COLGAN INSTITUTE
Estb. 1974
RESOURCES FOR HUMAN HEALTH & PERFORMANCE

The Sun Will Save Your Life

Dr Michael Colgan

Sunlight is our primary source of vitamin D. Pre-vitamin D is made from 7-dehydrocholesterol by keratinocytes in the epidermis of bare skin exposed to ultra-violet-B light (UV-B) in the wavelength range 290–315 nm, which then is converted in the skin cell by a heat dependent process to vitamin D3 (cholecalciferol). Michael Holick, and his colleagues, at Boston University Medical Center and the University of California, San Diego, have shown that less than 20% of vitamin D in Americans comes from food and vitamin supplements.[1]

Vitamin D is one of the most ancient hormonal signals used by evolution to develop life. This response to ultra-violet light is built into human DNA, and conserved across species for more than 500 million years from ammonites to man. Because of this ancient lineage, and its adoption by evolution for numerous purposes, Vitamin D is essential for the operation of multiple systems in the human body.[2] Any cultural changes that interfere with human access to sufficient UV-B cause multiple diseases.

One such cultural change began in the 1970s with the invention of effective sunscreens. Companies that make sunscreens began to issue dire warnings that exposing human skin to the sun inevitably results in a variety of skin cancers, including, horror of horrors, deadly melanoma. These companies gathered numerous dermatologists onto their staffs to support such warnings. They advise us all never to appear in sunshine unless we first cover every part of our exposed skin with their sunscreens. I want to show you why this is bad advice. By continually covering your skin with sunscreen, you are placing yourself at high risk of serious disease. By blocking UV-B, you are removing the most ancient, and essential, support of human life.

Because of increasing incidence of skin cancer in the latter part of the 20th century, and today, public health policies in the US and Canada, also contain warnings about sun bathing and skin cancer, and the advisability of sunscreens.[3,4] These policies, however are a phenomenon of the 1980s, a phenomenon that may be based more on pharmaceutical lobbying than medical science. For the 70 years prior to the invention of effective sunscreens, public health policies in both countries favoured regular sun exposure as an inexpensive and practical health measure to reduce disease.[3,4]

This article adds to many others by top medical scientists urging governments to once more advo-

cate the sensible use of sunshine as a potent weapon against multiple diseases. I want to show you some of the evidence that, If Canadians and Americans return to the practice of regular sunshine on bare skin, (or, as a safer alternative, dramatically increase Vitamin D3 supplementation), it will save hundreds of thousands of lives every year.

Does Sunshine Cause Disease

The relationship between ultra-violet radiation and disease is so complex that it is only now, in the second decade of the 21st century, that scientists cooperating from many different disciplines have been able to work it out. Here is a sketch of the latest science.

Over the last 3 million years, humanity evolved from earlier hominins in tropical Africa. During that time, most of the African forests were progressively destroyed by rising temperatures, and replaced by the savannah. Losing their shaded home and its abundant vegetarian diet, our ancestors had to evolve to survive as hunter/gatherers on sparse open plains.[5-8]

Their biggest problem was how to prevent overheating, especially during the long endurance running required to run down game. These hunts could continue non-stop for as much as a week. Evolution made three unique changes to those who survived to become our ancestors. First, unlike other primates, they evolved progressively less body hair until they were almost hairless. Second, again unlike other primates, they developed sweat glands all over their bodies. Sweat evaporation from bare skin is the most efficient cooling system in the entire animal kingdom.[5-8]

To solve the problem of bare skin exposure to intense solar radiation, evolution made a third change. We developed pigment glands, melanocytes, over almost all our skin, even on exposed sexual organs. Melanocytes secrete the pigment melanin, which absorbs and neutralizes ultraviolet radiation very efficiently. Other primates, protected by their hair, have always had light skins and few melanin glands.[5-9]

Human skin pigmentation became very dark, because only those born with massive numbers of melanin glands could produce sufficient color to survive against the damaging effects of the intense UV of tropical Africa.[5-8] Our ancestors there lived almost naked, as many people still do in Africa today. Those who survived, developed sufficient melanocytes to protect them from melanoma and other skin cancers, but not such an abundance of melanocytes that it prevented solar radiation from penetrating the skin to produce the vitamin D essential for many systems in the body. The incidence of melanoma in black races in Africa is only about 5% of the incidence in white people there.[10]

This state of pastoral equilibrium, became distorted as soon as humans began to wear clothes and build shelters from the sun, roughly 100,000 years ago. Exposure of the skin to sunlight declined dramatically. So, although African skins are designed by evolution to require long sun exposure to produce sufficient vitamin D, they were no longer getting it. Today, because of modern culture, sun-proof buildings, and clothing, African and Arabian countries, that have the highest levels of UV, also have some of the highest rates of vitamin D deficiency diseases in the world.[11-14]

As humans migrated in waves from the tropics, the reduction in solar radiation at higher latitudes (further from the sun) allowed people with lighter skins to survive. Natural selection also favoured lighter skin colour that matched the reduced solar radiation. Lighter skinned people became domi-

nant at higher latitudes primarily because their skins allowed sufficient UV to penetrate to make the vitamin D essential to maintain health.[9]

As people migrated to higher latitudes, those with dark skins became less able to survive and gradually died out. Their genetically given melanin protection against the tropical sun did not permit them to make sufficient vitamin D to protect them from multiple vitamin D deficiency diseases. By the time migration spread to northern latitudes, such as Scandinavia, all the people who survived to be the ancestors of the races there were white skinned.[9]

How Evolution Affects Your Skin Today

Our evolutionary history affects melanoma and skin cancer in us because of our DNA, which, in response to environmental inputs, expresses the codes for every one of the 100,000+ different proteins and lipids that compose us. Because of interbreeding between peoples of every skin color, the DNA of human skin is now inextricably mixed. There are no longer any pure races of a single skin color. Consequently, there is no way to assess any individual's skin susceptibility in any group or sample, unless you also know the individual ancestry of each subject. Also, because of frequent migrations back and forth across latitudes, many people do not have skin designed for the latitude at which they now live.[15-19]

