

The Truth About Vitamin D: Fourteen Reasons Why Misunderstanding Endures

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During the 1960s, a large number of studies began to point to the idea that estrogen therapy might ease the pangs of menopause. In a best selling book called *Feminine Forever*, a Brooklyn gynecologist named Robert Wilson argued that menopause was an illness rather than a natural state associated with aging. Soon, an increasing number of older women began to take supplemental estrogen in an effort to replace the hormones that their own bodies had stopped secreting. The treatment, known as hormone replacement therapy or HRT, became one of the most popular medical treatments in America.

The American Heart Association, the American College of Physicians, and the American College of Obstetricians and Gynecologists all agreed that a sufficient number of studies had been done to prove that HRT was unequivocally helpful in helping older women ward off heart disease and osteoporosis. By 2001, 15 million women were taking HRT, including 5 million elderly women.

Then, in 1998, a clinical trial concluded that estrogen therapy actually increases the likelihood that women with heart disease will suffer a second heart attack. It was followed by a trial in 2002 which concluded that HRT puts postmenopausal women at a greater risk for heart disease, stroke, blood clots, breast cancer and even dementia. Suddenly, It became painfully clear that HRT may offer a benefit to women who begin to use it early in life, but for those who start the treatment in their later years, it can be very dangerous.^[1]

Gary Taubes writes in *The New York Times Magazine*, “The question of how many women may have died prematurely or suffered strokes or breast cancer

because they were taking a pill that their physicians had prescribed to protect them against heart disease lingers unanswered. A reasonable estimate would be tens of thousands.”^[1]

This HRT story, which Harvard epidemiologist Jeffrey Avorn calls the “estrogen debacle” and a “case study waiting to be written” is a glaring example of how even the most widely held medical beliefs can turn out to be wrong. The story is fraught with biased studies, overconfident clinicians, and researchers who failed to think critically.

Most alarming, however, is the fact that the medical community is currently oblivious to yet another public health disaster of epic proportions - one that is affecting the entire population. In an effort to curb chronic disease, well-intentioned researchers are promoting vitamin D, a substance that, according to recent molecular modeling research, can act as an immunosuppressive steroid. Studies which incorrectly interpret the reason for low vitamin D in patients with chronic disease have been seized upon by the media, and form the basis of massive advertising campaigns – which, along with ill-informed recommendations by doctors and researchers, have created a perfect storm of misunderstanding and bad advice.

What follows are fourteen pieces of a puzzle that, when complete, reveal a massive misunderstanding of the actions of vitamin D.

1. Vitamin D is not a vitamin; it is an immunosuppressive steroid.
2. The vast majority of studies fail to account for the long-term effects of vitamin D.
3. Chronically ill people are not deficient in vitamin D.
4. Healthy people are not deficient in vitamin D and do not need to consume extra amounts of this steroid.
5. The public does not require extra sun exposure in order to prevent vitamin D “deficiency.”
6. Vitamin D does not reverse osteoporosis.
7. Extra vitamin D does not reduce the risk of cancer.
8. Vitamin D deficiency does not cause rickets.
9. Most researchers fail to consider the alternate hypothesis about vitamin D.
10. When it comes to vitamin D, the current medical climate of consensus is hostile to new ideas.
11. Research touting vitamin D’s benefits is often biased, methodologically weak, and ultimately misleading.
12. The dairy and supplement industries are intent on heavily promoting vitamin D.
13. The media is neither well-informed nor objective about vitamin D.
14. We must take immediate action to remedy the health crisis that has resulted from faulty conclusions about vitamin D in chronic disease.

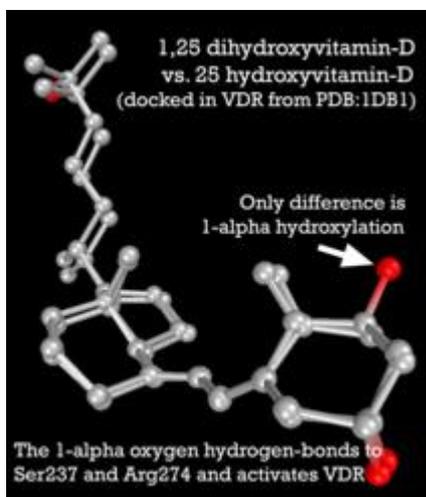
1. Vitamin D is not a vitamin; it is an immunosuppressive steroid.

Let's start with this fact: the vast majority of doctors touting the benefits of vitamin D are not aware of discoveries made by researchers in the field of molecular biology, which have clearly shown that the "vitamin" D derived from diet and supplements is not a vitamin, but a steroid with immunosuppressive properties when elevated.^[2]

There are several forms of vitamin D. The form of vitamin D we get from food, diet, supplements and sun exposure is called D3. D3 is converted by the liver into 25-D, which functions as a steroid. 1,25-D, the activated form of vitamin D, functions as both a steroid and a hormone. It is produced inside various types of cells, including those of the immune system and the kidneys, as well as in response to sunlight.^[3] In healthy individuals, the kidneys continually convert 25-D into its active form, 1,25-D.^{[4][3]}

According to [a paper](#) published by the Institute of Biomedical Research in Birmingham, England, "The active form of vitamin D, [1,25-D] is a potent immunomodulatory seco-steroid" meaning that it is a steroid-like molecule which is able to control the activity of the immune system."^[5]

Molecular modeling has shown that the hormonal 1,25-D form binds and activates the Vitamin D Receptor. The Vitamin D Receptor plays a fundamental role in the body. It transcribes 913 genes, and researchers at McGill University in Canada just released a [paper](#) saying it may actually transcribe 27,091.^[6] But, the Vitamin D Receptor also performs another critical function – it serves as a switch that regulates the activity of the innate immune system.^{[7][8][9]}



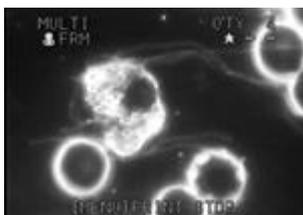
A molecular model comparing the structure of 25-D and of 1,25-D^[10] According to recent [10](#) According to recent [molecular models](#), the steroid 25-D binds the Vitamin D Receptor and affects the activity of the immune system as well, but in a manner opposite to 1,25-D. When the steroid 25-D binds the Vitamin D Receptor, it decreases the activity of the receptor, causing the innate immune system to slow down and shut off. This effect begins around 20 ng/ml and gradually increases with higher levels of 25-D, until the VDR becomes completely blocked.^[11]

At the moment, most researchers understand that 1,25-D activates the Vitamin D Receptor. However, they are unaware of the models which demonstrate that 25-D has the opposite effect. Consequently, they do not understand that when

people start to supplement with extra vitamin D (which is converted into 25-D) the Vitamin D Receptor begins to turn off, not on.

Most of these researchers are also unaware of a new understanding about the cause of many chronic diseases. As a person falls ill with a chronic disease, [L-form bacteria](#) begin to live inside the cells of the immune system and in various tissues.^{[12][13]} These bacteria create proteins that, just like elevated 25-D, are able to bind and block the Vitamin D Receptor.^[14] Together, elevated 25-D and bacterial proteins block the ability of the Vitamin D Receptor to turn on the immune system more than either substance alone.

[Molecular modeling](#) has also shown that the medication Olmesartan (called Benicar in the United States) is able to bind and activate the Vitamin D Receptor (VDR). Not only does Olmesartan activate the receptor, but, because its concentration can be controlled, it can reactivate the VDR even when it would normally be blocked by bacterial proteins or by excessive levels of 25-D. Olmesartan also binds a number of other receptors involved in the inflammatory response.^[15]



Long thin L-forms emerging from a cell as shown in a photo taken by Andy Wright Patients on a medical treatment known as the [Marshall Protocol](#) are able to use Olmesartan, along with pulsed, low-dose antibiotics to slowly eliminate L-form bacteria over the course of several years. These patients also eliminate vitamin D from their diets and block sunlight in an effort to lower the amount of 25-D blocking the Vitamin D Receptor. Hundreds of patients on the Marshall Protocol, who are sick with a wide array of previously incurable chronic diseases, are reporting symptomatic improvement or complete resolution of symptoms. Their case studies, many of which are documented on the Marshall Protocol study site, confirm in a clinical setting the molecular models which show that elevated 25-D is immunosuppressive.^{[16][10]}

The Vitamin D Receptor also directly controls the expression of many of the antimicrobial peptides (AMPs).^{[8][9][17][14]} The AMPs are proteins that kill bacteria, viruses, and fungus by a variety of mechanisms including disrupting membranes, interfering with metabolism, and targeting components of the machinery inside the cell. When 25-D reaches the level at which it inactivates the receptor, the AMPs are no longer produced, and bacteria can spread more easily throughout the body.

People infected with L-form bacteria are particularly prone to the effects of 25-D on the Vitamin D Receptor. That is because their L-form bacteria have created proteins which have already bound and deactivated the Vitamin D Receptor to varying degrees. Extra amounts of the steroid 25-D only bind and shut down the receptor even more, further inhibiting the innate immune system, the transcription of thousands of genes, and the production of the AMPs.

“It is when L-form bacteria die that they begin to cause a major increase in symptoms for the host, since as they die they release a large amount of toxins and cytokines, proteins that generate pain and fatigue.” The above scenario is all too familiar, since L-form bacteria are found everywhere in our environment, from soil, to water, to sperm, to inside the womb.^[18] Consequently, it seems that few people will remain free of them for long and most will acquire substantial levels of them as they age.^[19]

L-form bacteria have evolved mechanisms that allow them to live for long periods of time within the cells, and when alive, generally persist without generating too many symptoms.^[20]

It is when L-form bacteria die that they begin to cause a major increase in symptoms for the host, since as they die they release a large amount of toxins and cytokines, proteins that generate pain and fatigue. Furthermore, as L-form bacteria die, the cell that they have parasitized dies as well, and cellular debris is released into the bloodstream. These substances cause the tissues to become inflamed, resulting in what is known as “Th1 inflammation.”^[21]

As previously discussed, the innate immune system is responsible for killing L-form bacteria and is controlled by the Vitamin D Receptor (VDR). Elevated levels of the steroid 25-D and bacterial proteins bind and inactivate the VDR, causing the immune system to work less effectively.

As the immune system becomes increasingly inhibited, fewer L-form bacteria are killed. Furthermore, the Vitamin D Receptor is no longer able to transcribe the antimicrobial peptides, and fewer bacteria are killed by DNA fragmentation. As fewer bacteria die, fewer inflammatory cytokines are released, and fewer toxins and cellular debris enter the bloodstream. As the level of inflammation temporarily decreases, a patient will start to feel better.

This seeming wellness is illusory. Without the innate immune system and the antimicrobial peptides to keep L-form bacteria in check, the pathogens easily spread to new cells, new tissues, and new organs.

Many people who begin to supplement with vitamin D or spend extended periods of time in the sun only have a small or moderate amount of L-form bacteria in their bodies. Since these people’s immune systems are not yet severely compromised (their VDRs are not yet partially blocked by bacterial proteins), their bodies kill a fair share of the bacteria, resulting in minor aches and pains. But if 25-D rises to the level at which it inhibits their immune systems, less bacteria die, Th1 inflammation decreases, and their minor symptoms may be temporarily relieved.

Naturally, such patients feel that the extra vitamin D is helpful. It may take decades before their L-form bacterial load rises to the threshold at which they are diagnosed with an “autoimmune” illness, or have a stroke or heart attack. At this point later in life, they seldom make the connection between their current illness and the extra vitamin D they have been taking with no apparent ill effect for such a long period of time.

“If you think about it, it seems little wonder that vitamin D has become so popular. It’s basically an over-the-counter steroid.” It’s easy to see how people infected with even minor amounts of L-form bacteria tell their doctors that supplementation with vitamin D and increased exposure to the sun make them feel better. As Joyce Waterhouse, PhD, a researcher affiliated with Autoimmunity Research Foundation stated in [a discussion](#) of vitamin D in diseases caused by L-form bacteria, “If you think about it, it seems little wonder that vitamin D has become so popular. It’s basically an over-the-counter steroid.”^[22] Even the Vitamin D Council, a California non-profit agency who promote the use of vitamin D, say on their website that “vitamin” D is a steroid. Yet this group fails to question the full implications of their own statement.

Other steroids are commonly known to be immunosuppressive. Take the corticosteroid medication prednisone, a particularly effective immunosuppressant that affects virtually all of the immune system.^[23]

Prednisone is given to patients with diseases such as multiple sclerosis, rheumatoid arthritis, sarcoidosis, and lupus. Most doctors continue to think that these diseases are “autoimmune” in nature, and result when the body somehow mounts an immune response against its own cells and tissues. Hence, the desire to slow the immune system.

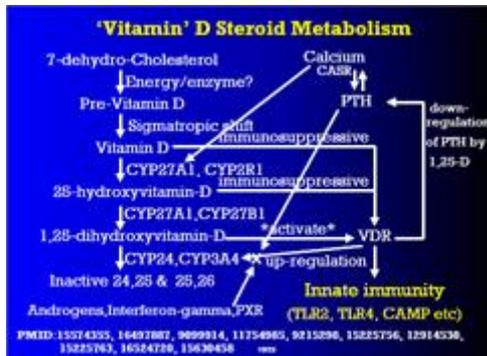
But when one understands that the diseases listed above are caused by L-form bacteria, the entire scenario becomes reversed. Prednisone, just like elevated 25-D, prevents the immune system from killing bacteria. Patients experience short-term relief and resolution of symptoms as the die-off slows down. But nobody would ever claim that prednisone actually cures “autoimmune” diseases. Instead, in the long run, patients taking prednisone generally become much more ill and require increasing amounts of palliative medication.

