** Grant, W. and Garland, C., *A critical review of studies on vitamin D in relation to colorectal cancer*. Nutr Cancer, 2004. in press.
- **127.** John, E. M., et al., Vitamin D and breast cancer risk: the NHANES I Epidemiologic follow-up study, 1971-1975 to 1992. National Health and Nutrition Examination Survey. Cancer Epidemiol Biomarkers Prev, 1999. **8(**5): p399-406.
- **128.** Freedman, D., Dosemeci, M., and McGlynn, K., Sunlight and mortality from breast, ovarian, colon, prostate, and non-melanoma skin cancer: a composite death certificate based case-control study. Occup Environ Med, 2002. **59**: p257-62.
- **129.** Robsahm, T., et al., Vitamin D3 from sunlight may improve the prognosis of breast, colon and prostate cancer. Cancer Causes and Control, 2004. 15.
- **130.** Luscombe, C., et al., Exposure to ultra-violet radiation: association with susceptibility and age at presentation with prostate cancer. Lancet, 2001. **358**: p641-2.
- 131. Feldman, D., Zhao, X. Y., and Krishnan, A. V., Vitamin D and prostate cancer. Endocrinology, 2000. 141(1): p5-9.
- van den Bemd, G. J. and Chang, G. T., Vitamin D and vitamin D analogs in cancer treatment. Curr Drug Targets, 2002.
   3(1): p85-94.
- 133. Mehta, R. G. and Mehta, R. R., Vitamin D and cancer. J Nutr Biochem, 2002. 13(5): p252-264.
- 134. Ylikomi, T., et al., Antiproliferative action of vitamin D. Vitam Horm, 2002. 64: p357-406.
- **135.** Lamprecht, S. and Lipkin, M., *Chemoprevention of colon cancer by calcium, vitamin D and folate: molecular mechanisms.* Nat Rev Cancer, 2003. **3**: p601-14.
- **136.** McCarty, M. F., Parathyroid hormone may be a cancer promoter an explanation for the decrease in cancer risk associated with ultra-violet light, calcium, and vitamin D. Med Hypotheses, 2000. **54**(3): p475-82.
- **137.** Tovar Sepulveda, V. A. and Falzon, M., *Regulation of PTH-related protein gene expression by vitamin D in PC-3 prostate cancer cells.* Mol Cell Endocrinol, 2002. **190**(1-2): p115-24.
- **138.** Chen, T., *et al.*, *Evaluation of vitamin D analogs as therapeutic agents for prostate cancer*. Recent Results Cancer Res., 2003. **164**: p273-88.
- **139.** Grant, W., Cost due to insufficient UV-B and /or vitamin D., Personal communication. 2003.
- **140.** Fleischer, A. J., et al., Commercial tanning bed treatment is an effective psoriasis treatment: results from an uncontrolled clinical trial. J. Invest Dermatol, 1997. **109**: p170-4.
- 141. Kira, M., Kobayashi, T., and Yoshikawa, K., Vitamin D and the skin. J Dermatol, 2003. 30: p429-37.
- 142. Chan, T., Vitamin D deficiency and susceptibility to tuberculosis. Calcified Tissue International, 2000. 66: p476-478.
- 143. Bellamy, R., Evidence of gene-environment interaction in development of tuberculosis. Lancet, 2000. 355(9204): p588.
- 144. Gallerani, M. and Manfredini, R., Seasonal variation in herpes zoster infection. Br J Dermatol, 2000. 142: p560-1.
- **145.** Chan, T., et al., A study of calcium and vitamin D metabolism in Chinese patients with pulmonary tuberculosis. Journal of Tropical Medicine and Hygiene, 1994. **97**: p26-30.
- **146.** Davies, P., A possible link between vitamin D deficiency and impaired host defence to Mycobacterium tuberculosis. Tubercle, 1985. **66**: p301-306.
- 147. Douglas, A., Strachan, D., and Maxwell, J., Seasonality of tuberculosis: the reverse of other respiratory diseases in the

*UK*. Thorax, 1996. **51**: p944-6.

