

# Vitamin D Could Anti-Age You

Higher levels are associated with better health  
and the equivalent of 5 more years of life

By Will Block

*Mad dogs and Englishmen go out in the midday sun.*

— Noel Coward

We have a love-hate relationship with the sun, don't we? As much as we may worship the sun for its comforting, life-giving light and warmth, and as much as our world literally revolves around it, we have nonetheless learned to fear its power to harm us—even kill us. Skin cancer is no laughing matter, and we are right to take precautions against excessive exposure to ultraviolet radiation, the “light” we cannot see.



The paradox of the sun is that, even as it's burning our skin and setting us up for cancer, it's also causing something almost magical to happen in that same skin: the synthesis of vitamin D. This formerly rather boring vitamin, which helps build strong bones and teeth and prevents rickets, is rapidly achieving stardom in the nutritional world, as scientists find out more and more about its ability to help prevent cancer and reduce our overall risk for death. (For more on these dramatic developments, see [“Vitamin D and Calcium Combat Cancer”](#) and [“Vitamin D Might Prolong Your Life”](#) in the August and November 2007 issues, respectively, of *Life Enhancement*.)

## Vitamin D—Sun or Supplement?

Vitamin D is the only nutrient we get from the sun—all the rest must be obtained from food or supplements. Of course, vitamin D can also be obtained from food or supplements, and for many people—mainly shut-ins or those who live in far northern (or far southern) latitudes and who don't get out much—that is the principal way, or the only way, they get their daily dose.

Actually, though, it's not that easy to get adequate vitamin D from food, because so few foods contain appreciable amounts of it. Unless you eat a good deal of fatty fish (such as salmon), fish-liver oils, or fortified milk or

cereals, you probably don't get enough vitamin D, and you should supplement to make up for the deficit.

The best supplemental form in which to take the “sunshine vitamin” is vitamin D3 (cholecalciferol), the same compound that's synthesized in your skin by ultraviolet radiation; a related compound, vitamin D2 (ergocalciferol), obtained mainly by synthesis from a plant precursor, is not recommended. Cholecalciferol is converted in your liver to 25-hydroxycholecalciferol [aka 25-hydroxyvitamin D, or 25(OH)D], the form in which vitamin D is most easily measured in the blood. This compound is converted, mainly in your kidneys, to 1,25-dihydroxycholecalciferol [aka 1,25-dihydroxyvitamin D, or 1,25(OH)2D], the most biologically active form of the vitamin.

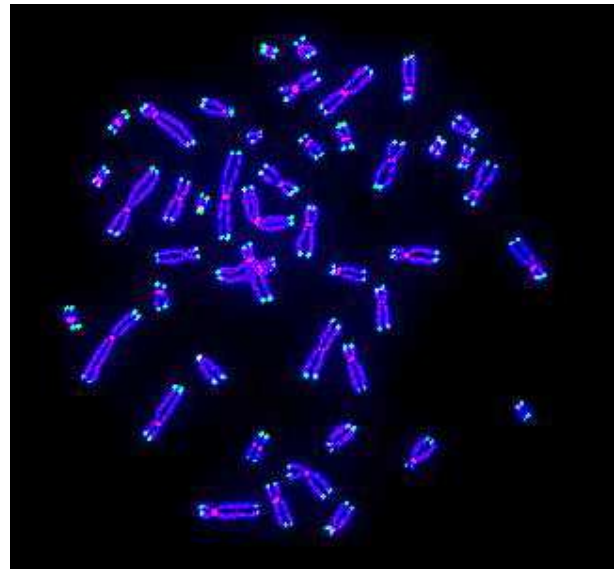
When Is “Enough” Not Nearly Enough?

How much vitamin D should you take? Most multivitamin formulations contain 400 IU, which is 100% of the RDA. But RDA values were established to prevent deficiency disease, not to promote optimal health; thus they represent a lower limit on what we need. And most Americans don't even get *that* much—the median adult intake is only about 230 IU/day. Clearly, an awful lot of us are not getting much sun exposure, which can easily give us all the vitamin D we need—except during winter, especially at latitudes above about 40° North. (For a map of the USA showing 40° N, see [page 25 of the August 2007 issue](#).)