Because of migration, especially over the last 500 years, some people whose skin DNA evolved for life at high latitudes with low UV, now live at low latitudes, with high UV. Their skin makes ample vitamin D, but does not have sufficient melanocytes, and cannot produce sufficient melanin, to protect them from the high UV. Consequently, they are at increased risk of skin cancers, including melanoma.[15-19]

Conversely, many people whose skin DNA evolved for life at tropical latitudes, now live at temperate latitudes with low UV, especially in the US and in the rapidly increasing population of Canada. Their skin is protected from melanoma, but has too many melanocytes, producing too much melanin. It does not permit sufficient of the reduced UV at American latitudes, and worse, Canadian latitudes, to penetrate the skin and form vitamin D. These people have chronically low serum vitamin D, and are at increased risk of all the diseases linked to vitamin D deficiency, including numerous forms of cancer.[15-19] To then add sunscreen, which prevents UV penetration, and thus prevents the skin from making vitamin D, is simply increasing the current hazard to their health.

Vitamin D Deficiency Diseases

The main marker of vitamin D status is the blood level of 25-hydroxyvitamin D, which is usually measured as serum 25-hydroxyvitamin D. For simplicity I will refer to it simply as serum vitamin D. The level of serum vitamin D required for minimal risk of deficiency diseases is often quoted as 40ng/mL, (100 nmol/L) with a minimum level for health of 32 ng/mL (70 nmol/L) and a top safe level of 100ng/mL (250 nmol/L).

Because the high levels of melanin in their skin is unsuited to the reduced UV at the latitudes where they now live, black Americans have very deficient serum vitamin D levels, an average of 16 ng/mL (40 nmol/L). Because of recent cultural practices of sun avoidance, and use of sunscreen, white Americans also have low serum vitamin D levels, on average 25 ng/ml (62.5 nmol/L). Canadians

also have deficient serum vitamin D, on average, 27ng/mL (67.7 nmol/L).[20-22] From the research on skin evolution and migration noted above, and the recent cultural practices of sun avoidance and use of sunscreen, it is not surprising that most Americans and Canadians have levels of serum vitamin D that put them at high risk of multiple diseases.

Latitude is a key variable. Michael Holick and colleagues have shown that, in Boston (latitude 42°N), UV is inadequate to generate healthy levels of vitamin D for one-third of each year, from November through February. In Edmonton (latitude 53°30'N), this period extends for half each year, from October through March.[23,24] Edmonton has a very high incidence of vitamin D deficiency.[23,24]

It has been established for decades that vitamin D deficiency causes rickets, and osteoporosis.[25] But it is only now, entering the second decade of the 21st century, that science has uncovered the full extent of vitamin D deficiency disorders. We will review some of the studies showing that, in addition to destruction of bone, vitamin D deficiency can lead to a wide variety of cancers, cardiovascular diseases, septicemia, adult-onset diabetes, multiple sclerosis, and senility.[26]

Vitamin D Deficiency Causes Cancer

Although still not commonly known to the public, the link between cancer and vitamin D deficiency has been known since the first accurate cancer maps were drawn up in the 1970s. Using these maps in 1980, Cedric and Frank Garland of Johns Hopkins University showed that colon cancer mortality rates increased with latitude, and reasoned that higher levels of vitamin D in the serum of people in the south were protecting them. Edward Gorham then showed a link between high serum vitamin D and reduced risk of colon cancer.[27] William Grant later carried out numerous ecologic studies showing that high serum vitamin D protected against numerous other cancers.[28]

In 2006, Michael Holick and colleagues at the University of California, San Diego, reviewed 63 studies of breast, colon, ovarian, and prostate cancer, showing that people with high levels of serum vitamin D have significantly lower risk of developing these cancers.[29] In 2009, these reviews were extended by other researchers to cover 18 different types of cancer.[30]

There are now more than 10,000 research papers on the association between low UV exposure, low serum vitamin D, and cancer. Ostensibly, it would take at least 10,000 pages to review them all adequately. There are many positive findings, negative findings, and neutral findings. In this short paper, I can mention only a tiny few, so have selected some of the largest, and most convincing studies of the worst and most frequent cancers. These are my best effort to give you evidence that is succinct, yet representative of the research overall.

A frequently quoted study by William Grant, published in Cancer in 2002, yields an excellent snapshot of the extent of cancer caused by insufficient exposure to the sun. He used all available US statistics to determine how many types of cancer are affected by solar radiation, and how many premature deaths from cancer occur due to insufficient (UV)-B radiation. Results confirmed previous studies. High solar UV-B radiation correlates with reduced risk of cancer of the breast, colon, ovary, and prostate as well as non-Hodgkin's lymphoma. Results also identified eight additional cancers linked to low UV-B exposure: bladder, esophageal, kidney, lung, pancreatic, rectal, stom-

ach, and uterine.[31]

He estimated the number of premature deaths from these cancers in the US, resulting from low UV-B exposure, at 23,800 per year, nearly as many people as are killed in traffic accidents. The study concluded that much of the geographic and seasonal variations in cancer mortality rates in the US is caused by variations in solar UV-B. Thus, a very large number of lives could be saved from cancer by the simple expedient of increased exposure to the sun, or more safely but a little less effectively, by regular vitamin D3 supplementation, especially in winter months.[31]

Breast Cancer

For breast cancer, a meta-analysis of 1,731 studies on vitamin D intake and breast cancer by Tina Gissel and colleagues at Aarhus University in Denmark, found that only six of the studies provided the original data. Reviewing these data, they found that most of the women involved had very low vitamin D intake, typically less than 150 IU per day, much lower even than the obsolete recommended intake of 400 IU per day. They separated out the groups of women who had vitamin D intake of at least 400 IU per day, and found that these groups had significantly fewer cases of breast cancer.[32]

In 2008, another large study from the University of California, San Diego measured the latitudinal variation in UV, serum vitamin D, and breast cancer in women in 107 countries. They controlled for interfering variables that are also associated with breast cancer including, overweight, smoking, and alcohol consumption. They found that the incidence of breast cancer declined as latitude declined towards the equator, and UV levels increased. They also found that women who had serum vitamin D levels above 22 ng/ml (55 mmol/L) had a much reduced incidence of breast cancer.[33]