“At the moment there is a significant gap in communication between the molecular biologists who have realized that “vitamin D” is a steroid, and doctors who continue to think of it as a nutrient. ”At the moment there is a significant gap in communication between the molecular biologists who have realized that “vitamin D” is a steroid, and doctors who continue to think of it as a nutrient. But now that the actions of 25-D and 1,25-D have been confirmed by molecular modeling, it seems unlikely that doctors will be able to cling to the “vitamin” label for much longer.

As John Arbuthnot, author of *Of the Laws of Chance* states, “There are very few things we know which are not capable of being reduced to mathematical reasoning.....and where a mathematical reasoning can be had, it’s as great a folly to make use of any other, as to grope for a thing in the dark when you have a candle standing by you.”^[24]

Of course, it’s only been over the past five years that biomedical researcher Trevor Marshall has revealed how L-form bacteria affect the Vitamin D Receptor, and exactly how 25-D affects the immune system. So clearly, before these very recent discoveries, researchers were forced to study vitamin D while missing vital pieces of the puzzle. However, it seems that in their enthusiasm to

identify vitamin D's benefits, many experts have not sufficiently absorbed the medical literature, literature that well before Marshall's work revealed the complexities associated with the Vitamin D Receptor, the lynchpin of the innate immune system.



If researchers made themselves aware of work done by colleagues such as Dr. Tian Tian Wang at McGill University, they would know that when 25-D and 1,25-D bind the vitamin D receptor, they adjust the transcription of at least 913 genes. A search on the website Pubmed reveals that an average of [\[24\]](#) is much more complicated than some clinicians and researchers realize. Click to enlarge. If researchers made themselves aware of work done by colleagues such as Dr. Tian Tian Wang at McGill University, they would know that when 25-D and 1,25-D bind the vitamin D receptor, they adjust the transcription of at least 913 genes. A search on the website Pubmed reveals that an average of [one paper a day](#) is published on the actions of the Vitamin D Receptor. [\[25\]](#) Perhaps, armed with an understanding of this research, they would hesitate before recommending that the public take such high doses of the “vitamin.” It is only prudent that such a powerful seco-steroid and its transcriptional activity be understood to a far greater extent lest we all further subject ourselves to what is nothing short of an unofficial clinical trial of historic proportions.

Not surprisingly, the few researchers who understand the complexities of vitamin D seem concerned about supplementation. Recently, Professor Ronald M. Evans, a Fellow of the Salk Institute, delivered a seminar to the FDA about the public health policy on vitamin D. Given that vitamin D is seco-steroid rather than a vitamin, he indicated that he would advise his family against adding vitamin D to their diets. [\[26\]](#)

Based on the above, it's not surprising that researchers at Duke University found that elderly men and women who consumed higher levels of calcium and, in particular, vitamin D are significantly more likely to have greater volumes of brain lesions, indicating regions of damage that can increase risk of cognitive impairment, dementia, depression and death. The team found that vitamin D intake, (mean 341 IU and maximum intake 1014 IU), was the only variable that retained a significant correlation with the brain lesions when analyzed by a multivariate analysis. [\[27\]](#)

Unaware of the latest research on the immunosuppressive properties of high levels of vitamin D, the researchers hypothesized that the calcium rather than the vitamin D was the main culprit in causing the lesions. They speculated that

in patients given extra calcium, the calcium might be deposited inside the blood vessels of the brain rather than the bone. According to their theory, vitamin D would accelerate the process because it is involved in regulating calcium absorption and metabolism.

A much more likely explanation is that the lesions result when L-form bacteria in the brain cause the release of cytokines that damage the tissues. Sometimes the resulting inflammation damages blood vessels and promotes calcification, but it is the L-form bacteria, not the calcium that is the true culprit.

The connection between bacteria and calcification in heart disease has already been noted. Researchers at the Hospital Das Clinicas in Brazil found significantly higher concentrations of *Chlamydia pneumoniae* and *Mycoplasma pneumoniae* in calcified nodes of blood vessels throughout the body, including the heart and the aorta - causing them to suggest that “these bacteria may be associated with the development of calcification and inflammation.”^{[28][29]}

2. The vast majority of studies fail to account for the long-term effects of vitamin D.

The decrease in bacterial die-off among patients consuming a lot of vitamin D does mean that, at least in the short-term, less cytokines and toxins are released into the tissues and inflammation decreases. Since these substances damage the tissues, we frequently hear about studies stating that vitamin D can correct problems with the kidneys, parathyroid function, or resolve other maladies.

Although the decrease in toxins, cytokines, and overall inflammation may be helpful in the short-term, over longer periods of time, the negative consequences of L-form bacteria spreading throughout the body inevitably surpass any temporary beneficial effect created by a decreased level of toxins and cytokines, particularly since L-form bacteria have been implicated in such a vast array of diseases.^[18]

In April of 2000 [a study](#) published in the *Archives of Internal Medicine* by doctors at the State University of New York at Buffalo found that five patients confined to wheelchairs with severe weakness and fatigue were able to walk after supplementing with 300,000 IU's of vitamin D (a huge amount!) over a period of six weeks. Sadly, the patients were not “cured”, and almost certainly relapsed in the months following the study. They were simply feeling the effect of a temporary decrease in cytokine and toxin release that resulted after the high levels of vitamin D completely shut down their innate immune systems. In fact, one of the patients actually died in the weeks during which vitamin D was administered.^[30]

“Instead, as in the *Archives* study, they track subjects over the course of weeks, months, or one or two years, during the period of time when study participants are usually feeling the palliative effects of the steroid.” One of the abiding weaknesses of studies on vitamin D is that researchers do not follow subjects consuming the steroid for a sufficient period of time. Instead, as in the *Archives*

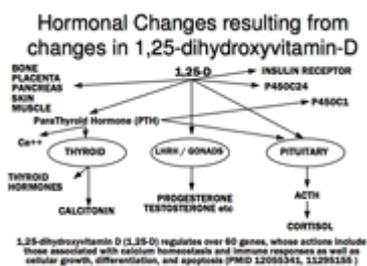
study, they track subjects over the course of weeks, months, or one or two years, during the period of time when study participants are usually feeling the palliative effects of the steroid.

Researchers will rarely, if ever, track subjects over the course of decades, the length of time needed to begin to note the negative changes that L-form bacteria cause later in life. In fact, L-form bacteria grow so slowly that researchers in the future will surely have to check back with their subjects at least 20-30 years after they begin supplementing with vitamin D in order to determine whether or not the steroid has contributed to the development of a chronic disease.

3. Chronically ill people are not deficient in vitamin D.

We are continually bombarded with studies claiming that patients with chronic disease are deficient in vitamin D. But this is not the case, and the misunderstanding comes from a misplaced focus on 25-D.

Numerous studies have demonstrated that the level of the hormone 1,25-D rises in patients with many chronic diseases.^[22] Chronically ill patients starting the Marshall Protocol sometimes have a level of 1,25-D exceeding five or six standard deviations above the “standard” value.^[31]



1,25-D affects a range of hormonal pathways. Click to enlarge. Sometimes called the body’s “master hormone”, 1,25-D directly controls the pathways that regulate the body’s other hormones including the thyroid, sex, and stress hormones. Consequently, elevated 1,25-D greatly contributes to the pathogenesis of chronic disease, since as a person’s level of 1,25-D rises to an unnaturally high level, the master hormone can no longer correctly regulate the above pathways.^[32]

A wide array of studies also point to the fact that 25-D is low in people with numerous chronic inflammatory diseases.

What explains these altered levels of 1,25-D and 25-D? (Note: If you find that the next few paragraphs seem complicated, hang in there! You will still be able to follow the rest of the article.)

As previously mentioned, in patients with chronic disease, L-form bacteria create proteins that affect the Vitamin D Receptor (VDR) in a manner similar to 25-D. They bind and inactivate the VDR, preventing it from transcribing a wide array of genes and enzymes.

In a paper recently published in *BioEssays*, “Vitamin D discovery outpaces FDA decision making,”^[33] Marshall describes how in healthy individuals, the VDR transcribes an enzyme called CYP24. CYP24 breaks down excess 1,25-D, ensuring that the level of 1,25-D in the body stays in the normal range. But in chronically ill individuals, the VDR (which is blocked by bacterial proteins) can no longer transcribe CYP24. The level of 1,25-D in the body becomes significantly elevated since there is no CYP24 to keep it in check.

1,25-D binds to the PXR receptor, a receptor that is involved in making another enzyme called CYP27A1. CYP27A1 is responsible for converting D3 into 25-D in the liver. Elevated 1,25-D affects the activity of the PXR receptor in a way that causes less D3 to be converted into 25-D, meaning that the level of 25-D in chronically ill individuals drops.

Yet another factor contributes to the low level of 25-D seen in patients with chronic disease. An enzyme called CYP27B1 normally regulates the amount of 25-D converted into 1,25-D. When more CYP27B1 is produced, conversion occurs at a greater rate.

L-form bacteria release cytokines, proteins that cause pain and fatigue. These cytokines activate a protein called Protein Kinase A (PKA). PKA in turn activates CYP27B1, causing more 25-D to be converted to 1,25-D. The level of 25-D in the body decreases, and the level of 1,25-D increases.^[34]

A [study](#) conducted by researchers at the University of South Carolina supports this scenario. The team gave healthy subjects high levels of 1,25-D and verified that it can indeed inhibit the conversion of vitamin D into 25-D. They found that this phenomenon also occurs in certain diseases in which patients naturally develop a high level of 1,25-D.^[35] Consequently, the low 25-D observed in patients with chronic disease is not a sign of vitamin D deficiency, but is an indicator of the disease process.

What happens when doctors and researchers take note of the low level of 25-D in patients with chronic disease? They all too often conclude that the low level of 25-D is *contributing* to or *causing* the disease. With such a mindset, doctors are all too eager to give patients oral supplements of 25-D in an effort to “remedy” the situation.

“Unfortunately the exact opposite is true. The level of 25-D in the body is not causing the illness, it is a *result* of the disease process.” Unfortunately the exact opposite is true. The level of 25-D in the body is not causing the illness, it is a *result* of the disease process, and as clear an indication as any that the patient is suffering from a significant degree of L-form bacterial infection. It’s similar to the connection between folic acid and heart disease - low levels of folic acid often lead to an increase in the amino acid homocysteine – a compound that at high levels has been linked to increased incidence of cardiovascular disease (CVD). Yet, extensive studies have revealed that giving patients with CVD folic acid supplements does not lower levels of homocysteine, and that high levels of the compound in patients with CVD is simply a result of the disease process.^[36]

Key to this misunderstanding are the doctors and researchers who fail to test the level of 1,25-D in patients with chronic disease. If they did, they might pick up on the fact that 25-D is low precisely because 1,25-D is elevated.

This certainly explains why a research team at the University of Wisconsin Osteoporosis Clinic, who did not test subjects' levels of 1,25-D, seemed puzzled by the results of [a study](#) which revealed that some participants getting abundant sun exposure still displayed low levels of 25-D.^[37]

Or take for example, scientists at Musgrave Park Hospital in Belfast, Ireland, who published in 2006 the results of [a study](#) that tested the level of 25-D in 75 patients with fibromyalgia, but failed to test the subjects' levels of 1,25-D.^[38] Surely the research team must have been perplexed by the fact that, although all of the subjects seemed to be consuming perfectly adequate levels of vitamin D, 69.3% were suffering from vitamin D "deficiency" because their serum levels of 25-D were considered to be too low.

As with most chronic diseases, fibromyalgia is an illness in which L-form bacteria create proteins that prevent the VDR from transcribing the enzymes needed to keep 1,25-D in the correct range.^[39] But since the researchers who conducted the study failed to test the level of 1,25-D in their subjects, they focused solely on the diminished level of 25-D and were, it seems, completely oblivious to the actual disease process. This led them to incorrectly conclude "Vitamin D deficiency is common in fibromyalgia and occurs more frequently in patients with anxiety and depression."

In a similar [study](#), published in 2003 in the *Mayo Clinic Proceedings*, researchers in Minneapolis tested vitamin D levels in patients suffering from chronic, non-specific, musculoskeletal pain: 93% of them turned out to be vitamin D "deficient."^[40] If these studies' authors can't understand the process, it's easy to understand how other researchers who look over their results would interpret the data to mean that patients with fibromyalgia need to consume even more 25-D, in order to correct the so called "deficiency."