- **148.** Douglas, A., Shaukat, A., and Bakhshi, S., *Does vitamin D deficiency account for ethnic differences in tuberculosis seasonality in the UK*? Ethnicity and Health, 1998. **3**(4): p247-53.
- 149. Hobday, R., The healing Sun sunlight and health in the 21st Century. 1999, London: Findhorn Press.
- **150.** Dowling, G. and Prosser-Thomas, E., *Treatment of lupus vulgaris with calciferol*. Lancet, 1946. i: p919-22.
- **151.** Thys-Jacobs, S., et al., Vitamin D and calcium dysregulation in the polycystic ovarian syndrome. Steroids, 1999. **64**(6): p430-435.
- 152. Thys-Jacobs, S., et al., Reduced bone mass in women with pre-menstrual syndrome. Journal of Women's Health, 1995.
  4: p161.
- **153.** Thys-Jacobs, S., Alleviation of migraines with therapeutic vitamin D and calcium. Headache, 1994. **34**(10): p590-592.
- **154.** Thys-Jacobs, S., Altered calcium and vitamin D in PMDD or severe PMS. 2003, National Institutes of Health.
- **155.** Thys-Jacobs, S. and M. Alvir, J., *Calcium-regulating hormones across the menstrual cycle: evidence of a secondary hyperparathyroidism in women with PMS.* Journal of Clinical Endocrinology and Metabolism, 1995. **80**(7): p2227-2232.
- **156.** Lamb, E., *et al.*, *Metabolic bone disease is present at diagnosis in patients with inflammatory bowel disease*. Alimentary Pharmacology and Therapy, 2002. **16**: p1895-1902.
- 157. Podolsky, D., Inflammatory bowel disease. New England Journal of Medicine, 1991. 325: p928-1016.
- **158.** Sonnenberg, A., McCarty, D., and Jacobsen, S., *Geographic variation of inflammatory bowel disease within the United States.* Gastroenterology, 1991. **100**: p143-9.
- **159.** Asian rickets in Britain. Lancet, 1981(August 22): p402.
- **160.** Ooms, M., et al., Prevention of bone loss by vitamin D supplementation in elderly women: a randomised double-blind trial. Journal of Clinical Endocrinology and Metabolism, 1995. **80**: p1052-8.
- 161. Peacock, M., Nutritional aspects of hip fractures. Challenges of Modern Medicine, 1995. 7: p213-222.
- **162.** Felton, D. and Stone, W., *Osteomalacia in Asian immigrants during pregnancy*. British Medical Journal, 1966. **1**: p1521-1522.
- 163. Chalmers, J., Vitamin D deficiency in elderly people. British Medical Journal, 1991. 303(6797): p314-5.
- **164.** Dawson-Hughes, B. and *et al*, *Vitamin D Round Table discussion about optimal vitamin D for osteoporosis*, Lausanne, Switzerland, May 2003; proceedings document in press, 2004.
- **165.** Diamond, T., et al., Hip fracture in elderly men: the importance of subclinical vitamin D deficiency and hypogonadism. Medical Journal of Australia, 1998. **169**: p138-41.
- **166.** LeBoff, M., *et al.*, *Dietary and lifestyle factors affecting Asian children living in England*. European Journal of Clinical Nutrition, 1999. **53**: p268-272.
- **167.** Javaid, M., et al., Maternal vitamin D status during late pregnancy and accrual of childhood bone mineral. J Bone Miner Res, 2003. **18**: pS1-S13.
- **168.** Namgung, R. and Tsang, R. C., Factors affecting newborn bone mineral content: in utero effects on newborn bone mineralization. Proc Nutr Soc, 2000. **59(1)**: p55-63.
- **169.** Tobias, J. and Cooper, C., *PTH/PTHrP Activity and the programming of skeletal development in utero*. Journal of Bone and Mineral Research, 2004. **19**(2): p177-182.
- **170.** Chapuy, M., et al., Vitamin D3 and calcium to prevent hip fractures in elderly women. New England Journal of Medicine, 1992. **327**: p1637-42.
- **171.** Trivedi, D., Doll, R., and Khaw, K., Effect of four monthly oral vitamin D3 (cholecalciferol) supplementation on fractures and mortality in men and women living in the community: randomised double blind controlled trial. British Medical Journal, 2003. 326.
- **172.** Riggs, B. and Melton, L., *The worldwide problem of osteoporosis: Insights afforded by epidemiology*. Bone, 1995. **17** Supplement(5): p505S-511S.
- 173. National Service Framework for Older People. 2001, Department of Health: London.
- **174.** Brooke, O., Butters, F., and Wood, C. M., *Intrauterine vitamin D nutrition and postnatal growth in Asian infants*. British Medical Journal, 1981. **283**: p1024-1025.
- **175.** Purvis, R., et al., Enamel hyoplasia of the teeth associated with neonatal tetany: manifestation of maternal vitamin deficiency. Lancet, 1973. **2**: p811-814.
- **176.** Skinner, M. F., Hadaway, W., and Dickie J., *Effects of ethnicity and birth month on localized enamel hypoplasia of the primary canine*. ASDC J Dent Child, 1994. **61**(2): p109-13.
- 177. East, B., Mean annual hours of sunshine and the incidence of dental caries. American Journal of Public Health, 1939.