Colorectal Cancer

For colorectal cancer, the Third National Health and Nutrition Examination Survey, covered 16,818 participants. Those with serum vitamin D levels above 32 ng/ml (80 nmol/L), had a 72% lower risk of death from colorectal cancer, than those with serum vitamin D levels below 20 ng/mL (50 nmol/L).[34]

In a large, representative, prospective study, researchers measured the serum vitamin D of 25,620 American volunteers to investigate the link with subsequent colon cancer. After 8 years, risk of colon cancer was reduced by 75% in subjects with serum vitamin D levels of 27-32 ng/mL (67.5-80 mmol/L), and by 80% in subjects with vitamin D levels of 33-41 ng/mL (82.5-102.5 nmol/L).[35] You can understand from this and similar findings why the minimum safe level of serum vitamin D is now often quoted as 32 ng/mL (80 nmol/L).

Vitamin D Deficiency Causers Cardiovascular Disease

In the large, long-term, Health Professionals Follow-up Study, Edward Giovannucci and colleagues at Harvard University analysed the vitamin D status of 18,225 men aged 40 to 75 years who were free of diagnosed cardiovascular disease at blood collection between 1993 and 1995. Over 10 years of follow-up, 454 of the men developed heart attacks or fatal coronary heart disease. The researchers controlled for all likely confounding variables including, family history of heart disease, Body Mass Index, alcohol consumption, physical activity, hypertension, ethnicity, blood

lipid levels, even omega-3 intake. Those deficient in vitamin D, with serum vitamin D levels of 15-29.9 ng/mL (37.5-74.7 nmol/L), had significantly higher risk of heart disease than those with serum vitamin D above 30 ng/ml (75 nmol/L).[36]

Vitamin D Deficiency Causes Septicemia

Septicemia (what used to be called "blood poisoning") is a systemic inflammatory response to toxic organisms or their products in the blood. High levels of serum vitamin D produce high levels of cathelicidin in the blood, a potent anti–sepsis agent.[37] Over the last 30 years, since the introduction of sunscreen and the reduction in sun exposure, incidence of septicemia, has skyrocketed to become the 10th leading cause of death in the US, now killing 125,000 people every year.[38] In Canada, there are now about 23,000 deaths per year from septicemia, a higher incidence than even the US.[39]

In the US and Canada, the highest incidence of septicemia occurs in winter and more northern latitudes, areas with the lowest UV, and the lowest incidence occurs in summer and fall and in southern latitudes and western longitudes, areas with the highest UV.[40] In accord with the explanation given earlier in this paper, of the evolution of skin and latitude at which people now live, incidence of septicemia is higher among dark skinned than light skinned people.[40] The incidence and distribution of septicemia parallels the distribution of low levels of serum vitamin D in the American and Canadian populations. A direct link between high vitamin D level and resistance to septicemia has yet to be tested in prospective trials, but the data to date suggest it may be of great benefit.

Vitamin D Deficiency Causes Adult-onset Diabetes

Thirty years ago, animal studies established that vitamin D deficiency reduces insulin secretion from the pancreas.[41] Recently, two large population-based studies in the US and The UK have shown inverse correlations between human serum vitamin D levels and risk of diabetes.[42,43]

The largest study, covering 33,951 women in the Women's Health Initiative Trials, found that supplemental vitamin D had no effect on diabetes risk.44 But, there was no control for the effect of sun exposure, nor was there any way to prevent the control group from also taking supplemental vitamin D. Worse, the researchers used a supplement of only 400 IU of vitamin D3 per day, which other research has shown to increase serum vitamin D levels by only about 7 ng/mL (17.5 nmol/L) in men.[45] Consequently, even with the large numbers of subjects involved, the intervention was too small to show an effect. Although this study is sometimes quoted as a negative finding, the data have little status.

Two recently published cohort studies provide a more accurate assessment. As part of the Finland Health Study, researchers measured the serum vitamin D levels of 4,097 men and women (53% men) and then followed them over 22 years. They found that low vitamin D status predicted future risk of adult-onset diabetes.[46] The Ely Prospective Study in Britain followed an English cohort for ten years. They found that serum vitamin D levels taken at the start, were inversely correlated with glucose and insulin levels ten years later.[47]

In 2010, several studies have just been published that further support vitamin D for preventing diabetes. In a sample from the long-term Framingham Offspring Study, researchers at Tufts University used initial histories to predict serum vitamin D levels and adult-onset diabetes seven years

later. After controlling for all likely confounds, such as family history of diabetes, weight, blood lipids, hypertension, and diet, those in the highest third of the range of predicted serum vitamin D scores, had a 40% reduced risk of developing diabetes.[48]

Looking at the problem from a different angle, Anthony Hanley and colleagues at the University of Toronto examined the vitamin D status of 712 subjects, already identified as at risk of adult-onset diabetes. They found that low vitamin D levels were independently associated with both poor insulin sensitivity and weak pancreatic beta cell function.[49]

Researchers at the Universities of Mississippi and Illinois examined the serum vitamin D status and hemoglobin A1C levels (a strong measure of blood sugar stability) in 9,773 adults aged 35-74, from the 2003-2006 US National Health and Nutrition Examination. They found that vitamin D levels were inversely associated with hemoglobin A1C levels. Their conclusion:

These findings support a mechanistic link between serum vitamin D concentrations, glucose homeostasis, and the evolution of diabetes in a large segment of the U.S. adult population.[50] The above studies are only a tiny sample of research showing that low vitamin D status causes high risk for adult onset diabetes (Type-2 diabetes). Current practices that limit sun exposure, vitamin D supplementation, and that encourage overuse of sunscreens in the US and Canada predispose a large proportion of the population to this preventable disease.