Incomplete Research: Eight Examples of Studies that Neglected to Test for the 1,25-D Metabolite

[Association of subclinical vitamin D deficiency with severe acute lower respiratory infection in Indian children under 5 y.](#)

[Vitamin D deficiency in systemic lupus erythematosus](#)

[Serum 25-hydroxyvitamin D and colon cancer: eight-year prospective study](#)

[Is vitamin D important for preserving cognition? A positive correlation of serum 25-hydroxyvitamin D concentration with cognitive function](#)

[Low vitamin D status: a contributing factor in the pathogenesis of congestive heart failure?](#)

[The vitamin D epidemic and its health consequences](#)

[Vitamin D status predicts physical performance and its decline in older persons](#)

[Bone mass and vitamin D deficiency in adults with advanced cystic fibrosis lung disease](#)

The failure to test for 1,25-D in subjects with chronic disease is as pervasive as it is troubling. The FDA continues to accept results from studies that do not bother to measure 1,25-D.

Naturally, when representatives of the FDA and other health agencies interpret the results of these studies they correlate chronic disease with vitamin D deficiency, and inevitably suggest that people should supplement with increased amounts of the “vitamin” in order to reverse or prevent chronic disease.

Of course, the media picks up on the conclusions of experts incorrectly attributing the low levels of 25-D in their patients to vitamin D deficiency. A [recent article](#) in *US News and World Report* states, “Research on vitamin D has flooded out over the past few months, linking a growing array of health ills to low levels of the nutrient.”^[41]

On the tanning website, tantoday.com, Jeffrey Dach, MD, laments in his article [“Vitamin D Deficiency, the Ignored Epidemic”](#) that the majority of people living in “sunny Florida” showed vitamin D deficiency (less than 20 ng/ml), or insufficiency (less than 40 ng/ml).^[42] This seems odd, considering the fact that if a person spends only 8-10 minutes in the sun they will obtain the entire RDA requirement for vitamin D even if they are not consuming foods with vitamin D or fortified products.

Citing published medical research, Dach goes on to report that vitamin D deficiency has been reported in 57% of 290 medical inpatients in Massachusetts, 93% of 150 patients with overt musculoskeletal pain in Minnesota, 48% of patients with multiple sclerosis, 50% of patients with lupus and fibromyalgia, 62% of the morbidly obese African American Women, 83% of 360 patients with low back pain in Saudi Arabia, 73% of Austrian patients with Ankylosing Spondylitis, 58% of Japanese girls with Graves’ Disease, and 40-70% of all Finnish medical patients.

What Dach doesn’t realize is that the opposite is true. The low 25-D measured in the above studies is a consequence, rather than a cause, of the disease process. In reality, the numbers cited are an indication that the diseases mentioned are caused by L-form bacteria.

An increasing body of research points to the fact that obesity is linked to certain species of bacteria in the gut. Sure enough, [a study](#) published in the *Journal of Clinical Endocrinology and Metabolism* by researchers at Tufts University found that subjects with the highest percentage of body fat had 20% lower blood levels of 25-D than those with the least body fat.^[43]

Michael F. Holick, one of the foremost vitamin D “experts” in the country, told the National Institutes of Health symposium “Vitamin D and Health in the 21st Century” that the nation faces “severe vitamin D deficiency” which, if not properly addressed, will have profound far-reaching health consequences. According to Holick and other “experts,” we are in the midst of a “silent epidemic of vitamin D deficiency.”^[44]

There's no disputing the extent to which broad segments of the population have "low" levels of 25-D or that there is an epidemic of chronic disease and obesity. According to the CDC, seven of every ten Americans who die each year, or more than 1.7 million people, die of a chronic disease,^[45] and according to researchers at John Hopkins University, 75% of U.S. adults and 24% of U.S. children will be overweight or obese by 2015.^[46]

"Or, is this epidemic actually due to excess vitamin D consumption, and the immunosuppressive effects of 25-D on people infected with L-form bacteria?" Could it really be true that Professor Holick's "silent epidemic" is one of vitamin D deficiency? Or is this epidemic actually due to excess vitamin D consumption and the immunosuppressive effects of 25-D on people infected with L-form bacteria?

To be fair, most researchers and "experts" who study vitamin D are well-intentioned. They are deeply concerned about the health of the public, and are trying their best to battle chronic disease.

But the failure of most doctors and experts to question the level of not just 25-D but 1,25-D in the subjects they examine shows a lack of understanding of the fundamental aspects of vitamin D metabolism. Given that this pair of metabolites is so closely tied to each other, wouldn't any kind of understanding of the true nature of the vitamins D warrant measuring both?

Some claim that 1,25-D levels are not really important because they sometimes appear to fluctuate, and so do not measure them. Or, when they do measure 1,25-D, they automatically attribute a high 1,25-D combined with a low 25D to secondary hyperparathyroidism, a condition in which the kidneys produce more 1,25-D to compensate for inadequate calcium intake. Rather than recommend more calcium, such clinicians mistakenly recommend more vitamin D.

But secondary hyperparathyroidism can be ruled out by measuring parathyroid hormones, which researchers usually fail to do.^{[47][32]} More thorough studies on several inflammatory diseases^{[48][49][50]} specifically ruled out secondary hyperparathyroidism as a cause for the high level of 1,25D relative to 25D.

Furthermore, some researchers and physicians who test 1,25-D do not realize that the sample must remain frozen before analysis in order for the resulting reading to be accurate. With the limited data most researchers are habitually collecting, it's easy to understand how they have made the mistake of interpreting the low levels of 25-D in their subjects as an indicator of "deficiency."

4. Healthy people are not deficient in vitamin D and do not need to consume extra amounts of this steroid.

Based on the misunderstanding discussed above, researchers working with vitamin D and doctors administering the steroid seem fixated on the idea that more is always better. When a study about vitamin D presents inconclusive data, researchers inevitably suggest that the reason they didn't generate a

significant result was that their subjects should have taken more of the substance, which the public invariably understands as an endorsement of the idea that there is no limit to the amount of the vitamin D one should consume.

The prospect of identifying and distributing a substance of near universal benefit to the public's health has always had an undeniable appeal to researchers, a fact which may have made them less careful about vitamin D. Janet Foutin, a board member of Autoimmunity Research Foundation writes, "There was a flurry of activity to discover vital substances early on, and in that rush, the various forms of vitamin D were confused with one another and have been since— causing regulatory agencies to base their recommendations on faulty assumptions."

Researchers picked up on the feel-good, seemingly salutary, effects of vitamin D decades ago. It was on this basis that the FDA now encourages food producers to fortify their products with vitamin D, to the point where it is extremely difficult to find non-fortified milk in the United States.

In the United States, the FDA has determined that vitamin D can be added to breakfast cereals, grain and pasta products, milk, milk products such as cheese and butters, and soy milk.^[51] Canada goes so far as to require milk, evaporated milk, powdered milk, goat milk, and margarine to be fortified with vitamin D.^[52] The drive to supplement dairy products is as aggressive as ever. Even many developing countries currently fortify their milk with vitamin D.

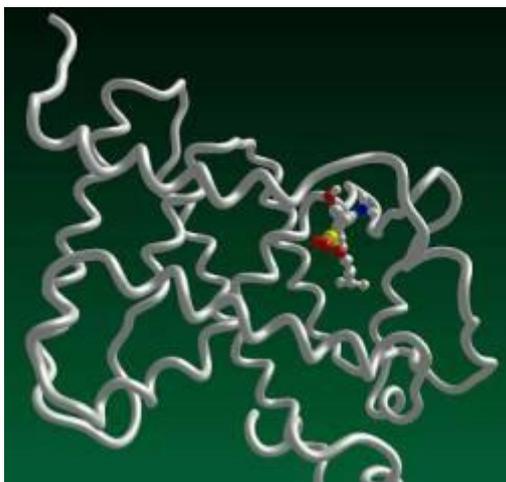
Furthermore, vitamin D is a fat-soluble substance that is stored for weeks or even months inside cells of fatty tissues and the liver.^[53] Unlike water-soluble vitamins that need regular replacement in the body, fat-soluble vitamins are eliminated much more slowly than water-soluble vitamins, meaning that it's relatively easy to maintain an adequate level of vitamin D in the body.

"Consequently, over the past few years, the "healthy" range for 25-D obtained from bloodwork has been adjusted upward" Countries in Europe do not require that products be fortified with vitamin D, and although many food producers fortify their products anyway, a fair amount of people obtain fresh food from local markets and supermarkets. Many people are puzzled by "the French Paradox" or the fact that some Europeans are able to eat a diet high in fat and still maintain a normal weight, whereas Americans eating a diet high in fat tend to become obese. A recent study by Thorpe et al found that nearly twice as many Americans are obese compared to their European counterparts. Perhaps this difference can be attributed to the amount of vitamin D in the food supply.

But since the vast majority of the public still consume large amounts of fortified products, it is difficult to find people who have a truly natural level of vitamin D in their bodies. Consequently, over the past few years, the "healthy" range for 25-D obtained from bloodwork has been adjusted upward to reflect the fact that people consume fortified dairy products. The FDA now suggests that people maintain a level of 25-D between 30-32 ng/ml, which is in the range at which it becomes immunosuppressive.^[54] This means that the levels of 25-D in people

eating a diet without fortified foods is inevitably considered to be too low, out of range, and ultimately a menace to their health.

With all the extra vitamin D we have added to the food chain, we no longer know what amount of 25-D the body would maintain under natural circumstances. Could it be that the people we call “Vitamin D deficient” actually have a normal level of 25-D? Studies which have tested the level of 25-D in people who live in countries where vitamin D is not added to the food chain prove this scenario to be true. A study which tested the level of 25-D in 90 “healthy, ambulatory Chilean women” showed that 27% of the premenopausal and 60% of the postmenopausal women had 25-D levels under 20 ng/ml.^[55] A study on healthy Bangladeshi women found that approximately 80% of the women had a level of 25-D under 16 ng/ml.^[56]



A molecular model showing caprine, a type of protein created by L-form bacteria bound into the Vitamin D Receptor. Add to this mess the fact that the vast majority of people considered to be “healthy” already harbor L-form bacteria. These people can tolerate even less 25-D because their Vitamin D Receptors have already been deactivated by bacterial proteins.

Nevertheless, Dr. Holick has [advised the FDA](#): “The 1997 daily recommended allowances for Vitamin D are totally inadequate to protect public health. New science supports a significant revision of the recommendation. Adults should be getting 1000 International Units (IU) of vitamin D a day, not the 200-600 (IU) that was recommended in 1997. Rewriting the recommended daily requirements as soon as possible should be a top priority.”^[44]

Similarly, Dr. Rainhold Vieth, another outspoken advocate for extra vitamin D, is adamant that the daily requirement of vitamin D should be in the range of 4,000 IU, or ten times the Recommended Daily Allowance.^[57] The FDA seem to be listening, considering that they are close to accepting a rule change that will amend one of the first health claims authorized in 1993 through the Nutrition Labeling and Education Act on the relationship between calcium and osteoporosis.

“The addition of even more vitamin D to the food supply will, without a doubt, continue to raise the average person’s level of 25-D well past the point at which

it becomes immunosuppressive. ” They have proposed to change the claim by adding high levels of vitamin D into the equation, with calcium, for a reduced risk of osteoporosis. The addition of even more vitamin D to the food supply will, without a doubt, continue to raise the average person’s level of 25-D well past the point at which it becomes immunosuppressive.^[58]

According to Clarisse Douaud at NutraIngredients-USA.com, a news source for the food and supplement industry, “The proposal is likely to be welcomed by the vitamin industry - at both supply and finished product levels - since it communicates the importance of vitamin D. Moreover, it does not restrict the advice to just some demographics, which could help marketers target new sectors of the market more effectively.”^[59]

Should the FDA really get into the business of systematic immunosuppression any more than it already has?

5. The public does not require extra sun exposure in order to prevent vitamin D “deficiency.”

One of the complaints of vitamin D promoters is that people have been trained to cover up with sunscreen and heavy clothing due to concerns that they will get wrinkles or skin cancer.^[60] Many such promoters of vitamin D including Holick, who is the author of the book *The UV Advantage*, advise people not to wear sunscreen despite the elevated risk of skin cancer that might result. This is a major problem, considering the fact that exposure to sunlight greatly elevates the level of vitamin D in the body, which directly fuels the ability of L-form bacteria to dysregulate the immune system.

[Holick told](#) the New York Times, “I recommend that whatever your ethnicity or skin tone, you get outdoors without a sunscreen somewhere around 20 percent of the amount of time it would take to cause a sunburn, however long that might be.”^[61]

Holick stands by this advice despite the fact that in February he was [rebuked and forced to resign](#) from the dermatology department at Boston University’s medical school.^[62] Part of the reason given was that his work is partly funded, and actively promoted, by the Indoor Tanning Association, an industry group with obvious financial interests.

On the official website of the Vitamin D Council, a group that heavily promotes consumption of vitamin D, executive director John Jacob Cannell [states](#), “We are saying that brief full body sun exposure may slightly increase your risk of skin cancer but it is a much smarter thing to do than dying of vitamin D deficiency. The only way to be sure you have adequate levels of vitamin D in your blood is to regularly go into the sun, or use a sun bed (avoiding sunburn).”^[63]

According to [the Council](#), if you totally avoid the sun, “You need about 4,000 units of vitamin D a day, which means you can’t get enough vitamin D from milk

(unless you drink 40 glasses a day) or from a multivitamin (unless you take about 10 tablets a day), neither of which is recommended.”^[64]

However, it’s been questioned whether sunscreen even blocks the majority of vitamin D production in the body. Scientists at the University of Melbourne took note of the level of 25-D and 1,25-D generated in two separate groups of study participants. One group wore SPF 17 sunscreen during the study period while the other group used a placebo.