As discussed in the articles mentioned above, scientists are coming to realize that much larger amounts of vitamin D are beneficial, and perhaps essential, for optimizing our health. Some of the leading authorities in this field are now recommending at least 2000 IU/day, while recognizing that 4000 IU/day or more is probably better. It's important to note that there is no evidence of any harm from taking up to 10,000 IU/day in supplemental form. (The daily amount obtained via sun exposure is often much higher than that.)

A Major Discovery—Really!

New evidence that high levels of vitamin D are beneficial comes from a study of 2160 women, aged 18–80, in the United Kingdom.<sup>1</sup> The women, who were representative of the general British population, were being studied in regard to a variety of health issues, and one group of researchers analyzed data related to their blood levels of vitamin D (regardless of its source). *They found a strong correlation between the women’s vitamin D levels and their leukocyte telomere length.*



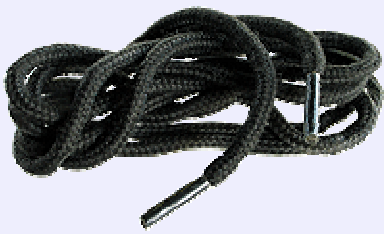
*A set of human chromosomes (seen with fluorescence microscopy) during the process of cell division, when the newly replicated DNA molecules are still linked, forming pairs called dyads. The telomeres—two per DNA molecule—are the bright spots at the ends.*

Wait! Don’t go away! You’ll see in a moment—after an overview of what that term means—why this is important in terms of our health and longevity. First of all, *leukocytes* are white blood cells—the backbone, so to speak, of the body’s immune system, whose primary function is to seek and destroy foreign invaders. Like all our other cells, leukocytes contain our chromosomes, which consist of DNA and associated proteins. The DNA molecule encodes all of our genes and thus transmits all our hereditary information from one generation of cells to the next during the process of cell division.

Because leukocytes divide more often than most other cells, especially when there is chronic inflammation, they’re convenient to use for studies involving cell division—but the results are applicable, in principle, to cells in general. In this case, the study pertains to *telomeres*, which are tiny terminal segments at each end of the enormously long DNA molecule. Although they carry no genetic information, they serve a vital role by protecting the DNA from damage during cell division.\* (This is explained in the sidebar below, and now would be a good time to read it.)

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\*Telomeres also prevent the ends of DNA molecules from joining to each other to form loops, or from joining end-to-end with the DNA in other chromosomes—disastrous scenarios both.



## Of Shoelaces, Rattlesnakes, and Telomeres

Little things matter. Think how much your life has been improved by aglets, those tiny plastic thingies on the ends of your shoelaces. Without them, the laces would fray, and so would your temper.

Angry people have shorter life expectancies than cool people. You can thank your aglets for helping you live longer.

You can also thank your telomeres, which are the terminal segments of the double-stranded DNA molecules of your chromosomes. Telomeres act like molecular “aglets,” preventing the chromosomal DNA from fraying during the process of replication, which precedes every cell division.

In replication, the two strands of each DNA molecule are pulled apart, from one end to the other, like a helical zipper being unzipped. While this is occurring, each exposed strand serves as a template for the synthesis of a new strand, which is a mirror image of the original. As the two sets of strands are zipped back together, they form two identical DNA molecules, one for each of the two daughter cells that the mother cell is about to become through cell division. (They’re never called father or son cells—that’s sexism!)

The delicate processes of DNA unzipping and zipping are performed by enzymes, which are large, bulky proteins. The sheer size of these molecular “machines” gets in their way as they approach the end of the DNA molecule, preventing them from replicating the strands all the way to their ends. It’s like a tape recorder, which can’t record on the first or last few inches of a cassette tape.