Vitamin D Deficiency Causes Multiple Sclerosis

In medical research the accepted animal model of human multiple sclerosis (MS) is autoimmune encephalomyelitis. Research since the 1990s, shows that supplements of vitamin D3 act as an immune regulator and effectively prevent this disease in mice.[51]

The incidence of human MS also varies with the level of UV-B. There are very few cases at tropical latitudes. Incidence increases in step with increasing latitude and declining UV-B towards the poles. In addition, Norway has the expected high rates of MS inland, but lower rates along the coasts where the local population eat a great number of cold-water ocean fish that are high in vitamin D.[52-54] Case control research in Norway indicates that cod liver oil, which is very high in vitamin D, is protective against MS.[55]

Few prospective studies of vitamin D and MS have yet been published. One recent prospective, nested, case-control study examined the serum Vitamin D of 7 million military veterans. When they compared vitamin D levels of 257 MS patients (before diagnosis) with those of matched controls, they found that vitamin D was inversely related to occurrence of MS.[56] Another recent case-control study compared the serum vitamin D levels of 103 MS patients with controls. Results in women showed, that for every 4 ng/mL (10 nmol/mL) increase in serum vitamin D levels, the risk of MS was reduced by 19%.57 The common denominator in all this MS research is the serum level of vitamin D.

A number of studies on serum levels of vitamin D in MS, show that the great majority of patients, are deficient, including at early stages of the disease.[57] There are yet no controlled trials of Vitamin D as treatment for MS. Nevertheless, recent immunological findings in MS patients show that vitamin D significantly reduces the autoimmune effect of regulatory T lymphocyte cells, whose

role is well known MS pathogenesis.[58] It seems likely that vitamin D treatment for MS would be effective in at least a proportion of cases, and may also prevent MS in people who are at risk.

Vitamin D Deficiency Damages the Brain

Research on the multiple effects of vitamin D deficiency on brain function is in its infancy. I will note only two of the most recent studies that may alert you to the necessity of sufficient sunlight or vitamin D supplementation to maintain the human brain. David Llewellyn, of the University of Exeter, England, and colleagues assessed serum levels of vitamin D in 858 adults who were age 65 or older when the study began in 1998. At the outset, and again after three and six years, they repeated tests of cognitive function: one assessing overall cognition, one focusing on attention and one on executive functions, the cognitive ability to plan, organize and prioritize.

After controlling for a large number of possible confounds, subjects who were clearly deficient in vitamin D, with serum levels of less than 10 ng/mL (25 nmol/L), were 60% more likely to have suffered substantial cognitive decline over the six-year period, and 30% more likely to show declines in executive functions, than those with vitamin D levels above 30 ng/mL (75 nmol/L).[59]

In a study aimed at a more specific diagnosis of brain degeneration, Paul Knekt and colleagues examined the effect of vitamin D deficiency on development of Parkinson's disease, over a 29-year term.[60] The study covered 3,173 men and women aged 50-79 who had been free of Parkinson's in 1980. As part of the Mini-Finland Health Survey, conducted from 1978 to 1980, they had blood samples taken and frozen. These samples were used to 29 years later to determine their initial vitamin D status. Results showed that subjects with serum vitamin D concentrations in the highest quartile, had a 33% reduced risk of Parkinson's compared with those in the lowest quartile.

These studies are the tip of a coming iceberg of research on the effects of vitamin D on brain function. Brain degeneration is subtle and long-term.[61] Once cognition has declined, however, there is little hope of recovery. It seems prudent for everyone to correct any deficit in vitamin D status.

Optimum Levels of Vitamin D

The above sketch of the evidence in favour of vitamin D, covers only a fraction of the diseases it helps to prevent. In addition to the above, there is firm evidence that vitamin D inhibits osteoporosis and hip fractures in the elderly, rheumatoid arthritis, inflammatory bowel disease, Crohn's disease, and a host of skin diseases.[62,63] Even the annual influenza epidemic waxes and wanes to the tune of available vitamin D from sunlight.[64] There is no longer any doubt that Americans, and especially Canadians, need more vitamin D. The controversial questions now concern the optimum level of serum 25-hydroxyvitamin D, and the best combination of sunlight and supplemental vitamin D3 to achieve it year round.

Skeletal effects of vitamin D are the most researched and best known. Michael Holick and colleagues have determined that the lowest threshold level of serum vitamin D for prevention of fractures in osteoporosis is 30ng/mL (75 nmol/L).[65] That is the same minimum level found to be protective in cardiovascular disease.36 For colon cancer, the most protective range of serum vitamin D is 32-41 ng/mL (80-102.5 nmol/L).[34,35] For breast cancer, recent research indicates a reduced risk in women, especially the most vulnerable group, older women, with serum vitamin D levels above 40 ng/mL (100 nmol/L.[66]

In a review of the research on vitamin D sensitive disorders, experts William Grant and Michael Holick concluded that a sufficient and safe range of serum vitamin D for the US and Canada is 33-80 ng/mL (82.5-200 nmol/L). They posit a norm of 54-90 ng/mL (135-225 nmol/L) for people in sunnier climes.[67] Serum vitamin D levels are not considered to become toxic until they exceed 200 ng/mL (500 nmol/L).[68] At the Colgan Institute, we have adopted a range of 40-80 ng/mL (100-200 nmol/L) as a safe and practical range.

Achieving Optimum Vitamin D Levels

How do you get enough vitamin D to reach and maintain the range 40-80 ng/ml (100-200 nmol/L)? Under our current cultural and dietary norms, over 80% of vitamin D is produced by sunlight.1 White Americans currently have an average serum vitamin D level of only 25 ng/mL (62.5 nmol/L). White Canadians currently have an average serum vitamin D level of only 27 ng/mL (67.5 nmol/L).[20-22] Using sunlight to raise these levels to our adopted threshold for prevention of disease,40 ng/mL (100 nmol/L) would require a radical cultural change to more than double current sun exposure.[69]

Black Americans in the US have an average serum vitamin D of only about 13 ng/mL (33 mmol/L) in winter and 20 ng/mL (50 nmol/L) in summer. Together with their potent melanin protection from the sun, they are unlikely to be able to make sufficient change in effective exposure to UV-B to increase their vitamin D to 40 ng/ml (100 nmol/L). Laura Hall and colleagues at the University of California, Davis, have shown that it would require supplements of about 2600IU of vitamin D3 per day, year round, to raise their serum vitamin D to even 30 ng/mL (75 nmol/L).