[They concluded](#) that, “No person, including those aged 70 years and over, developed any vitamin D levels outside the normal reference range during the period of the study. The data suggest that over an Australian summer sufficient sunlight is received, probably through both the sunscreen itself and the lack of total skin cover at all times, to allow adequate vitamin D production in people who are recommended to use sunscreens regularly.”^[65]

“In my two decades of practice, I’ve never seen vitamin D deficiency caused by lack of sun exposure due to sunscreen use, yet the evidence that UV rays from the sun cause skin cancer is overwhelming.” The fact that people wearing sunscreen can still produce vitamin D has been confirmed by data collected from patients on the Marshall Protocol study site. Many patients on the Marshall Protocol are forced to avoid the sun in order to keep 1,25-D in the correct range, and experience an increase in symptoms when the metabolite is increased. Even when wearing heavy loads of sunscreen, patients still report symptom increase in response to light, suggesting that vitamin D can be created from the UVA rays which most standard sunscreens do not block adequately^[66].

The [American Academy of Dermatology says](#) it is “deeply concerned” about the current claims about vitamin D and sun exposure put forth by Holick. “I am not aware of any scientific studies that support this claim,” said Dr. David J. Leffell of the Yale School of Medicine Department of Dermatology. “In my two decades of practice, I’ve never seen vitamin D deficiency caused by lack of sun exposure due to sunscreen use, yet the evidence that UV rays from the sun cause skin cancer is overwhelming.”^[67]

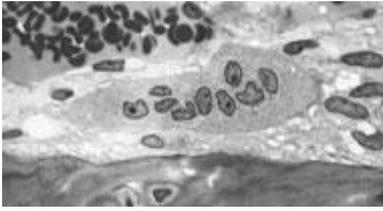
“I read better things in ladies’ magazines,” said Dr. Barbara Gilchrest, chair of the dermatology department at Boston University, and an authority on melanoma, the deadliest form of skin cancer. Holick’s book “is an embarrassment for this institution and an embarrassment for him.”^[62]

6. Vitamin D does not reverse osteoporosis.

Doesn’t vitamin D help reverse bone loss? No. An increasing number of large, recent studies are demonstrating that this is not the case.

Instead, current research has demonstrated that osteoporosis and osteopenia are often the direct result of infection with L-form bacteria which produce inflammatory cytokines and inactivate the Vitamin D Receptor. The only way to

achieve long-term reversal of bone loss is to kill the L-form bacteria driving the disease process.

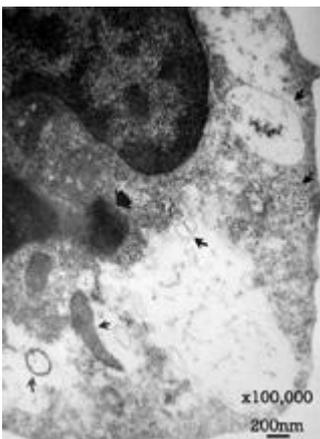


An osteoclast Osteoporosis and osteopenia result when the level of the hormone 1,25-D in the body rises above a certain range (above 43 pg/ml). Elevated levels of 1,25-D actually stimulate bone osteoclasts, cells that remove minerals from the bone. ^[68]

Stimulated osteoclasts dissolve bone material, causing it to be reabsorbed into the bloodstream. Not only does this lead to osteoporosis, but it can also lead to calcium being deposited in the soft tissues of the body, including those in the lungs, breasts and the kidneys (where it forms kidney stones). ^[69]

The elevated 1,25-D seen in people with osteoporosis is generally the result of L-form bacterial infection. ^[69] As previously discussed, L-form bacteria create proteins that bind and block the Vitamin D Receptor, preventing it from transcribing the enzyme CYP24. Since CYP24 is needed to keep levels of 1,25-D in check, the level of 1,25-D becomes greatly elevated in individuals without the active enzyme.

Furthermore, in chronically ill individuals, the cytokine release stimulated by L-form bacteria activates the pathway which causes increased production of CYP27B1, the enzyme that converts 25-D into 1,25-D. As more conversion occurs, the level of 1,25-D in the body rises.



L-form bacteria inside a white blood cell, picture by Emil Wirotkolf osteoporosis results in part from an increase in cytokines generated by L-form bacteria, then it would make sense that treatment to decrease cytokine release would, in the short term, reverse bone loss. Several studies have shown this to be true.

One of the inflammatory cytokines released as a result of infection by L-form bacteria is called TNF-alpha. A research team at the Rheumatoid Arthritis Center in Lyon, France found that a drug which blocks the production of TNF-

Alpha led to an increase in the subjects' spine and femoral bone mineral density (3.9% and 2.5% respectively). Another [study](#), this one by researchers at the Kerckhoff Clinic and Foundation in Germany, on a different group of subjects, confirmed the results, this time finding a 2.7% and 13% increase in bone density of the spine and femur.^[70]

It should be noted that these TNF-alpha blocking drugs do not provide a permanent solution to osteoporosis, since L-form bacteria will continue to spread as the drug is administered. Also, TNF-alpha blocking medications are known to have serious side effects. However, the research is of interest since it confirms the importance of Th1 inflammation in osteoporosis.

So how can osteoporosis and osteopenia be reversed? Some clinicians have patients supplement with vitamin D and calcium in an attempt to reverse bone loss. To begin with, patients with chronic disease may obtain less of a benefit from calcium supplements since the calcium metabolism of patients suffering from chronic disease is different from that of healthy individuals.^{[71][51]}

Supplementation with vitamin D only exacerbates the disease process. Supplements are taken orally in the form of vitamin D which is converted to 25-D in the liver. 25-D further blocks the ability of the VDR to transcribe the enzymes which keep 1,25-D in the correct range. This results in greater bone loss as even more 1,25D is produced.

A problem with many studies on bone mass is that participants are given both calcium and vitamin D supplements at the same time. If participants demonstrate a small increase in bone density, which of the two supplements should be given credit for their improvement? Based on what we know about the actions of elevated 1,25-D, certainly the calcium, not the vitamin D, accounts for any positive changes in bone mass noted among study participants.

The [largest meta-analysis](#) of calcium and vitamin D trials in people over 50 was recently published in the *Lancet*. It combined the results of 29 randomized trials in which researchers had given participants supplements of calcium and vitamin D. The researchers state on page 663 of their paper that the "addition of vitamin D supplementation was not shown to offer additional risk reduction over and above the use of calcium alone." They did find a small reduction in fracture risk (12%) correlated with calcium supplementation.^[72]

Similarly, a study by researchers at the Indiana University School of Medicine found that calcium supplementation (750 mg) improved bone density over a four-year period, whereas vitamin D supplementation (600 IU) had no effect. In fact, the effect of calcium on bone loss was blunted in subjects with the highest levels of vitamin D, causing the team to point out the danger of over-supplementation of the elderly with vitamin D if they are on an adequate calcium intake.^[73]

Another study, published in the *Archives of Internal Medicine*, also found that simply taking vitamin D as a supplement did nothing to improve bone health in

black women. In [the study](#), researchers randomly assigned 208 healthy black women, aged 50 to 75 years, to receive either 20 micrograms a day of vitamin D3 or a placebo. In addition, all the women received calcium supplements. After two years, the researchers increased the dose of vitamin D3 to 50 micrograms per day. All of the women underwent bone mineral density scans every six months during the three years of the study, to check for changes in bone health.

“There was really no difference in bone loss with vitamin D supplementation: our conclusion is that it does not need to be increased.” According to the study’s lead author, “There was really no difference in bone loss with vitamin D supplementation: our conclusion is that it does not need to be increased. Raising vitamin D levels did not show an advantage in terms of bone health.” However, calcium supplementation did cause an increase in bone mineral density in both groups.^[74]

On the other hand, some large studies have demonstrated that both calcium and vitamin D supplements do nothing to help strengthen the bones. In 2005, researchers at the University of York in the UK published in the *British Medical Journal* [a study](#) on 3314 people aged 70 years and older who were at risk for hip fractures because of decreased bone mass. The women supplemented with 1000 mg of calcium and 800 IU of vitamin D over a period of 24- 62 months.

By the study’s end, there was no measurable change in the bone quality of any of the women. The researchers found “no evidence that calcium and vitamin D supplementation reduce the risk of clinical fractures in women with one or more risk factors for hip fracture.”^[75]

[Another study](#) published in 2005 in the *The Lancet* by researchers at the University of Aberdeen in the UK generated the same results.^[76] Yet a [76\]](#) Yet a [third study](#), this one published in 2006, conducted by the Women’s Health Initiative, came to the identical conclusion.^[77]

And then there are studies showing that vitamin D actually *decreases* bone mineral density. In 1999, researchers at Cedars-Sinai Medical Center in Los Angeles conducted a [small study](#) on patients with osteoporosis and hypercalciuria, a disease in which excessive calcium is excreted in the urine. The participants were taking supplements containing high levels of vitamin D. They were asked to stop taking the supplements for three years, and their bone mass was monitored during that period of time. After stopping the supplements, the level of 25-D in their blood returned to the normal range, the hypercalciuria resolved, and there were annual increases in bone density of all subjects involved.

The study’s authors concluded: “Occult vitamin D intoxication was detected in patients who were using dietary supplements that contained an unadvertised high level of vitamin D. Resolution of vitamin D intoxication was associated with a rebound in bone mineral density.” Their study is particularly valuable because their 3-year follow-up phase showed that the increase in bone mineral density persisted after initial recovery.^[78]

Similarly, researchers at the University of Science and Technology in Norway just released the results of a study that measured the forearm bone mineral density of 3,042 Norwegian women, aged 50 - 70 years old. They found that those women who had not taken cod liver oil (a substance that contains high levels of vitamin D) during childhood had higher bone mineral density compared to those who had ingested cod liver oil.^[79] Since the study compared childhood intake of vitamin D to bone density at least 4-5 decades after ingestion, it is a good example of how only those studies which track vitamin D intake over long periods of time are likely to pick up on the harm the secosteroid causes in the longterm.

“Resolution of vitamin D intoxication was associated with a rebound in bone mineral density.” In the long run, the best way to reverse the condition is to bring the level of 1,25D in the body back into a range where minerals will no longer be leached from the bones and the level of inflammatory cytokines can return to normal. In the meantime, getting the RDA of calcium from foods and supplements without vitamin D can be helpful.^[69]

Another misconception among some clinicians is the idea that vitamin D enhances the absorption of calcium. This is not the case. 25-D is a simple steroid which does not affect the genes responsible for calcium absorption. In contrast, the Vitamin D Receptor is a receptor that transcribes thousands of genes,^[6] some of which do affect the metabolism of calcium.

As biomedical researcher [Trevor Marshall says](#), “In chronic disease the two things (vitamin D itself and the VDR) are NOT synonymous.”^[80] In patients with chronic disease, the VDR is unable to function properly. As previously discussed, this is due in large part to L-form bacteria that create proteins which inactivate the VDR, to a point where it can no longer correctly transcribe a wide array of genes, including some involved in calcium metabolism.

Once again then, it is only by killing the bacteria responsible for causing the disease process in the first place that the VDR can function properly, allowing the genes that affect the absorption of calcium to be turned on in the correct fashion. The mistaken notion that more vitamin D automatically means more activation of the Vitamin D Receptor, and hence greater calcium absorption, is probably the single greatest reason why vitamin D has been incorrectly identified as the solution to bone loss in people with chronic inflammatory disease.

In the same vein, low calcium in the bloodstream can lead to a condition called secondary hyperparathyroidism. The condition alters the level of Parathyroid Hormone in the body, which can result in bone loss. In patients with the disease, the kidneys try to compensate for the low level of calcium by increasing the conversion of 25-D to 1,25-D. Because the illness involves the vitamins D, many doctors mistakenly think that supplementation with the steroid might help the problem. However, the truth is that this condition is best corrected by bringing the level of calcium intake back into range.

Joyce Waterhouse, Ph.D. has recently [described in detail](#) a number of flaws in studies that use the relationship between low 25D and secondary hyperparathyroidism to estimate the optimal level of 25D. One problem is that they usually fail to ensure that subjects are consuming adequate calcium before assessing the relationship between 25-D and PTH. Thus, when researchers at Winthrop University Hospital in New York made sure that subjects consumed adequate calcium, they found that only a small percentage of patients with low 25-D actually had elevated levels of PTH, and that just 16 ng/ml of 25-D is usually enough to keep PTH in the correct range.^[81] This was confirmed by a recent study which found that PTH levels frequently remain normal even in patients with very low 25-D. The bone density of the elderly subjects in the study also remained the same as subjects taking higher levels of 25-D as long as their PTH remained normal.^[82]

When it comes down to it, 25-D accounts for only a very small percentage of variation in PTH levels, especially when subjects are taking adequate calcium. Several studies have shown that low magnesium, increasing age, or elevated serum phosphate and creatinine due to kidney disease also greatly contribute to the level of PTH, causing researchers at the University Hospital of New Norway to conclude that elevated PTH “is therefore probably a result of a combination of factors.”^[83] It’s not surprising then, that several studies have noted that giving vitamin D to patients with low levels of 25-D often does nothing to bring PTH back to normal levels.^[84]

In the end, it is perfectly possible that when calcium intake is adequate, most of what remain of the association between low 25-D and elevated PTH is simply part of the pathogenesis of chronic disease and osteoporosis. Just as the low 25-D seen in patients with chronic disease is the RESULT rather than the CAUSE of the disease process, elevated PTH in patients with low 25-D may simply be an indicator of inflammation caused by L-form bacteria.