Enter the telomeres. By extending the length of the DNA strands with their genetically blank material, they give the enzymes enough space to work with in replicating the genetically *useful* material all the way to *its* end, thus preserving every last gene. Were it not for this protective action, some of that material would be lost in each replication, resulting in a gradual deterioration of cell function, and of our health.

It's a great system—but there is a catch. Because the enzymes cannot replicate the telomeres all the way to *their* ends, the telomeres lose an end segment with each replication, becoming shorter with time. They thus act as a kind of sacrificial molecular “clock,” telling (very roughly, because there's a lot of natural variation) our age in terms of cell divisions. It's like a rattlesnake's tail in reverse. There a new segment is *added* with each successive molt (usually several times a year), so the growing tail length signals the snake's age in terms of molts.

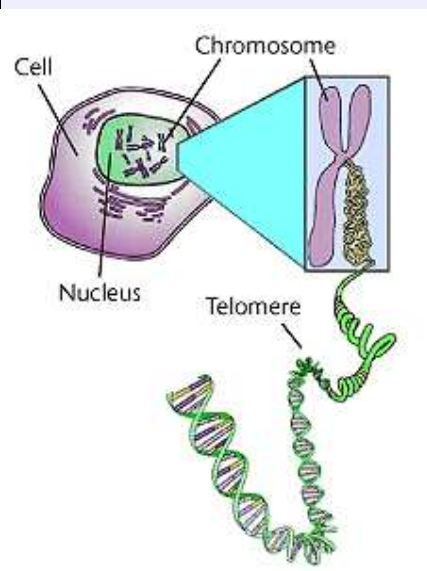


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This process cannot go on indefinitely, because our telomeres are only so long. It turns out that they're limited to about 50 cell divisions, on average, before they become too short to function any more (this is called the Hayflick limit after its discoverer, the microbiologist Leonard Hayflick). When that happens, our chromosomes are in serious trouble—and so are

we. The cells in question will no longer divide properly (or at all), and they may become inactive, senescent, or cancerous.



But wait—there is still hope, in the form of a ribonucleoprotein called *telomerase*, whose enzymatic function is to *extend* the length of telomeres, thereby maintaining their function—and thus, perhaps, extending our lives. That's the good news. The bad news is that appreciable amounts of telomerase are generally found only in sex cells, stem cells, and . . . cancer cells.

In a cruel irony, telomerase becomes activated in most incipient cancer cells when their telomeres become very short through frequent cell division. By continually regenerating the ends of these continually shortening telomeres, the telomerase prevents the cells from dying the natural (and necessary) death of almost other cells, and it enables their runaway proliferation. They become literally immortal, able to live forever unless we kill them.

If scientists could learn how to block telomerase in cancer cells, they might be able to fight cancer by allowing the cells to age and die. But could they

learn how to activate telomerase in *healthy* cells without incurring unintended consequences, such as increasing our vulnerability to cancer? That's the crucial question; there is currently no answer.

## Telomeres—The Short and the Long of It

*Leukocyte telomere length* (LTL) is related to disease and aging, although the relationship is complex and not well understood. Longer is better with LTL, and shorter is worse—except with cancer cells, where we *want* the telomeres to get shorter until they “burn out.” Unusually shortened (but not, alas, terminally shortened) telomeres have been found in many cancers, including those of the pancreas, bone, prostate, bladder, lung, kidney, head, and neck.

Short LTL has been found to be associated with a variety of diseases—notably inflammatory diseases, autoimmune diseases, and vascular diseases. Lack of exercise, obesity, insulin resistance, and smoking are also associated with accelerated telomere shortening over time. (Telomeres, by the way, do not shorten in tissues in which the cells do not continually divide, such as the heart.)

Short LTL has also been found to be associated with a shortened life expectancy, but it's not known whether shorter telomeres are a *cause* of aging or a *consequence* of aging, like wrinkles. Put another way, do longer telomeres slow the aging process, or does the aging process cause shorter telomeres? We don't know the answer to this question, or to many others regarding telomeres. For example, why do humans have much shorter telomeres than many short-lived species, such as mice?