The seasonality of available UV-B at temperate and cold latitudes makes it difficult to estimate a daily dose. Evolution designed us to ingest and digest all other essential nutrients. But vitamin D, the most ancient hormonal signal of all, is made by light. Until the age of supplementation, very little vitamin D came via ingestion. When sufficient UV-B is available, supplementation is unnecessary, and certainly inferior. Our problem of insufficiency arises from having skin that is genetically unsuited to the reduced UV-B at the higher latitudes where we have now chosen to live. Most Americans and Canadians are suffering from a deficiency of sunshine.

Canada for example was practically uninhabited until the 16th century. Its 400 years of white settlement is only a tiny fraction of the estimated 50,000 years or more of habitation that it would take for Canadians to make the evolutionary response to a changed environment to evolve skin suited to its reduced levels of UV-B.[70]

A large part of Canada is above latitude 45°N, and has insufficient UV-B for most inhabitants for half the year.[23,24] It is also too cold for half the year for exposure of much skin to the sun. People may accumulate a reasonable store of vitamin D from UV-B exposure in the summer months, but the obligate use of vitamin D by the body for multiple purposes every unexposed winter day can exhaust the supply within weeks, because the tissues were not designed by evolution for long-term storage.

In a typical study, Robert Heaney and colleagues at Creighton University gave graded doses of vitamin D3 to 67 healthy men in Omaha (41°N) during 20 weeks of winter and measured their daily use of vitamin D required to maintain the levels they showed during fall before the study began

(average 28.1 ng/mL, 70.3 nmol/L). Note that, although these men appeared healthy, most of them did not have vitamin D levels discussed above as high enough to prevent disease. The oral intake of vitamin D before the study averaged 500IU, and the men required another 3200 IU of vitamin D daily in order to maintain their status during winter.[71] Thus, during winter, they required a total of 3800 IU of vitamin D every day, even to maintain what the research indicates is an insufficient level of serum vitamin D to prevent disease.

There is another difficult cultural problem in trying to decide an optimum daily amount of vitamin D. We now live a lot longer than designed for by evolution, but our cultural imperative is to extend lifespan as far as possible The ability of the skin to produce pre-vitamin D declines with aging.[72] People in their seventies may have less than half the pre-vitamin D production capacity of people in their twenties.

There are numerous detrimental changes by the time we reach age 50, the limit of reproductive efficiency for which evolution designed us, that also reduce Vitamin D production, or require higher vitamin D levels to offset them. Prominent are hyperparathyroidism, and declines in the hormone cascade, calcium metabolism, and renal function.[73-75] Discussion of these changes is beyond the scope of this short paper. Suffice to say here that people over 45 need greater amounts of supplemental vitamin D than young people to achieve the level of serum vitamin D that the studies discussed above indicate is sufficient to prevent disease.

In estimating an optimal daily dose for vitamin D, we have to allow for the above research on requirements, for polyglot racial heritage, for the latitude at which people live, for their habitual sun exposure without sunscreen, and for their age. Because sufficient sun exposure is unlikely to occur for many people, the answer lies in food and vitamin D supplements. These matters are given little consideration in formation of current government recommendations for vitamin D.

The high levels of vitamin D deficiency in the US and Canada indicate that current health policies regarding supplemental vitamin D intake and the fortification of milk and other staple foods with vitamin D are not based on current science, and are exposing the public to high risk of multiple diseases. In fact, since the changes in US and Canadian health policy in the 1980s, to recommend less sun exposure and use of sunscreens, and the continuing deceptive promotion of sunscreens for commercial reasons, vitamin D status of the population has declined further during this century.[76]

The current US and Canadian Tolerable Upper Limit for vitamin D intake is 2000 IU (50ug), which is based on early dosage studies and the appearance of hypercalcemia, the accepted indicator of vitamin D toxicity. The No Observed Adverse Effect Level was derived from two small, methodologically flawed studies as 2400 IU,[77,78] which was then divided by an arbitrary "uncertainty factor" of 1.2 to yield the 2000 IU Tolerable Upper Limit. Numerous scientists have pointed out that such a basis for an extremely important health recommendation is incomprehensible.

The current government recommendation for Vitamin D is based on a tiny sample the science of 50 years ago, which bears little relation to modern biochemistry and shows little understanding of the biochemistry of living systems. Recent dosage studies using modern pharmacokinetic methods, show that in people who are vitamin D deficient, incremental increases in vitamin D3 supplementation by 40 IU per day increase serum 25(OH)D by approximately 0.4 ng/mL (1.0 nmol/L).[79] But this increase occurs only while vitamin serum 25(OH)D levels are low. As serum levels rise above

40 ng/mL (100 nmol/L) the increase in serum vitamin D begins to plateau.[80,81]

Depending on the individual's age, health, initial serum level of vitamin D, sun exposure during supplementation, and cold water ocean fish in the diet, to achieve a serum level of 40 ng/ml (100 nmol/L) may require a daily dose of Vitamin D3 of 3,000-5,000 IU. As there is no reported toxicity at this level, it seems reasonable to consider the plateau in serum vitamin D levels as a response by the body to reduce the bioavailability of oral vitamin D that indicates sufficiency in the tissues.

We are designed to obtain vitamin D from interaction with sunshine. It seems logical therefore to also relate supplementation to the supply of vitamin D created by interaction with the sun. Research has shown repeatedly that approximately 30 minutes of whole body exposure of a light-skinned, young adult to summer sun in the Southern US, will manufacture enough pre–vitamin D to provide at least 10,000IU of vitamin D3. People with abundant exposure to sunlight may have serum vitamin D levels of 60 ng/mL (150 nmol/L), yet show no vitamin D toxicity.[82] On average, this level would be equivalent to a daily vitamin D3 supplement of 4000 IU. This represents the center point of the range derived above from pharmacokinetic studies.