7. Extra vitamin D does not reduce the risk of cancer.

The language of some studies, especially in the sections where researchers are asked to interpret their results, has suggested that supplementing with vitamin D might help people ward off cancer. Other research provides evidence that this is untrue.

In fact, the latest study by the National Cancer Institute - the first study to actually look at the relationship between measured vitamin D in the blood and subsequent total cancer deaths - failed to show an association between baseline vitamin D status and overall cancer risk in men, women, non-Hispanic whites, non-Hispanic blacks, Mexican Americans, and in persons younger than 70 or 70 years or older.^[85]

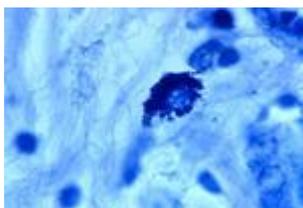
The findings, which appear in the Journal of the National Cancer Institute, are based on an analysis of data for 16,818 subjects who participated in the Third National Health and Nutrition Examination Survey. The subjects were at least 17 years of age when the survey was undertaken between 1988 and 1994 and they were followed through 2000. The researchers did find an association

between vitamin D and colorectal cancer risk, most likely for reasons that will be addressed later in this section.

When asked by a correspondent from CBS News if vitamin D can reduce the risk of cancer, David Fishman, head of the National Ovarian Cancer Early Detection Program at New York University said, “I don’t believe vitamin D is the answer. I wish it was as simple as saying ‘If you take vitamin D, cancer will be cured. I don’t think it’s that simple.”^[86]

The Mayo Clinic’s website [states](#), “It remains unclear if vitamin D deficiency raises cancer risk, or if an increased intake of vitamin D is protective against some cancers. Until additional trials are conducted, it is premature to advise the use of regular vitamin D supplementation to prevent cancer.”^[87]

L-form bacteria may be responsible for at least part of the pathogenesis of cancer. For one thing, L-form bacteria have been found in the tissues of patients with cancer. Some studies have found that people with certain types of cancer, such as prostate cancer, display the same dysregulated vitamin D metabolism observed in people with other chronic diseases now known to be bacterial in origin.^[22]



L-forms of various shapes and sizes inside the cells of a patient with breast cancer, photo taken by Alan Cantwell Several forms of bacteria have already been linked to cancer. Researcher [Alan Cantwell](#) used acid-fast staining to identify L-form bacteria in patients with Hodgkin’s Disease, lymphoma, prostate cancer and other immunological diseases.^[88] Both gastric cancer and gastric MALT lymphoma (lymphoma of the mucosa-associated lymphoid tissue) have been associated with *H. pylori* bacteria, and the bacterium has been categorized as a group I carcinogen by the International Agency for Research on Cancer (IARC).^[89]

Other research has shown a link between a cancer of the eye, ocular adnexal lymphoma (OAL) and *Chlamydia* bacteria. In October, researchers at the San Raffaele H. Scientific Institute in Milan published, in *Journal of the National Cancer Institute*, the results of [a study](#) which demonstrated that the antibiotic doxycycline is proving to be an effective treatment for this form of cancer. “Our prospective trial revealed that doxycycline is a fast, safe, and active treatment for OAL, both at initial diagnosis and at relapse,” the study’s authors wrote.^[90]

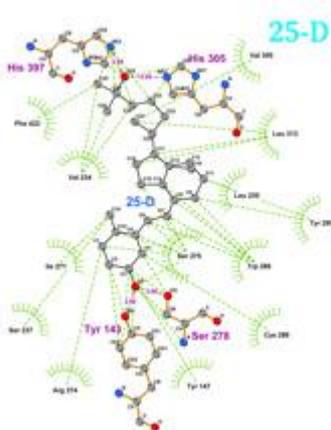
In 2006, D.L. Mager and team published a [review article](#) in the *Journal of Translational Medicine* called, “Bacteria and Cancer: Cause, or Cure?” According to Mager, “An overwhelming body of evidence has determined that relationships among certain bacteria and cancers exist.” In the paper, Mager details how research teams around the world have implicated *Salmonella typhi* in gallbladder cancer, *Streptococcus bovis* and *E.coli* in colon cancer, and

Chlamydia pneumoniae in lung cancer. According to Mager, the mechanisms by which bacterial agents may induce carcinogenesis include “chronic infection, immune evasion, and immune suppression.”^[91]

“Everybody knows inflammation induces cancer.” This suggests that, just as in other chronic diseases, long-term supplementation with vitamin D slows the immune system and facilitates the proliferation of L-form bacteria, ultimately driving the progression of cancer. Over time, L-form bacteria release more cytokines into the tissues, resulting in elevated levels of inflammation.

“Everybody knows inflammation induces cancer”, stated Francesco Marincola, MD, Senior NIH Investigator, at a recent conference. But how? [According to](#) biomedical researcher Trevor Marshall, “Th1 inflammation feeds the initial proliferative stage of cancer. Without Th1 inflammation the cancer cells can’t get adhesion to the ‘healthy’ cells and tissues, and can’t become proliferative. Then, as the cancer starts to metastasize, the inflamed stem cells are critical in enabling the spread of the inflammation, and the metastasis of the cancer.”^[92]

Furthermore, the Vitamin D Receptor is known to transcribe genes that work to prevent the spread of cancer. These include Metastasis Suppressor Protein, a protein that slows the creation of cancer cells, and Mitochondrial Tumor Suppressor 1 gene.



A molecule of 25-D Because inflammation induces cancer, it’s no surprise that research teams who follow their subjects for only a few years find that vitamin D seems to be “preventing” cancer. What they actually pick up on is the temporary decrease in cytokine production that results when 25-D slows the immune system and less L-form bacteria are killed. In the short term, as less bacteria die, less cytokines are released into the tissues, resulting in a temporary decrease in inflammation.

But in the long run, L-form bacteria will take full advantage of the subjects’ weakened immune systems. The bacteria will increase in number and spread to new tissues and organs. Decades later, the subjects will display higher levels of inflammation and higher rates of cancer and/or other chronic diseases, because even consistent immunosuppression with vitamin D will no longer sufficiently prevent so many L-form bacteria, both alive and dead, from releasing cytokines into the tissues. Consequently, researchers who follow their study participants

for the longest periods of time are often the ones to claim that supplementation with vitamin D offers no benefit when it comes to fighting the cancer.

Several months ago, researchers at Creighton University published the results of a study which found that vitamin D might lower the incidence of colorectal cancer.^[93] But Jacques Rossouw at the National Institutes of Health criticized the study. His group conducted a [93](#)] But Jacques Rossouw at the National Institutes of Health criticized the study. His group conducted a [similar study](#) that tracked the effects of vitamin D on 46,282 postmenopausal women with colorectal cancer and monitored the women over a longer period of time. “In our study we found absolutely no indication of an effect of calcium or vitamin D [on cancer] — zero,” he said. “And that’s over a seven-year period. It was a much larger study and a much longer study,” Rossouw told the press.^[94]

Dr. John Milner, chief of the Nutrition Science Research Group at the National Cancer Institute, agrees that skepticism is necessary. “We need to put this in the context of the entire diet and lifestyle and understand why we’re getting some effect,” Milner said. “I don’t want to minimize it, but let’s see a little bit more before we start jumping into public health policies.”^[95]

“In our study we found absolutely no indication of an effect of calcium or vitamin D [on cancer] — zero. And that’s over a seven-year period. It was a much larger study and a much longer study” Researchers at the Moores Cancer Center in California have published several disastrously misleading studies in which they incorrectly interpret the role of vitamin D in the pathogenesis of cancer. [One study](#), published recently in *Nutrition Reviews*, combines data from researchers who tested the level of 25-D in subjects around the globe during the winter months. Not surprisingly, the researchers, who failed to question the subjects’ levels of 1,25-D, picked up on the fact that patients at a higher risk for colorectal and breast cancer had lower levels of 25-D. In reality, the low 25-D observed in the subjects resulted from the downregulation of 25-D under the influence of elevated levels of 1,25-D

The researchers incorrectly state that higher levels of vitamin D offer a “protective effect” against cancer. Their conclusion: supplementing with up to 2,000 IU’s of vitamin D daily could prevent an estimated 600,000 cases of cancer. Unfortunately, virtually the opposite is true.^[96] In reality, the “protective effect” they are picking up on is simply the point at which 25-D becomes immunosuppressive and a temporary decrease in cytokine release begins.

These studies are the equivalent of giving subjects prednisone and concluding that prednisone offers a “protective effect” against cancer because it slows the immune system, leading to a temporary decrease in bacterial die-off. In addition, no study to date has tested whether study participants given high doses of vitamin D later develop a wide array of other chronic illnesses such as diabetes, arthritis, and heart disease. Surely if they looked, they would pick up on a higher incidence of inflammatory disease in the groups of subjects taking vitamin D.

A [particularly telling study](#) on vitamin D and prostate cancer by researchers at the University of Tampere in Finland revealed that the highest rate of prostate cancer occurred when subjects' levels of 25-D were either particularly low (under 8 ng/ml) or particularly high (over 33 ng/ml), giving a U-shaped curve.^[97]

It is very likely that the subjects with low 25-D were displaying the dysregulated vitamin D ratio (low 25-D, high 1,25-D) seen in patients with chronic disease, and that the patients with high 25-D were consuming very large amounts of vitamin D, amounts so large that the liver had no choice but to convert much of it into 25-D. These patients were sick indeed, since the high levels of 25-D suppressed their immune systems, disabling their ability to fight the progression of the cancer. Consequently, the researchers concluded that high levels of vitamin D might be associated with a higher risk of prostate cancer.

Similarly, a team of researchers at the National Cancer Institute in Rockville, Maryland conducted [a study on men](#) to determine the relationship between their levels of 25-D and pancreatic cancer risk. The researchers tracked the men for over 16 years. They found that in the long term, high 25-D levels greater than 26 ng/ml were associated with a three-fold increased risk for pancreatic cancer, suggesting that individuals consuming high levels of vitamin D were more likely to fall ill with the disease. Again, according to molecular modeling research, 26 ng/ml is near the range when 25-D significantly shuts off the Vitamin D Receptor, particularly when it is already partially blocked by bacterial proteins.

“Contrary to expectations, subjects with higher prediagnostic vitamin D status had an increased pancreatic cancer risk compared with those with lower status” They stated, “Contrary to expectations, subjects with higher prediagnostic vitamin D status had an increased pancreatic cancer risk compared with those with lower status.... Our results are intriguing and may provide clues that further the understanding of the etiology of this highly fatal cancer.”^[98]

Researchers at the Chinese Academy of Medical Sciences in China [found a similar association](#) between excessive vitamin D intake and esophageal and gastric cancers in men. Male subjects with levels of 25-D in the range of 48.7 ng/ml were much more likely to develop one of the two forms of cancer.^[99]

This same association between very high levels of 25-D (suggesting subjects are consuming large amounts of vitamin D) and higher rates of illness has also been observed in heart disease. A group of researchers at the Sree Chitra Tirunal Institute for Medical Sciences and Technology in India [explored the relationship](#) between elevated vitamin D (due to excessive sun exposure) and heart disease. The researchers tested the level of 25-D in 143 men with heart disease and 70 healthy control subjects. They found that the subjects with heart disease had much higher levels of 25-D in their blood, levels over 89 ng/ml, which is well beyond the level that causes the VDR to completely shut down.^[100]

Many people who hear about studies on cancer and vitamin D also don't realize how easy it is for researchers to manipulate statistics in order to demonstrate positive associations.

Take, for example, a [recent study](#) published in the *American Journal of Clinical Nutrition* by Lappe et al, who gave study participants 1,100 IU's of vitamin D over the course of four years (during the time when the short-term immunosuppressive effects of the steroid would be at its strongest). The researchers divided the participants into three groups. One group took no vitamin D, a second took calcium, and a third group took calcium and vitamin D. The team concluded that vitamin D significantly reduces the risk of cancer.^[93]

The study's biggest flaw is that the researchers discarded the data of subjects who developed cancer during the first year of the study. Their excuse: cancers during the first year would have been present but undiagnosed at entry. Of the 50 people who developed cancer during the four-year study, 13 were removed based on this premise, and only 37 cases of cancer were actually analyzed. But the 13 people who developed cancer during the first year were likely to be the study participants with the highest loads of L-form bacteria. They would have been the people to suffer the most from the negative impact of elevated 25-D on the immune system. If data from the 13 participants would have been included in the study, the results would have reflected much less of a "benefit" from vitamin D. Even the researchers admit that "their conclusion was strengthened by both the observational, substantial improvement in risk reduction when cancers occurring early in the trial were excluded."

Recall for a moment the study on breast cancer by the Women's Health Initiative discussed earlier in this paper. The researchers concluded that vitamin D might offer a small benefit in preventing breast cancer. The story was picked up by the media and advertised *ad nauseam*. The authors' inboxes were no doubt brimming with media requests.