If shorter telomeres *are* a cause of aging, they share that dubious distinction with other causes, such as *glycation*—the chemical reactions of glucose with proteins to form advanced glycation end products (AGEs), which are deleterious—and *oxidative stress*, which is the cumulative, deleterious action of reactive oxygen species. Studies have shown that short LTL is associated with various indexes of oxidative stress and inflammation.

## Why Vitamin D and LTL Are So Important

Now you see why the discovery that vitamin D appears to keep LTL longer is so important. Leukocytes have vitamin D receptors, and if the leukocytes' telomeres are kept long by vitamin D, these vital immune-system cells will thrive and help fend off disease. But if there is insufficient vitamin D for

this purpose, their function may deteriorate, making us more vulnerable to disease and death. It's probably no coincidence that low levels of vitamin D are associated with autoimmune diseases, such as multiple sclerosis, rheumatoid arthritis, and type 1 diabetes.



Nor does it appear coincidental that, as we age, both our LTL and our vitamin D levels decline, while there is an increase in the incidence of systemic inflammation, which underlies many degenerative diseases. Vitamin D acts as an inhibitor of inflammation. Yet another clue to the connection is that C-reactive protein (CRP), a marker of systemic inflammation and a predictor of cardiovascular disease, is inversely related to both vitamin D and LTL: higher vitamin D levels or longer LTL mean lower CRP levels, and vice versa.

Higher Vitamin D = Longer LTL = Slower Aging

In the British study, the women were divided into tertiles (three equal-sized groups: top, middle, and bottom) according to their blood vitamin D levels, measured as 25-hydroxyvitamin D. The data obtained were corrected for possible confounding factors, such as age, body mass index, fasting insulin and serum leptin concentrations, smoking status, physical activity status, menopausal status, use of hormone replacement therapy, and the season of the year (which is important because of the changing amounts of sunshine).

The analysis showed that higher vitamin D levels were correlated with higher LTL values, suggesting a protective effect of vitamin D. (Furthermore, lower CRP levels were correlated with higher vitamin D and higher LTL.) Still wide open, however, is the question of whether or not vitamin D *causes* telomere length to be preserved. It's possible that something else does that—something that also alters the way vitamin D is synthesized and metabolized in the body, thereby creating the correlation. We just don't know.

In any case, it looks as though those Englishwomen in the top vitamin D tertile had been spending time in the midday sun (probably in the company of Englishmen, and possibly even with a mad dog or two)—or perhaps they had been taking plenty of supplemental vitamin D. Whatever the source of their vitamin D, the benefit to their expected longevity was substantial: the authors calculated that the difference in LTL between the women in the top

tertile and those in the bottom tertile was *equivalent to 5 years of telomeric aging, which is to say, 5 years of chronological aging. In other words, people with higher vitamin D levels seem to age more slowly.*

### This Is No Pie in the Sky

That's remarkable! One is tempted to say that it's D-lightful, D-licious, and D-lovely (with apologies to Cole Porter), but that would be silly, wouldn't it? The authors, being of a more sober temperament, stated,<sup>1</sup>

In conclusion, our study provides evidence that a longer LTL is associated with increased serum vitamin D concentrations in women. Although both LTL and serum vitamin D concentrations decrease with age and are thus possible markers of aging in general, we have shown that the positive association between LTL and vitamin D concentrations is independent of age and many other covariates. . . . our data suggest another potential benefit of vitamin D—on the aging process and age-related disease.

That's not bad for a nutrient that basically falls out of the sky for free. And for those who don't get outdoors that much or who are concerned about skin cancer, it can be obtained in large quantities, at modest cost, as a supplement. That's not bad either. It may be D bargain of your life.

### Reference

1. Richards JB, Valdes AM, Gardner JP, Paximadas D, Kimura M, Nessa A, Lu X, Surdulescu GL, Swaminathan R, Spector TD, Aviv A. Higher serum vitamin D concentrations are associated with longer leukocyte telomere length in women. *Am J Clin Nutr* 2007;86:1420-5.

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