**29**: p777-780.

- **178.** Pitts, N. B. and Palmer, J. D., *The dental caries experience of 5-, 12- and 14-year-old children in Great Britain. Surveys coordinated by the British Association for the Study of Community Dentistry in 1991/92, 1992/3 and 1990-91.* Community Dent Health, 1994. **11**(1): p42-52.
- 179. Krall, E. A., et al., Calcium and vitamin D supplements reduce tooth loss in the elderly. Am J Med, 2001. 111(6): p452-6.
- **180.** DeLuca, H. F. and Cantorna, M. T., *Vitamin D: its role and uses in immunology.* FASEB Journal, 2001. **15**: p2579-2585.
- **181.** Heaney, R., *Vitamin D: How much do we need, and how much is too much?* Osteoporosis International, 2000. **11**: p553-555.
- **182.** Safe upper levels for vitamins and minerals. Report of the expert group on vitamins and minerals. 2002, Department of Health: London. p133-140.
- **183.** Finkel, E., Sorting the hype from the facts in melanoma. Lancet, 1998. **351**(9119).
- **184.** Hakansson, N., et al., Occupational sunlight exposure and cancer incidence among Swedish construction workers. Epidemiology, 2001. **12**(5): p552-7.
- **185.** Kaskel, P., et al., Outdoor activities in childhood: a protective factor for cutaneous melanoma? Results of a case-control study in 271 matched pairs. Br J Dermatol, 2001. **145**(4): p602-9.
- 186. Elwood, J., Melanoma and sun exposure. Seminars in Oncology, 1996. 23(6): p650-666.
- 187. Rivers, J., Is there more than one road to melanoma? Lancet, 2004. 363: p728-30.
- **188.** Elwood, J. and Gallagher, R., Body site distribution of cutaneous malignant melanoma in relationship to patterns of body exposure. Int J Cancer, 1998. **78**(3): p276-80.
- 189. Crombie, I. K., Racial differences in melanoma incidence. Br J Cancer, 1979. 40(2): p185-93.
- **190.** Koh, H., et al., Prevention and early detection strategies for melanoma and skin cancer. Arch Dermatol, 1996. **132**: p436-442.
- 191. Begg, C. B., The search for cancer risk factors: when can we stop looking? Am J Public Health, 2001. 91(3): p360-4.
- **192.** Shors, A. R., *et al.*, *Melanoma risk in relation to height, weight, and exercise (United States)*. Cancer Causes Control, 2001. 12(7): p599-606.
- 193. Kirkpatrick, C. S., White, E., and Lee, J. A., *Case-control study of malignant melanoma in Washington State. II. Diet, alcohol, and obesity.* Am J Epidemiol, 1994. 139(9): p869-80.
- **194.** Grant, W., Melanoma has a complex etiology that includes UV exposure, skin pigmentation and type, diet and obesity. British Medical Journal, 2003. **327**: p1306 rapid response.
- **195.** Martinez-Gonzalez, M., et al., Physical inactivity, sedentary lifestyle and obesity in the European Union. International Journal of Obesity, 1999. **23**: p1192-1201.
- 196. Bray, G., Obesity: a time bomb to be defused. Lancet, 1998. 352: p160-161.
- **197.** Prentice, A. M. and Jebb, S. A., *Fast foods, energy density and obesity: a possible mechanistic link*. Obes Rev, 2003. **4**(4): p187-94.
- **198.** Ness, R. B. and Roberts, J. M., *Heterogeneous causes constituting the single syndrome of pre-eclampsia: a hypothesis and its implications.* Am J Obstet Gynecol, 1996. **175**(5): p1365-70.
- 199. Wong, C., R. Strange, and Lear, J., *Basal cell carcinoma*. British Medical Journal, 2003. 327: p794-798.
- 200. Black, H. S., et al., Evidence that a low-fat diet reduces the occurrence of non-melanoma skin cancer. Int J Cancer, 1995.
   62(2): p165-9.
- 201. Black, H. S., Influence of dietary factors on actinically-induced skin cancer. Mutat Res, 1998. 422(1): p185-90.
- **202.** Hunter, D., et al., Diet and risk of basal cell carcinoma of the skin in a prospective cohort of women. AEP, 1992. **2**(3): p231-239.
- **203.** van Dam, R. M., et al., Diet and basal cell carcinoma of the skin in a prospective cohort of men. Am J Clin Nutr, 2000. **71**(1): p135-41.
- **204.** Autier, P. and Dore, J. F., Influence of sun exposures during childhood and during adulthood on melanoma risk. EPIMEL and EORTC Melanoma Cooperative Group. European Organisation for Research and Treatment of Cancer. Int J Cancer, 1998. **77**(4): p533-7.
- **205.** Autier, P., et al., Melanoma risk and residence in sunny areas. EORTC Melanoma Co-operative Group. European Organization for Research and Treatment of Cancer. Br J Cancer, 1997. **76**(11): p1521-4.
- 206. Whiteman, D. C., Whiteman, C. A., and Green, A. C., *Childhood sun exposure as a risk factor for melanoma: a systematic review of epidemiologic studies.* Cancer Causes Control, 2001. 12(1): p69-82.
- **207.** Pfahlberg, A., Kolmel, K. F., and Gefeller, O., *Timing of excessive ultra-violet radiation and melanoma: epidemiology does not support the existence of a critical period of high susceptibility to solar ultra-violet radiation- induced melanoma.*

Br J Dermatol, 2001. 144(3): p471-5.