"Neither use of supplemental calcium nor vitamin D intake was associated with [breast cancer] risk." However, the media does not seem interested in broadcasting the results of other studies which have determined that vitamin D offers no benefit whatsoever in preventing breast cancer. A [second study](#) by the Women's Health Initiative found no reduction in risk of breast cancer among postmenopausal women supplementing with 1000 mg calcium and 440 IUs of vitamin D.^[101] Researchers at the American Cancer Society conducted [101](#) Researchers at the American Cancer Society conducted [a study](#) on 68,567 postmenopausal women and found that "neither use of supplemental calcium nor vitamin D intake was associated with [breast cancer] risk."^[102] And researchers at the Northern California Cancer Center [102](#) And researchers at the Northern California Cancer Center [found no association](#) between dietary vitamin D intake during adolescence and subsequent breast cancer risk.^[103]

Of course, since we know our picture of cancer remains relatively incomplete, we shouldn't reject the possibility that vitamin D might offer some small benefit in preventing the disease. If L-form bacteria didn't exist, then it might make sense to supplement with vitamin D in the hope that this might be true. But is it worth the risk when 25-D also slows the immune system, facilitating the spread of L-form bacteria into new tissues and organs, and also prevents the Vitamin D Receptor from transcribing the antimicrobial peptides and several anti-cancer genes? To say nothing of evidence which points to the probability that L-form

bacteria and their subsequent dysregulation of the immune system are the ultimate cause of cancer in the first place.

Take, for example, 1,25-D. Several laboratory studies have shown that elevated 1,25-D may have a small anti-tumor effect.^[104] But elevated 1,25-D is also key in allowing L-form bacteria to spread unchecked from cell to cell. Elevated levels of 1,25-D leach calcium from the bones. High levels of 1,25-D affect muscle function, particularly the cardiac muscle.^{[105][106]} Studies which have detailed the intricate feedback pathways between the two forms of vitamin D have shown that elevated levels of 1,25-D are also immunosuppressive.

Consequently, any effect that 1,25-D might have in reducing tumors comes with a price - a price so large that it puts patients at much greater risk for Alzheimers, arthritis, diabetes, heart disease, strokes, and a vast array of other chronic diseases. And it may even have long term cancer promotion effects that cancel out any purported short-term benefit.

So, although elevated 1,25-D might possibly turn out to have a short-term beneficial effect on one small part of the complex disease process that is cancer, the effects on the L-form bacteria that also contribute to the illness mean that, in the end, it generates a plethora of negative consequences, consequences which contribute to more insidious, far-reaching and long-lasting aspects of the disease.

8. Vitamin D deficiency does not cause rickets.

Rickets is a softening of the bones that leads to fractures and deformity. The majority of cases occur among children in developing countries who suffer from severe malnutrition.



This child's bowed legs are a symptom of rickets. This past March a team of biologists at Harvard Medical School published the results of [a study](#) on rickets. The researchers engineered mice without vitamin D receptors (VDRs). Since vitamin D can have no effect on the body unless it can bind to the VDR, the mice could use no vitamin D whatsoever in their bodies. The researchers found that if the mice were given a diet high in calcium and phosphorous they did not develop rickets and their bones were just as strong as normal mice with active Vitamin D Receptors.^[107]

[A second study](#), by the same research team, corrected rickets by replacing calcium and phosphate ions in the bloodstream of mice without Vitamin D Receptors, thereby confirming the results. The team concluded that rickets is not caused by a deficiency of vitamin D but instead results from [hypophosphatemia](#), a condition where the level of phosphorous in the blood is too low.^[108]

Clearly, low calcium was part of the problem as well. Diminished levels of calcium cause an increase in Parathyroid Hormone, which subsequently causes the body to excrete too much phosphorous. This causes the level of phosphate in the body to drop, leading to the altered bone formation seen in rickets. Joyce Waterhouse, PhD, a researcher associated with Autoimmunity Research Foundation writes, “Low phosphorus is the proximate cause — but low calcium intake is generally the ultimate cause.”

In 2004, [a study](#) published in the *American Journal of Clinical Nutrition* by researchers from the Mayo Clinic, Oregon University School of Medicine, and other institutions confirmed that a low level of calcium can lead to rickets. The team assessed the absorption of calcium in 15 Nigerian children with active rickets. They found that all 15 children had resolution or improvement of rickets after six months of treatment with calcium supplements.^[109]

“Rickets in toddlers is a large problem in parts of Africa, especially Nigeria. It is not due to vitamin D deficiency but is caused by not having enough calcium in the diet.” Consequently, the US Department of Agriculture website [clearly states](#) “Rickets in toddlers is a large problem in parts of Africa, especially Nigeria. It is not due to vitamin D deficiency but is caused by not having enough calcium in the diet.”^[110] It’s certainly no surprise that children in Africa, who get copious amounts of sunlight, are not suffering from a disease caused by vitamin D deficiency.

In North America, too, the role of calcium is being reexamined. [DeLucia et al emphasize](#) that “Nutritional calcium deficiency may occur in North American infants and is not limited to the setting of developing countries.”^[111] Some attribute a recent small increase in rickets in North America to an increase in breast feeding, claiming that this is due to breast milk being low in vitamin D. However, breast feeding is also often accompanied by a diet low in calcium, particularly after weaning (e.g., juices rather than milk). Also, in recent years, more people have begun to avoid milk due to a greater awareness of lactose intolerance.

[Marshall suggests](#) that rickets may be related to Th1 inflammation, as a number of patients on the Marshall Protocol with Th1 illnesses and their close relatives report having had rickets as children.^[112] Vitamin D proponents claim that vitamin D added to the food supply was responsible for a historical decrease in rickets. But the history of rickets shows that a typical rickets case often had a history of smallpox, measles, or whooping cough.^[113] Plus, a [112](#) Vitamin D proponents claim that vitamin D added to the food supply was responsible for a historical decrease in rickets. But the history of rickets shows that a typical rickets case often had a history of smallpox, measles, or whooping cough.^[113]

Plus, a [1997 study](#) in Ethiopia found a high association between pneumonia and rickets.^[114] This provides more suggestive evidence that infection, either obvious and acute, or subtle and chronic, may play a role in the development of rickets and may exacerbate the effects of a low-calcium diet.

Does vitamin D come into the picture at all?

If a child with rickets is severely deficient in vitamin D, as well as in calcium and phosphorous, administering a small amount of vitamin D (which will be immediately converted into 1,25-D) can help by allowing the Vitamin D Receptor to turn on genes that affect the absorption of calcium. This probably explains why in the early 19th century, some children given high doses of vitamin D were said to be cured from rickets.

However, if supplementation is continued, the level of the precursor form of vitamin D (25-D) in the body will soon reach the point at which it becomes immunosuppressive. With the negative effects of this situation in mind, it makes much more sense that patients low in calcium should simply be given extra calcium, which can remedy the situation without the need for vitamin D.

Thus, it goes without saying that the involvement of vitamin D in the above process does not justify the high levels of vitamin D currently added to the food chain in the name of “preventing rickets”, and that the health of the public would be much better served by regulations ensuring that they obtain adequate calcium and phosphorous rather than vitamin D.

9. Most researchers fail to consider the alternate hypothesis about vitamin D.

When it comes to studies about vitamin D, researchers simply do not consider the alternate hypothesis, the idea that additional supplemental vitamin D might be harmful or unnecessary, and that that low 25-D in many chronic diseases is a consequence of the disease process rather than a cause. At a recent conference on vitamin D organized by the American Cancer Society, Dr. Len Lichtenfeld, Deputy Chief Medical Officer of the organization, [stated unequivocally](#), “There is no dispute among medical professionals that vitamin D is beneficial for our health.”^[115]

“This study is “not as ringing an endorsement of calcium and vitamin D as one might like.” If the results of a study show that vitamin D didn’t help the subjects involved, the study’s authors seem all too eager to explain away the results or discard the findings. Take the [recent \\$18 million dollar study](#) on vitamin D and calcium conducted by the Women’s Health Initiative. The study of more than 36,000 middle-aged and older women – the largest ever to test the health benefits of vitamin D – found that calcium and vitamin D had essentially no benefit on the bone density of the women involved.

After seven years of taking the supplements the group given supplements showed a 1% increase in hip-bone density but ranked no better statistically in avoiding fractures of all kinds.^[77] This study is “not as ringing an endorsement of

calcium and vitamin D as one might like,” said one of the study’s authors, Dr. Norman Lasser at New Jersey Medical School. For one, the researchers might have considered whether the 1% increase in hip-bone density was due to the calcium, and not the vitamin D.

Nevertheless, the researchers who conducted the Women’s Health Initiative study tried to explain away the findings. [An article about the study](#) from the Associated Press stated, “Many experts downplayed the meaning of the negative finding. Dr. Bess Dawson-Hughes, a Tufts University vitamin expert who helped shape the dietary guidelines, said they should remain unchanged for now.” The article went on to state that, “Some researchers said the effect would have been clearer with higher doses of vitamin D, perhaps up to 1,000 units daily.” Furthermore, nearly every researcher, including the team who conducted the study, denied the implications of the findings, urging people to continue taking vitamin D despite the fact that the study showed it had no beneficial effect. About this, the article wrote, “Even so, experts are urging women to stick with government advice to keep taking the supplements anyway.”^[116]

In fact, the leader of the study, Rebecca Jackson at Ohio State University stated, “Based on our findings, women, particularly those over 60, should consider taking calcium with vitamin D for bone health and to guard against fracture.”^[117] Jackson’s advice is at odds with her own findings. It’s as if she is referring to data that doesn’t exist. It seems that researchers like Jackson are so prepared to say that vitamin D is beneficial that even when studies prove it isn’t, they say it’s helpful anyway.

Meanwhile, a second analysis of the same study group found that the supplements did not lower the women’s risk of colorectal cancer. Naturally, the researchers once again questioned the data. [The San Diego Union Tribune wrote](#), “While the results were also disappointing, researchers speculated that a benefit might show up with more time.”^[118] No one bothered to comment on the finding that the women taking vitamin D had a 17% increased risk of developing kidney stones.

Perhaps the most recent example of researchers’ blindness to the implications of even their own results is on display in the August 25, 2007 issue of the *Lancet*. The [largest such meta-analysis](#) to date, Dr. Tang and team statistically analyzed a total of 29 studies involving 63,897 study participants with an eye towards definitively measuring the connection between fractures and bone loss, and vitamin D and calcium supplementation. Their abstract states clearly enough in the interpretation section, “We recommend minimum doses of 1200 mg of calcium, and 800 IU of vitamin D.”

“The addition of vitamin D to calcium did not change treatment effect significantly.... It was not significant.” This language is interesting in that it is just not supported by the substance of the findings. On page 661: “The addition of vitamin D to calcium did not change treatment effect significantly.... It was not significant.” On page 663: “Although addition of vitamin D supplementation was not shown to offer additional risk reduction over and above the use of calcium

alone, a significant difference was observed between the effects of different vitamin D doses. This discrepancy could be due to statistical artifact.”^[76]

What is this “artifact”? “Our analysis was limited by the scarcity of vitamin D doses higher than 800 IU,” the researchers claim. “It is possible that vitamin D does have a beneficial effect when the dose is large enough (>800 IU).”

It’s also possible that these researchers are having it both ways, using ambiguous results to support two nearly opposite conclusions. In one breath (above), they blame the “scarcity” of vitamin D data at higher levels (>800 IU) for not being able to measure a significant effect. And then, in the abstract no less, they conclude that “treatment effect,” meaning fracture risk reduction, was better with “vitamin D doses of 800 IU or more than with doses less than 800 IU of vitamin D.”

A similar bias can be seen in a recent [meta-analysis](#) by researchers in Lyon, France, who concluded that subjects who began taking vitamin D were 7% less likely to die in the next few years than those who did not. In the paper, the team fails to mention that the benefit of vitamin D given alone, without calcium, was not statistically significant. Furthermore, four of the studies analyzed actually showed a greater rate of death among subjects taking vitamin D (though the death rate was only statistically significant in one of the studies). Two of these four studies were using a single injection with a very large amount (300,000 IU) of the steroid.^[119]

How can these contradictions be allowed to persist?

It seems that many researchers are under pressure to demonstrate that their data is statistically significant. Researchers obtain the money to conduct a study by applying for a grant, and inconclusive or insignificant results are not likely to impress the institutions in charge of distributing resources. It may be that many of the scientists who bolster claims of statistical significance have already received large grants and are eager to show that the money was spent on an analysis worthy of note.

The lockstep perception of the healthiness of ingesting vitamin D has even led some experts to downplay the effects of its toxicity. According to the school of nutrition at Colorado State University, “Because fat-soluble vitamins are stored for long periods, they generally pose a greater risk for toxicity than water-soluble vitamins when consumed in excess.”^[120] In excess, vitamin D is highly toxic. It causes calcification of soft tissues, and may cause calcified kidneys and kidney failure. Too much vitamin D may disrupt the level of calcium in the blood and produce fatigue and mental confusion.