I have sketched a small part of the evidence that vitamin D, our oldest hormonal signal, is essential in considerable amounts, every day, for multiple bodily systems. I have outlined how it is available best from the interaction of bare skin with UV-B in sunlight. Because of the certain deficiency of UV-B at temperate and northern latitudes, in the US and especially in Canada, adequate vitamin D to prevent multiple diseases can be obtained only by oral supplements of vitamin D3.

Current government recommendations for supplementation, and current fortification of foods with vitamin D are obsolete and ineffective, shown decisively by the high incidence of serum vitamin D deficiency in the US and Canada. The evidence reviewed indicates, that in order to prevent numerous diseases including cancers, cardiovascular diseases, adult-onset diabetes, multiple sclerosis, and senility, adults require a daily supplement of 3,000-5,000 IU of vitamin D3 to raise serum vitamin D levels into a safe and healthy range of 40-80 ng/mL (100-200 nmol/L).

Changing Health Policy for Vitamin D

Vitamin D supplementation is simple and inexpensive. The research above leaves little doubt that Vitamin D sufficient to bring serum vitamin D into the range 40-80 ng/mL (100-200 nmol/L) would save many lives. It would also cut health care costs substantially, especially for seniors. The questions remain, exactly how many lives vitamin D supplementation would save per year?

William Grant has recently compiled convincing estimates that about 400,000 American lives could be saved by changes in health policy to persuade the public to embrace vitamin D. His paper, however, suggests increasing sun exposure to more than double the current level as part of the solution.[69] As he acknowledges, this cultural change could increase the incidence of UV-B related deaths, primarily from skin cancers, by up 12,000 deaths each year. Such a likely effect makes any proposal involving sunlight or, by association, vitamin D, highly controversial, no matter how many lives it might save. Especially so, because the incidence of skin cancer in light-skinned populations continues to increase.[82]

My proposal does not mandate any obligatory change in sun exposure. In fact, in many parts of the US and Canada, such a cultural change would not occur because the temperature for up to half the

year effectively discourages uncovering the skin. Also, it is highly unlikely that people with the dark skins of more recent African descent, could absorb sufficient UV-B at latitudes above 50°N, no matter how much exposure they receive. In contrast, taking supplemental oral vitamin D poses no risk of melanoma, or other skin cancers, and offers a non-toxic, inexpensive, alternative, easily adjusted for latitude, weather, and skin color.

Unfortunately, convincing governments to change recommended intakes of nutrients hinges more on political probity than merit. A potent example is the recent prestigious report on vitamin D by the World Health Organization, International Agency for Research on Cancer (IARC), used as expert testimony by both the US and Canadian governments. Citing 1,370 of the research papers on the subject, analysed by 12 scientists specializing in Vitamin D from research institutions worldwide, it provides a comprehensive summary of the evidence. The IARC Report did not recommend increasing the use of vitamin D to prevent cancer or all-cause mortality.[82] It came to this conclusion despite having among its scientists some whose research I have quoted herein, that are in favour of increasing vitamin D to combat disease.

Numerous faults in the IARC Report are well detailed elsewhere by 14 acknowledged experts on vitamin D.[83] I will confine remarks here to other matters, some of what appear to be internal inconsistencies in the report. In its conclusions, the IARC report states:

Setting a lower limit of "adequate" serum 25-hydroxyvitamin D levels at 20 or 30 ng/mL is currently inappropriate since there are no results from randomised trials suggesting that maintenance of such "adequate" serum 25-hydroxyvitamin D levels actually prevents any cancer and any other chronic condition (p143).[82]

This conclusion is inconsistent with their own analysis, which quotes the well-known Nebraska 4-year randomized trial, in which women received 1100 IU of vitamin D3 plus calcium, calcium alone, or placebo:

A 4-year, population-based, double-blind, randomised placebo-controlled trial of vitamin D and calcium was conducted with the primary outcome being fracture incidence, and the principal secondary outcome cancer incidence (Lappe et al.,2007) The subjects in the study were 1,179 community-dwelling women randomly selected from the population of healthy postmenopausal women aged 55 and over in a 9-county rural area of Nebraska, USA. Subjects were randomly assigned to receive each day 1.4-1.5 g supplemental elementary calcium alone (Ca-only), supplemental calcium plus 27.5 µg vitamin D3 (Ca + D), or placebo. When analysed by intention to treat, cancer incidence was lower in the (Ca + D) women than in the placebo control subjects (P <0.03). Authors concluded that improving calcium and vitamin D nutritional status substantially reduces all-cancer risk in postmenopausal women (Lappe et al.,2007) (p114).[82]

Their analysis also plots an almost linear dose-response curve showing that, as vitamin D increases up to a serum level of 40 ng/mL the risk of colorectal cancer declines. This further appears inconsistent with the conclusion on p143 quoted above.

Thus, based on multiple studies of circulating 25-hydroxyvitamin D and colorectal cancer risk, individuals in the high quartile or quintile of 25-hydroxyvitamin D had about half the risk of colorectal cancer as did those in the lowest group. The dose-response appears fairly linear up to a

25-hydroxyvitamin D level of at least 35-40 ng/mL (p94).[82]

Another inconsistency with the conclusion again refers to colorectal cancer:

Observational studies provide evidence of a decreased risk of colorectal cancer associated with higher serum 25-hydroxyvitamin D. This evidence is supported by a decrease in colonic adenoma with higher serum 25-hydroxyvitamin D (p105).[82]

Colorectal Cancer is the third most frequent cancer in Canada and kills over 9,000 people per year.84 Colorectal cancer is the second most frequent cancer in the US with over 140,000 new cases per year and over 51,000 deaths.[85] If simple, non-toxic supplementation with vitamin D could prevent even 10% of these deaths, it would save 6,000 lives every year.