- **208.** Corona, R., et al., Risk factors for basal cell carcinoma in a Mediterranean population. Arch Dermatol, 2002. **137**: p1162-8.
- **209.** Gallagher, R., et al., Sunlight exposure, pigmentary factors, and risk of nonmelanocytic skin cancer. Arch Dermatol, 1995. 131: p157-63.
- **210.** Van Dam, R., et al., Risk factors for basal cell carcinoma of the skin in men: results from the health professionals follow-up study. American Journal of Epidemiology, 1999. **150(**5): p459-460.
- 211. Hillebrand, G. G., et al., Quantitative evaluation of skin condition in an epidemiological survey of females living in northern versus southern Japan. J Dermatol Sci, 2001. 27 Suppl 1: pS42-52.
- **212.** Leung, W. and Harvey, I., Is skin ageing in the elderly caused by sun exposure or smoking? Br J Dermatol, 2002. **147**(6): p1187-91.
- 213. Brooke, R. C., et al., Discordance between facial wrinkling and the presence of basal cell carcinoma. Arch Dermatol, 2001. 137(6): p751-4.
- 214. Purba, M. B., et al., Skin wrinkling: can food make a difference? J Am Coll Nutr, 2001. 20(1): p71-80.
- 215. Grant, W. and Heaney, R., personal communication. 2004.
- 216. Grant, W., Comments on Fry A, Verne J. Preventing skin cancer. British Medical Journal, 2003. 326: p114-5.
- 217. Choosing Health? A consultation on improving people's health. 2004, Department of Health: London.
- **218.** Evans, J. M., et al., Impact of type 1 and type 2 diabetes on patterns and costs of drug prescribing: a population-based study. Diabetes Care, 2000. **23**(6): p770-4.
- **219.** *Preventing accidental injury priorities for action.* 2002, Department of Health: London.
- **220.** Felton, M., *Sunsafety and evaluation report and conclusions 2003.* 2004, Brighton and Hove City Council Education Department: Brighton and Hove, UK.
- 221. Sun Safety for Children. 2004, National Health Service: London, UK.
- 222. Diffey, B. and Gies, H., The confounding influence of sun exposure in melanoma. Lancet, 1998. 351 (April 11): p1101-2.
- 223. SunSmart: Be safe in the sun. 2004, Cancer Research UK.
- 224. Bauer, J. and Garbe, C., Acquired melanocytic nevi as risk factor for melanoma development. A comprehensive review of epidemiological data. Pigment Cell Research, 2003. 16: p297-306.
- **225.** Evans, R., et al., Risk factors for the development of malignant melanoma I: review of case-control studies. J Dermatol Surg Oncol, 1988. **14**: p393-408.
- **226.** Clemens, T. L., et al., Increased skin pigment reduces the capacity of the skin to synthesise vitamin D3. Lancet, 1989. **2**: p1104-5.
- 227. Christensen, D., *Data still cloudy on association between sunscreen use and melanoma risk*. Journal of the National Cancer Institute, 2003. 95(13): p932-3.
- 228. Diffey, B., Sun protection: have we gone too far? Br J Dermatol, 1998. 138: p562-3.
- 229. Matsuoka, L., et al., Chronic sunscreen use decreases circulating concentrations of 25-hydroxyvitamin D. Arch Dermatol, 1988. 124: p1802-1804.
- 230. Marks, R., Primary prevention of skin cancer. British Medical Journal, 1994. 309: p285-286.
- 231. White, A., Kirkpatrick, C., and Lee, J., *Case-control study of malignant melanoma in Washington State. 1. Constitutional factors and sun exposure.* Am J Epidemiol, 1994. 139: p857-868.
- 232. Fry, A. and Verne, J., Preventing skin cancer. British Medical Journal, 2003. 326: p114-115.
- **233.** Fuller, K. E., *Low-birth-weight infants: The continuing ethnic disparity and the interaction of biology and environment.* Ethn Dis, 2000. **10**: p432-45.
- Hill, A., *The Environment and Disease: Association or Causation*. Proceedings of the Royal Society of Medicine, 1965.
   58: p295-300.

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