In 1997, researcher Bernadette Marriott [wrote an editorial](#) in *Annals of Internal Medicine* entitled “Vitamin D Supplementation: A Word of Caution” in which she argues that vitamin D supplements be recommended with caution and care.^[121]

In a section on their website called “The Truth About Vitamin D Toxicity” John Jacob Cannell, the Vitamin D Council’s executive director tries to invalidate Marriott’s concerns by repeating the advice of vitamin D “expert” Reinhold Vieth,

who feels that fear of vitamin D toxicity is unwarranted, and such unwarranted fear, bordering on hysteria, is rampant in the medical profession.^[63] Cannell himself states that “In fact, living in America today while worrying about vitamin D toxicity is like dying of thirst in the desert while worrying about drowning.”

And to whom is the NIH listening?

The National Institutes of Health held a recent conference that examined a range of scientific perspectives related to vitamin D and bone health across the life cycle. They invited Professor Reinhold Vieth, one of the most vocal advocates for very high vitamin D supplementation, to advise them on the issue of vitamin D toxicity. He was scheduled to give a speech about “Potential Adverse Outcomes of Vitamin D.”^[122] Although the transcripts of [122](#) Although the transcripts of [the conference](#) are not yet publicly available, it would be reasonable to think he mentioned few, if any, causes for real concern.

10. When it comes to vitamin D, the current medical climate of consensus is hostile to new ideas.

Sir Isaac Newton once wrote, “If I have seen farther than others it is because I have stood on the shoulders of giants.” But even intellectual giants get it wrong from time to time. Many researchers today seem to regard the work of the most prestigious among them— at least in the field of vitamin D— as giants, and there is little room for questioning of basic assumptions.

“For many current scientific fields, claimed research findings may often be simply accurate measures of the prevailing bias.” The fact that researchers seem so hesitant to say anything negative about vitamin D reflects the blanket assumption that vitamin D simply cannot be harmful. Arguments to the contrary are assumed to be untenable and entirely without merit. This is due in no small part to the reality that researchers today are overly committed to the idea of replication, the requirement that new findings must be supported by and stem from earlier research. As John P. A. Ioannidis writes in the *Journal of PLOS Medicine*, “For many current scientific fields, claimed research findings may often be simply accurate measures of the prevailing bias.”^[123]

New research draws on the inferences of other studies conducted in the field, and it is required that conclusions be reviewed and accepted by peers who must share the same set of assumptions. If and when someone puts forth findings that turn an established area of medicine on its head, those findings are unceremoniously questioned and dismissed.

Janet Foutin, a staff member of Autoimmunity Research Foundation writes, “Even the most credible researchers must start upon a research foundation of previous empirical studies or it won’t be taken seriously in the community from which it springs. This consensus contamination is widespread and makes it very difficult to introduce process-or-content foundation-level changes.” In other words, when new medical research is always derived from previous work, fundamental changes to researchers’ assumptions are slow in coming.

The case of Barry Marshall and Robin Warren, the team of researchers who discovered that ulcers are caused by *H.pylori* bacteria rather than stress is particularly instructive. When that duo first put forth their findings, [published in 1982](#),^[124] other doctors and researchers rejected their data and walked out of their lectures. It's not as if published explanations of ulcers invoking stress and food intake weren't internally consistent or validated in other peer-reviewed papers. Those papers just had one little problem: they were wrong. The medical community's refusal to consider the alternative and, ultimately correct, explanation offered by Marshall and Warren lasted decades.

11. Research touting vitamin D's benefits is often biased, methodologically weak, and ultimately misleading.

Studies about vitamin D can often be biased, methodologically weak, or both. Publication bias is the tendency of scientists to report findings that report statistical significance, but to bury examples that are inconclusive. The tendency towards publication bias is well-suited to prolonging any number of false positive conclusions, including the proposition that consumption of vitamin D is healthy.

[An article](#) published last September in the *Wall Street Journal* discussed how scientific journals are much more likely to publish studies which reveal positive qualities of a medication or supplement rather than those which demonstrate a negative or null effect.^[125]

For example, throughout the 1990s, publication bias gave the impression of a link between oral contraceptives and cervical cancer. But in reality, a 2000 analysis concluded that the studies finding no link between the two factors had seldom been published. In the end, the analysis found there was only "a spurious statistical connection" between oral contraceptives and cervical cancer.

In fact, another analysis, conducted in 1999, found that the percentage of positive studies in some fields routinely tops 90%. According to Lee Sigelman of George Washington University, "That is statistically implausible, suggesting that negative results are being deep-sixed." As a result, "what we read in the journals may bear only the slightest resemblance" to reality.^[126] "You hear stories about negative studies getting stuck in a file drawer, but rigorous analyses also support the suspicion that journals are biased in favor of positive studies," says David Lehrer of the University of Helsinki.

Various forms of bias may also be perpetuated by drug and supplement companies looking to make a profit. Several reports have revealed that some companies will even pay their own staff to help researchers write up the results of studies. The final papers eventually end up in scientific journals.

For example, in 2001, the *American Journal of Kidney Diseases* published [an article](#) that touted the use of synthetic vitamin D. Its author was listed as Alex J. Brown, an associate professor at Washington University in St. Louis. But recently, that same article was featured as a work sample by a different person:

Michael Anello, a freelance medical writer, who posted a summary of it on his web site. Mr. Anello says he was hired to write the article by a communications firm working for Abbott Laboratories, which makes a version of the vitamin D product.

“Promotion has a different goal than publishing a legitimate research study” According to Anna Wilde Mathews, a staff reporter at *The Wall Street Journal*, “It’s an example of an open secret in medicine: many of the articles that appear in scientific journals under the bylines of prominent academics are actually written by ghostwriters in the pay of drug companies. These seemingly objective articles, which doctors around the world use to guide their care of patients, are often part of a marketing campaign by companies to promote a product or play up the condition it treats.”^[127]

Sabine Kleinert, an executive editor at *The Lancet*, says she makes a genuine effort to reject articles that have a marketing spin. “Promotion has a different goal than publishing a legitimate research study,” says Dr. Kleinert. She suspects companies sometimes influence medical writers “to write it up in a certain way to make a product sound more efficacious than it is.”^[128]

As opposed to research bias, methodological flaws are easier to identify— if one cares to look. So conditioned are we to hearing that vitamin D is helpful, when a study reveals the substance’s “benefits” few people take the time to analyze how the data was collected or ultimately interpreted. Because of this, the methods that researchers use to collect data on patients taking vitamin D rarely come under scrutiny.

Results from recent research attempting to demonstrate a protective effect of ingested vitamin D have been alternately inconclusive and lacking in sound methodology. Yet, all too often, researchers (and their colleagues and the media) conclude that such an effect exists. [One such study](#), published in *Annals of Internal Medicine* in May of this year, claims as a part of its conclusion: “Findings from this study suggest that higher intakes of calcium and vitamin D may be associated with a lower risk of developing premenopausal breast cancer.”^[129] Before you run out and begin gulping down vitamin D pills as the local news anchor might have you do, what is the basis for this claim? Let’s take a look.

The study was conducted on 10,579 premenopausal women and 20,909 postmenopausal women by the Women’s Health Study Group. The women were asked to fill out baseline questionnaires about lifestyle, medical history, and were required to specify how often they ate certain foods. Participants self-reported whether they were taking vitamin D supplements, calcium supplements, and multivitamins. Then, the researchers followed up with the women over a ten-year time period to determine if they developed breast cancer.

The media took the results of the study and generalized the conclusion to all women despite the fact that in the results section of their paper, the researchers had clearly stated that among the postmenopausal women subjects, vitamin D

intake was not inversely associated with breast cancer risk. There was also no association between calcium and vitamin D intakes and more aggressive breast cancer in postmenopausal women.

Furthermore, although the difference between those who ingested the highest and lowest levels of vitamin D did result in differing rates of breast cancer, those groups differed in other very substantial ways. Women in the group who consumed higher levels of vitamin D were 67% less likely to be a smoker, burned over 39% more calories doing physical activity, and drank about a fifth less alcohol. The researchers here assure us that they statistically controlled for these factors. But, if you have a purported effect of just a few percentage points, how strong can your conclusion be when you are studying two such substantially different groups?

This phenomenon is known as the “healthy-user bias.” Until the power of this effect is sufficiently and widely appreciated, researchers will continue to publish the results of studies that fail to account for a wide variety of lifestyle differences, many of which may be impossible to quantify.

Jerry Avron, a Harvard epidemiologist argues that when it comes to large epidemiological studies the healthy-user bias has the potential for “big mischief.” For example, in one large population studied by Elizabeth Barrett-Cinner, an epidemiologist at the University of California, San Diego, having gone to college was associated with a 50% lower risk of heart disease. Other studies have established a connection between a person’s income and a lower risk of heart disease. Considering all these factors, is it possible to isolate one single factor, such as vitamin D, as the reason for a small decrease in disease noted in a particular study?^[1]

“Women in the group who consumed higher levels of vitamin D were 67% less likely to be a smoker, burned over 39% more calories doing physical activity, and drank about a fifth less alcohol.” It wouldn’t so far-fetched to assume that women who take vitamin D are more likely to get better health care and be aware of prevailing health advice. After all, they’ve probably heard about studies like this one! Why else would they be consuming extra vitamin D? For a substance that ultimately is said to have a negative or absent net effect on the development of cancer, these circumstances represent an infinite loop of self-fulfilling prophecy.

In fact, even with a marginally significant correlation between vitamin D intake and a lesser risk of cancer in premenopausal women coupled with a study population, the enormity of which can only be described as a statistician’s dream, the 95% confidence interval was gaping: 0.42 - 1.00. You can think of confidence intervals as an indication of how reliable an estimate is. In this case, the answer would be not very reliable.

Compounding matters was the method used to gather food frequency data from study participants. No researcher’s first choice of gathering data is a survey. But, in this particular case, the data was gathered in an especially problematic

way. Study participants were asked to remember back to what they consumed over the previous year, a method at which people appear to be notoriously poor.

A different group of researchers performed two simultaneous tests on the same participants. For one test, subjects were asked to recall the amount of vitamin D they had consumed in the previous year and for the second they were to fill out four one-week dietary records at regular intervals over the course of a year. The researchers found only a 0.35 correlation between the amount of vitamin D the subjects reported taking with the first method as compared to the second, meaning that the subjects had a very low tendency to correctly recollect the amount of vitamin D their food diaries suggested they were taking.^[130]

This methodological flaw is a problem and shows up from time to time in studies done in this manner, precisely because it magnifies the potential for systematic error. Once more, the problem with this particular survey is that the study participants who are more likely to engage in a wide range of known and unknown cancer-protective behavior by limiting alcohol intake, being more physically active, etc. are also more likely to know that researchers think they should be having more vitamin D and remember it that way. Therefore, it is probable that they over-reported their vitamin D intake. With the results of the study mentioned above showing that there was a very weak correlation between the amount of vitamin D participants reported taking over the course of a year and the amounts of vitamin D they reported consuming when given a weekly survey, it would be hard to argue otherwise.

Why would researchers use this kind of methodologically weak survey? The short answer is that their in-house statistician told them they needed the numbers— tens of thousands of participants. The one-year recollection survey is a crude instrument, but it is relatively easy to administer to a study cohort the size of a small city, and that is a minimum number you'll need to demonstrate any statistical connection between vitamin D intake and cancer. Even with these numbers, this study, for the record, comes up just short on statistical significance in spite of its methodological flaws. Returning once more to the original concluding language of the *Annals* paper, it would seem like a stretch to conclude that if you're a premenopausal woman, taking vitamin D has any demonstrable effect on cancer.

Even when researchers give their subjects vitamin D, they may fail to account for differences in lifestyle between women who carefully follow instructions and take supplements as directed and those who do not correctly follow the guidelines. This phenomenon is known as compliance effect. Avorn argues, "Girl Scouts in the group, the compliant ongoing users, are probably doing a lot of other preventative things as well."^[1]

David Freedman, a statistician at the University of California, Berkeley, has written books on clinical trial design and analysis. Freedman says in *The New York Times*, "Women who take their pills as directed year in and year out are known to be different from ordinary women, so it is a mistake to generalize from them to the entire population."^[131]

“Women who take their pills as directed year in and year out are known to be different from ordinary women, so it is a mistake to generalize from them to the entire population.” In fact, in an article in *The New York Times Magazine*, Gary Taubes puts forth Freedman’s findings, explaining that “whenever epidemiological studies compare people who faithfully engage in some activity with those who don’t - whether taking prescription pills, or vitamins, or exercising regularly or eating what they consider a healthy diet - the researchers need to account for the compliance effect or they will most likely infer the wrong answer. They’ll conclude that this behavior, whatever it is, prevents disease and saves lives, when all they are really doing is comparing two different groups of people who are, in effect, incomparable.”

Taubes explains, “No matter how well designed and how many tens of thousands of subjects they might include, they [observational studies] have a fundamental limitation. They can distinguish associations between two events. But they cannot inherently determine causation - the conclusion that one event causes the other. As a result, observational studies provide what researchers call hypothesis generated evidence - what a defense attorney would call circumstantial evidence.”^[1]

While no research methodology can be completely free of bias or methodological weakness, one potential bright spot, in certain cases, is that of molecular modeling. Increasing numbers of researchers are using molecular modeling to accompany or replace experiments using human volunteers. Molecular models display exactly how molecules in the body fit together. For example, software can take a virtual molecule of 25-D or 1,25-D and demonstrate exactly how they fit into the Vitamin D Receptor.