Further inconsistencies in the IARC Report question the safety of Vitamin D supplementation. The report concludes:

There is no data available on the health hazards of long-term maintenance of high 25-hydroxyvitamin D serum levels in healthy subjects over long periods (p148).[82]

This conclusion seems inconsistent with:

Studies on the safety intakes of high dose vitamin D (i.e., 100 µg up to 1,250 µg per day) were done over short periods, from a few weeks to 6 months, and rarely for one year or more (Vieth, 1999;SCF, 2002; Heaney et al.,2003; Kimball et al.,2007; Vieth et al.,2004). Hypercalcemia was not found in these studies despite 25-hydroxyvitamin D levels that could reach 155 ng/mL over 6 months (Kimball et al.,2007) (p21).[82]

Note that the dosages of vitamin D in these studies range from a low point of, 4000 IU up to very high doses of 50,000 IU per day, and the periods of supplementation are up to one year. Yet the primary indicator of vitamin D toxicity, hypercalcemia, did not occur.

There are three other problems with the IARC's conclusion of a lack of data on safety quoted above from p148 of their report. First, no vitamin D researcher that I know is advocating a change in health policy that would raise the recommended intake of vitamin D intake above even 5,000 IU per day, which near the bottom of the range found to be non-toxic. Second, as I noted earlier, after reaching a level of 40 ng/mL, (100 nmol/L) serum vitamin D begins to plateau.[80,81] Larger doses are likely unnecessary. Third, most of the research quoted herein in favour of a revision of policy, suggests that 40ng/ml (100 nmol/L) is a sufficient level of serum vitamin D to prevent disease, a level that is again near the bottom of serum levels achieved with supra-physiological doses of vitamin D given to test for toxicity. In sum, existing safety data appear adequate to support a change in policy to increase the Tolerable Upper Limit of daily intake from its current 2,000 IU to at least 5,000 IU, and to specify an average serum level of vitamin D of 40 ng/mL (100 nmol/L) to prevent disease.

Instead, the IARC Report affirms the status quo, which may have been the least offending conclusion, then calls for more randomized trials, a call most scientists would applaud. In affirming the status quo for vitamin D, however, the need for diplomacy may have outweighed the science. Both the US and Canada are currently considering revisions of policy regarding vitamin D. For the better health of North America, I hope they give more weight to the 30 years of research reviewed

herein than to summary reports of committees, no matter how high their prestige.

References

1. Holick MF: High prevalence of vitamin D inadequacy and implications for health. Mayo Clin Proc, 2006;81:353–373.
2. Nordin C, Morris HA. Osteoporosis and vitamin D. J Cellular Biochemistry, 1992;49:19-25.
3. Albert MR, Ostheimer KG. The evolution of current medical and popular attitudes toward ultraviolet light exposure: part 3. J Am Acad Dermatol 2003; 49:1096-1106.
4. Albert MR, Ostheimer KG. The evolution of current medical and popular attitudestoward ultraviolet light exposure: part 2. J Am Acad Dermatol 2003; 48:909-918.
5. Colgan M. Bare naked feet and human performance. Part 2. Colgan Institute Newsletter, July/August 2010.
6. Bramble D, Lieberman DE. Endurance running and evolution of Homo. Nature, 2004;432:345-352.
7. Montagna, W. The evolution of human skin. Journal of Human Evolution, 1985;14: 3-22.
8. Jablonski NG, Chaplin G. The evolution of human skin coloration. J Hum Evol 2000;39:57-106.
9. Chaplin G, Jablonski NG. Vitamin D and the evolution of human depigmentation. Am J Phys Anthropol 2009; 139:451-61.
10. US Centers for Disease Control http://www.cdc.gov/cancer/skin/statistics/race.htm. Accessed 31 July 2010.
11. Baroncelli G, Bereket A, El Kholy M, et al. Rickets in the Middle East: role of environment and genetic predisposition. J Clin Endocrinol Metab, 2008;93:1743-1750.
12. Pettifor JM. Nutritional rickets: deficiency of vitamin D, calcium, or both? Am J Clin Nutr 2004;80:1725S-1729S.
13. Kimball S, Fuleihan GEH, Vieth R. Vitamin D: A growing perspective. Crit Rev Clin Lab Sci, 2008;45:339-414.
14. El-Hajj Fuleihan G Vitamin D deficiency in the Middle East and its health consequences. In Holick MF (ed) Vitamin D: Physiology, Molecular Biology, and Clinical Applications - Second Edition. Humana Press, 2009.
15. Grant WB. Lower vitamin-D production from solar ultraviolet-B irradiance may explain some differences in cancer survival rates. J Natl Med Assoc 2006; 98:357-364.
16. Harris SS. Vitamin D and African Americans. J Nutr 2006; 136:1126-1129.
17. Grant WB. An estimate of premature cancer mortality in the U.S. due to inadequate doses of solar ultraviolet-B radiation. Cancer 2002; 94:1867-1875.
18. Garland CF, Garland FC, Gorham ED, Lipkin M, Newmark H, Mohr SB, et al. The role of vitamin D in cancer prevention. Am J Public Health 2006; 96:252-261.
19. Grant WB, Garland CF. The association of solar ultraviolet B (UVB) with reducing risk of cancer: multifactorial ecologic analysis of geographic variation in age-adjusted cancer mortality rates. Anticancer Res 2006; 26:2687-2699.
20. Harris SS. Vitamin D and African Americans. J Nutr, 2006;136:1126-1129.
21. Looker AC, Pfeiffer CM, Lacher DA, Schleicher RL, Picciano MF, Yetley EA. Serum 25-hydroxyvitamin D status of the US population: 1988–1994 compared with 2000–2004. Am J Clin Nutr, 2008;88:1519-1527.
22. Health Canada Vitamin D levels 2007-2009. http://www.statcan.gc.ca/pub/82-625-x/2010001/article/11137-eng.htm. Accessed 1 August 2010.
23. Rucker D, et al. Vitamin D insufficiency in a population of healthy western Canadians. Can Med Assoc J, 2002;166:1517-1524.
24. Webb AR, Kline L, Holick MF. Influence of season and latitude on the cutaneous synthesis of vitamin D3: exposure to winter sunlight in Boston and Edmonton will not promote vitamin D3 synthesis in human skin. J Clin Endocrinol Metab 1988;67:373-378.
25. Gartner LM, Greer FR. Prevention of rickets and vitamin D deficiency: new guidelines for vitamin D intake". Pediatrics, 2003;111(4 Pt 1):908–910.
26. Reichrath J. Vitamin D and the skin: an ancient friend, revisited. Exp Dermatol, 2007; 16(7):618-625.
27. Mohr SB. A brief history of vitamin D and cancer prevention. Ann Epidemiol. 2009 (2):79-83.
28. Garland CF, et al. The role of vitamin D in cancer prevention. Am J Public Health. 2006:96(2):252-261.
29. Holick MF. Vitamin D deficiency. N Engl J Med;2007;357:266-281.
30. Grant WB, Mohr SB Ecological studies of ultraviolet B, vitamin D and cancer since 2000. Ann Epidemiol, 2009;19(7):446-454.
31. Grant WB. An estimate of premature cancer mortality in the U.S. due to inadequate doses of solar ultraviolet-B