It was molecular modeling software that allowed a research team at McGill University to identify over 900 different genes transcribed by the Vitamin D Receptor.^[6] And it was molecular modeling that allowed biochemical researcher Trevor Marshall to discover exactly how 25-D binds and inactivates the Vitamin D Receptor, proving with inarguable precision that the molecule is immunosuppressive as it reaches higher levels. As Marshall says, “The primary difference between mathematical science and evidence-based medicine is that one is definitive and one is interpretive. As we enter the 21st century, the tools to reduce some important medical dilemma to mathematical precision are now available in Molecular Genomics.”^[11] And when molecular modeling evidence is combined with clinical data showing the reversal of many chronic diseases, the evidence is even stronger.

12. The dairy and supplement industries are intent on heavily promoting vitamin D.

In order to understand why we hear such unreservedly positive things about vitamin D, one must appreciate the extent to which supplement and food interest groups such as the one representing the dairy industry promote its use. Thanks in no small part to media campaigns sponsored by the dairy and supplement industries, vitamin D has become widely known as the “sunshine

vitamin” and is touted to health-conscious individuals in an effort to make them feel that vitamin D is part of a responsible lifestyle.

Let’s start with the supplement industry, a syndicate which is certainly cashing in on vitamin D’s health “benefits.”

On the same online supplement retailer which [lists](#) how vitamin D can be used in connection with 21 different health conditions, we are offered oils and spreads, protein powders, breakfast bars, any number of other nutraceuticals, and some 40 different multi-vitamins, all of which contain the secosteroid.^[132] NEEDS, another online retailer, sells 37 different supplements with vitamin D and approximately 130 fish oils containing vitamin D, some priced over \$30.00 a bottle.^[133]

If you think that vitamin D isn’t heavily promoted by the food and dairy industries, think again. Consider the recent marketing battles that have emerged after the FDA proposed a rule change in its guidelines about vitamin D. Within the next few months, the FDA is expected to smooth the way for manufacturers intent on adding “high” levels of vitamin D to a variety of food products.

According to an article in *Packaging World Magazine* by Stephen Barlas, “It is predicted that this will set off a scramble by both milk, milk product, and juice marketers to redo their product labels and packaging in order to take advantage of the new guidelines.”^[134]

A petition from the Beverage Institute for Health and Wellness, which is funded by the Coca-Cola Co.— one brand of which is Minute Maid fruit juices and drinks— is petitioning the FDA to allow a broader claim about vitamin D to be made. At present, few product retailers choose to make the osteoporosis/vitamin D health claim because of the qualifications required to accompany it, such as noting that calcium only benefits “young adult white and Asian women who engage in regular physical activity.”



Minute Maid’s new line of Vitamin D-fortified

products Under the request advocated by the Beverage Institute, a food would have to be considered “high” in both calcium and Vitamin D before it could make an osteoporosis health claim. That would mean a product would have to contain at least 20% of the Daily Value (DV) of Vitamin D and/or calcium per reference amount customarily consumed. Minute Maid’s orange juice and some of its offshoots contain more calcium than the milk products now eligible to use the calcium/osteoporosis claim, and contain about the same level of Vitamin D.

On April 25 Minute Maid introduced its Enhanced Juice line. It includes new Minute Maid Multi-Vitamin and Minute Maid Active variety, and a calcium-fortified orange juice offering: Home Squeezed Style + Calcium + Vitamin D.

But according to Barlas, “producers of reduced-fat, low-fat and fat-free milk and yogurts will also benefit from a more streamlined and expanded claim. Once the FDA decision is finalized, milk and fruit juice marketers will begin battling one another for new market share.”

Cary Frye, vice president of regulatory affairs for the Intl. Dairy Foods Assn. (IDFA), says, “It is certainly clear that the intent of the petition is to be able to make stronger claims for fortified orange juice in preventing osteoporosis, to compete with milk in that regard. But the new simpler claim will be available to all foods, and it will be up to the dairy industry to leverage its nutrient-dense foods so as to market milk as the superior beverage choice.”

Of course, companies such as Colombo, Dannon and Yoplait, all of which sell vitamin D-fortified yogurts, are unlikely to take the challenge sitting down. Tom Nagle, senior vice president of marketing for IDFA, stated, “Fortified juices have made progress in consumer perception, but it is our intention to protect our number-one position with consumers.” In order to remain competitive, these companies are predicted to add the vitamin D/osteoporosis claims to their products as well.

But don't leave out the fishing industry. Vital Choices seafood company sends out an [“official newsletter”](#) with articles such as “Wild Salmon Affirmed as Top Vitamin D Source”^[135] and “Vitamin D May Lower Risk of Ovarian, Breast, Kidney, and Colon Cancers”, accompanied by a chart showing exactly which of their products are highest in vitamin D.^[136]

With vitamin D appearing in so many foods, and people eating them, often regardless of the total amount of the substance they are consuming, along with the additional amount produced by sun exposure, the danger of this trend becomes obvious.

The dairy industry generates millions off the claim that vitamin D increases the absorption of calcium, a claim to which more and more studies are taking exception. It's no small secret that industry marketing campaigns have nothing to do with science and are all about generating a profit.

Not long ago, the National Dairy Council partially funded a small study to measure the effects of consuming dairy on weight loss. That study consisted of about 30 participants, only 11 of which were in the high dairy group. The subjects were instructed to follow a reduced-calorie diet, which many have argued was actually the key to their weight loss success.



An advertisement by the dairy industry touting milk's supposed weight-loss benefits On the basis of that suspiciously humble study, the Council mounted the “3-A-Day” campaign, a multi-million dollar marketing offensive spent on advertisements connecting milk to weight loss. In 2006 the milk producers even enlisted high-profile celebrities for their “Great American Weight Loss Challenge” and “Body by Milk” promotions.^[137]

The Dairy Council has persisted with their campaigns despite a number of research studies showing a contrary association, linking milk and dairy consumption to weight gain, and regardless of the fact that the Physicians for Responsible Medicine have filed a petition stating that the weight loss claims are misleading.

Now that an increasing number of studies are showing no association between vitamin D and osteoporosis, will the dairy and supplement industries persist with their campaigns involving vitamin D? If left unchecked, the answer is probably yes. And the public will continue to get information about vitamin D off the side of a beverage carton rather than from the molecular biologists who truly understand the actions of the steroid.

13. The media is neither well-informed nor objective about vitamin D.

There is no doubt about it— the media love vitamin D. Here’s a sample of articles that turn up on the web.

- Sunshine Vitamin D - Sunshine Vitamin Brightens Smiles
- The Sunshine Vitamin, The Crucial Need For Vitamin D
- Critics Now Accept the Sun as Your Healthiest Source of Vitamin D
- Vitamin D Deficiency, the Ignored Epidemic of the Developed World
- How The Sunshine Vitamin Zaps Disease
- Let the Sunshine Vitamin Brighten Your Day
- The Wonderful Healing Properties Of The Sunshine Vitamin D
- Sunshine: Don't Leave Home Without It
- Vitamin D - The Sunshine Vitamin!
- Almost Everyone Needs More of the Sunshine Vitamin
- Shining a Light on the Health Benefits of Vitamin D
- Vitamin D Deficiency: the Silent Epidemic

- The Miracle of Vitamin D

It's not as if the media is purposefully intending to deceive. The supposedly and unequivocally positive benefits of vitamin D make for good copy. The vitamin D story is one of the media's rare opportunities to convey a positive message, and they seize it whenever possible. Consumers of media are weary of hearing about skyrocketing obesity, about the failure of once promising drugs like Vioxx. They are also scared of cancer. Nothing attracts viewer interest more than the promise that the information presented will offer some way to get an edge on chronic disease. Vitamin D as the freely available anti-cancer wonder drug fits the bill very nicely. What members of the media do not realize, just like researchers who don't dig deep enough to seek out the alternative hypothesis, is that high-level vitamin D's feel-good effects are due entirely to its immunosuppressive nature.

"The vitamin D story is one of the media's rare opportunities to convey a positive message to their viewers, and they seize it whenever possible." Just like the supplement industry, the media will often unknowingly take a small study about vitamin D and blow it completely out of proportion. Newspapers, magazines, TV shows, talk shows, radio programs - all pick up the same story and repeat it over and over again, until it seems as if twenty different studies have been published on the subject rather than one. And the language is often overly dramatic. In [an article](#) about vitamin D published in *US News and World Report* called "The ABCs of D, Almost Everyone Needs More of the Sunshine Vitamin", the author states, "A single nutrient keeps bones strong, wards off diabetes, and protects against tuberculosis, cancer, colds, and the flu. Sound too good to be true? There's more: It's free. But you're almost certainly not getting enough."^[41]

So many articles about vitamin D end with copious amounts of advice about how to get more of the "vitamin." We see tables, which are attempts to help the reader maximize D intake. The authors often chime in with advice, basing their statements on data that is already misleading and on biases already unchecked. The vitamin D publicity sequence: from data to interpretation to scientific publication to media generalization is not unlike the children's game telephone, in which each subsequent interpretation is less accurate than the one that preceded it.

Not surprisingly, the media invites vitamin D "experts" to weigh in, communicating their mistaken views on vitamin D to a vast number of viewers. The following was taken from [an interview](#) with vitamin D expert Michael Holick that was published in *The New York Times*, "A visit to the office of Dr. Michael F. Holick in the Boston University Medical Center quickly conveys his enthusiasm for his favorite hormone, vitamin D. On the office walls are letters from sixth graders responding to a talk he gave."^[61]

"The important fact I learned from you yesterday is that most living things or persons need vitamin D," one child wrote. Added another, "Even frogs need vitamin D." To advance his point he handed a reporter a copy of a paper he had

recently written, "Vitamin D: The Underappreciated D-lightful Hormone That Is Important for Skeletal and Cellular Health."

Oh dear.

14. We must take immediate action to remedy the health crisis that has resulted from faulty conclusions about vitamin D in chronic disease.

When it comes to public health, a few false assumptions can have disastrous and far-reaching consequences. The failure of researchers to understand that the low levels of 25-D observed in their subjects is not the cause, but the result of chronic disease, has put the public at greater risk for developing diseases that range from Alzheimers, to diabetes, to cancer, and is directly feeding an epidemic of chronic disease. The chronic diseases caused by L-form bacteria are far more common than currently realized, and are often only noticed as subtle signs of aging, such as osteoporosis, obesity, fatigue and arthritis.^[19]

As I write, well-intentioned researchers striving to improve public health, are unknowingly touting a steroid that allows L-form bacteria to proliferate and spread. People taking extra vitamin D are not getting better. Instead, they are feeling a short-term palliative effect from a steroid that slows the immune system, preventing L-form bacteria from releasing the cytokines that result in Th1 inflammation.

It is critical that everyone from clinicians to medical researchers to policymakers understand that recent research has shown the immunosuppressive effects of 25-D and the transcriptional effects of 1,25-D. New research that has also revealed how to correctly activate the Vitamin D Receptor once it has already been turned off by proteins created by L-form bacteria. Given this research, it's no less important that the various industries touting the benefits of vitamin D immediately stop those marketing campaigns.

"It is incumbent upon the FDA and every other food regulatory agency around the world to scale back and reverse plans to add even more vitamin D to the food supply." Most importantly though, it is incumbent upon the FDA and every other food regulatory agency around the world to scale back and reverse plans to add even more vitamin D to the food supply. Given the FDA's explicit mission "to protect the public health" and the balance of research which is rapidly accumulating against vitamin D, the agency will simply have no alternative.

Vitamin D is far more often a cause, and not a cure for disease. And that discrepancy makes a world of difference. It is the difference between advising the public to supplement with vitamin D and telling people to avoid supplementation at all costs. It is the difference between preventing a disease and speeding its progression, the difference between fighting an epidemic of chronic disease, and watching more and more people fall ill every day. And it's a change that needs to happen right now.

In *The Structure of Scientific Revolutions*, Kuhn argues that, at first, a scientific revolution takes place slowly, and that changes in mentality happen gradually as serious thinkers reconsider ideas. He states that “If the paradigm is one destined to win its fight, the number and strength of the persuasive arguments in its favor will increase. More scientists will then be converted, and the exploration of the new paradigm will go on. Gradually the number of experiments, instruments, articles, and books based upon the new paradigm will multiply.”

But when it comes to vitamin D, there is no time for Kuhn’s incremental revolution.

It’s no secret that the scientific community is particularly resistant to the long-term harmful effects of vitamin D. Understanding its role in chronic illness will require a vast number of people to admit they have been wrong. Uttering a collective “oops” will not come easily to the medical profession. One can hope that the urgency of this health crisis will inspire doctors, researchers, experts, and everyone with a stake in vitamin D to swallow their pride and strive to make things right for the common good.

Along with the revolution in attitude towards vitamin D is the equally important acceptance of the role of L-form bacteria that block the Vitamin D Receptor, and [the treatment](#) that can eliminate them. Instead of using short term palliatives, like vitamin D supplements, there is evidence that these bacteria can now be killed, meaning that many debilitating chronic diseases can now be cured.

It’s hard to say how this scientific revolution will pan out. One likely scenario is that those with the greatest stake in it, those most sickened by chronic disease, will simply demand that everyone - from the industry to the media to their doctors to medical researchers and regulatory agencies - take good note of the future of conventional wisdom about vitamin D.

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