radiation. Cancer, 2002;94(6):1867-1875.
32. Gissel T, Rejnmark L, Mosekilde L, Vestergaard P. Intake of vitamin D and risk of breast cancer--a meta-analysis. Journal of Steroid Biochemistry and Molecular Biology, 2008;111(3–5):195–199.
33. Mohr SB, et al. Relationship between low ultraviolet B irradiance and higher breast cancer risk in 107 countries. Breast J,2008;14(3):255-260.
34. Hartman TJ, Albert PS, Snyder K, et al. The association of calcium and vitamin D with risk of colorectal adenomas. Journal of Nutrition, 2005;135(2):252–259.
35. Garland CF, et al. Serum hydroxyvitamin D and colon cancer: an eight-year prospective study. Lancet, 1989;334:1176-1178.
36. Giovannucci E, et al. 25-hydroxyvitamin D and risk of myocardial infarction in men: A prospective study. Arch Intern Med, 2008;168(11):1174-1180.
37. Mookherjee N, Rehaume LM, Hancock RE. Cathelicidins and functional analogues as antisepsis molecules. Expert Opin Ther Targets 2007;11:993-1004.
38. Martin GS, Mannino DM, Eaton S, Moss M. The epidemiology of sepsis in the United States from 1979 through 2000. N Engl J Med 2003;348:1546-1554.
39. Health Canada http://www.health.gov.on.ca/english/providers/program/critical_care/docs/ccs_sepsis _p_ 01_ 2007 1001.pdf . Accessed 31 July 2010.
40. Grant WB. Solar ultraviolet-B irradiance and vitamin D may reduce the risk of septicemia. Dermato-Endocrinology, 2009;1:25-30.
41. Norman AW, Frankel JB, Heldt AM, Grodsky GM: Vitamin D deficiency inhibits pancreatic secretion of insulin. Science, 1980;209:823–825.
42. Scragg R, Sowers M, Bell C: Serum 25-hydroxyvitamin D, diabetes, and ethnicity in the Third National Health and Nutrition Examination Survey. Diabetes Care, 2007;27:2813–2818.
43. Hypponen E, Power C: Vitamin D status and glucose homeostasis in the 1958 British birth cohort: the role of obesity. Diabetes Care, 2006;29:2244–2246.
44. de Boer IH, et al. Calcium plus vitamin D supplementation and the risk of incident diabetes in the Women's Health Initiative. Diabetes Care, 2008;31:701–707.
45. Heaney RP, et al. Human serum 25-hydroxycholecalciferol response to extended oral dosing with cholecalciferol. Am J Clin Nutr, 2003;77:204–210.
46. Mattila C, et al. Serum 25-hydroxyvitamin D concentration and subsequent risk of type 2 diabetes. Diabetes Care, 2007;30:2569–2570.
47. Forouhi NG, et al. Baseline serum 25-hydroxy vitamin D is predictive of future glycemic status and insulin resistance: the Medical Research Council Ely prospective study 1990–2000. Diabetes, 2008;57:2619–2625.
48. Enju L, et al. Predicted 25-hydroxyvitamin D score and incident type 2 diabetes in the Framingham Offspring Sudy Am J Clin Nutr, 2010;91:1627-1633.
49. Kayaniyil S, et al. Association of vitamin D with insulin resistance and βcell dysfunction in subjects at risk for Type 2 diabetes. Diabetes Care, 2010;33:1379-1381.
50. Kositsawat J, et al. Association of A1C Levels With Vitamin D Status in U.S. Adults.Data from the National Health and Nutrition Examination Survey. Diabetes Care, 2010;33:1236-1238.
51. Cantorna MT, Hayes CE, DeLuca HF. 1,25-Dihydroxyvitamin D3 reversibly blocks the progression of relapsing encephalomyelitis, a model of multiple sclerosis. Proc Natl Acad Sci U S A ,1996;93:7861-7864.
52. Hayes CE, et al. Vitamin D and multiple sclerosis. Proc Soc Exp Biol Med, 1997;216(1):21-27.
53. Kampman MT, Brustad M. Vitamin D: a candidate for the environmental effect in multiple sclerosis - observations from Norway. Neuroepidemiology, 2008;30(3):140-146.
54. Milo RK, Kahana E. Multiple sclerosis: geoepidemiology, genetics and the environment. Autoimmun Rev. 2010;9(5):A387-A394.
55. Kampman MT, Wilsgaard T, Mellgren SI. Outdoor activities and diet in childhood and adolescence relate to MS risk above the Arctic Circle. J Neurol, 2007;254:471–477.
56. Munger KL, et al. Serum 25-hydroxyvitamin D levels and risk of multiple sclerosis. JAMA, 2006;296:2832–2838.
57. Kragt JJ, et al. Higher levels of 25-hydroxyvitamin D are associated with a lower incidence of multiple sclerosis only in women. Mult Scler, 2009;15:9–15.
58. Pierrot-Deseilligny C, Souberbielle JC. Is hypovitaminosis D one of the environmental risk factors for multiple sclerosis? Brain, 2010;133(Pt 7):1869-1888.

