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Vitamin D May Reduce COVID-19 Infection and Mortality

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STORY AT-A-GLANCE -

- A Spanish study found giving supplemental vitamin D3 (calcifediol) to hospitalized patients with PCR-confirmed COVID-19 — in addition to standard care — reduced ICU admissions by 82% and mortality by 64%
- People who already had higher vitamin D at baseline were 60% less likely to die
- Many are now calling for official vitamin D recommendations to be issued by their governments
- Other recent research found vitamin D is a contributing factor to COVID-19 outbreaks and infection severity. Surges in daily positive tests during the fall of 2020 in 18 European countries linearly correlate with latitude, and, hence, sun exposure and vitamin D levels
- One of the reasons vitamin D is so important against COVID-19 has to do with its influence on T cell responses. Vitamin D receptor signals regulate T cell responses and therefore play an important role in your body's defense against viral and bacterial infections

This article was previously published February 22, 2021, and has been updated with new information.

Vitamin D plays an important role in most diseases, including infectious disease, which is why from the very beginning of the COVID-19 pandemic, I suspected that optimizing vitamin D levels among the general population would significantly lower COVID-19 incidence and death.

Since then, mounting evidence reveals this is indeed the case, as researchers have repeatedly demonstrated that higher vitamin D levels reduce rates of positive tests, hospitalizations and mortality related to this infection.

Vitamin D3 Reduces COVID Infection and Mortality

A now-retracted, preprint Spanish study[1,2] found that giving supplemental vitamin D3 (calcifediol) to hospitalized patients with PCR-confirmed COVID-19 reduced ICU admissions by 82% and mortality by 64%.[3] People who already had higher vitamin D at baseline were 60% less likely to die.

Another study was published in June 2021.[4] In this study, researchers concluded that "vitamin D deficiency is associated with an increased risk of COVID-19 infection and mortality across a wide range of countries."

Other studies have shown that "vitamin D deficiency to address COVID-19 warrants aggressive pursuit and study."[5] Retrospective data demonstrated that a deficiency was also associated with an increased risk of COVID1-19 infection.[6]

Renewed Calls for Vitamin D Recommendations

In response to the now-retracted study, British MP David Davis tweeted that hospitals should consider giving vitamin D3 to every COVID patient in every hospital in the temperate latitudes. Since other, peer-reviewed, studies like the ones I mentioned above support higher vitamin D levels being connected to a better chance of survival from COVID, it seems reasonable that Davis' suggestion is not out of line, regardless of the one retracted article.

Many others are also calling for official vitamin D recommendations to be issued by their governments. Among them is Emer Higgins,[7] a member of the Irish political party Fine Gael, who called on the Irish health minister, Stephen Donnelly, to include vitamin D supplementation in its "Living with COVID-19" strategy, slated for launch at the end of February 2021.

Higgins leaned on evidence from the Irish Covit-D Consortium,[8] which shows vitamin D helps optimize your immune response. "There is negligible risk in this strategy and potentially a massive gain," she said. According to the Covit-D Consortium, the nutrient can lower the risk of death from COVID-19 in the elderly by as much as 700%.[9]

Low Vitamin D Linked to COVID-19 Outbreaks and Severity

Another study[10] published in the journal Scientific Reports confirmed vitamin D is a contributing factor to COVID-19 outbreaks and infection severity. According to the authors, the surges in daily positive test results during the fall of 2020 in 18 European countries linearly correlate with latitude, and hence sun exposure and vitamin D levels. They point out that:

> *"The country surge date corresponds to the time when its sun UV daily dose drops below ≈ 34% of that of 0° latitude. Introducing reported seasonal blood 25-hydroxyvitamin D (25(OH)D) concentration variation into the reported link between acute respiratory tract infection risk and 25(OH)D concentration quantitatively explains the surge dynamics ...*
>
> *The date of the surge is an intrapopulation observation and has the benefit of being triggered only by a parameter globally affecting the population, i.e. decreases in the sun UV daily dose.*
>
> *The results indicate that a low 25(OH)D concentration is a contributing factor to COVID-19 severity, which, combined with previous studies, provides a convincing set of evidence."*

While it's well-recognized that most elderly individuals are deficient in vitamin D, the problem is widespread in all age categories, including children.

As noted in a February 2021 study[11] comparing vitamin D levels in breast milk collected in 1989 and 2016/2017, vitamin D concentrations are consistently higher during the summer but, overall, vitamin D levels have declined since 1989. As a result, pregnant

and lactating mothers and their infants may require vitamin D supplementation for optimal health.

Vitamin D Is Crucial for Optimal T Cell Responses

One of the reasons why vitamin D is so important against COVID-19 has to do with its influence on T cell responses. Animal research[12] published in 2014 explained how vitamin D receptor signals regulate T cell responses and therefore play an important role in your body's defense against viral and bacterial infections.

As noted in that study, when vitamin D signaling is impaired, it significantly impacts the quantity, quality, breadth and location of CD8 T cell immunity, resulting in more severe viral and bacterial infections.

> **"Strong antibody response correlates with more severe clinical disease while T-cell response is correlated with less severe disease."**

What's more, according to a December 11, 2020, paper[13] in the journal Vaccine: X, high-quality T cell response actually appears to be far more important than antibodies when it comes to providing protective immunity against SARS-CoV-2 specifically:[14]

> *"The first SARS-CoV-2 vaccine(s) will likely be licensed based on neutralizing antibodies in Phase 2 trials, but there are significant concerns about using antibody response in coronavirus infections as a sole metric of protective immunity.*
>
> *Antibody response is often a poor marker of prior coronavirus infection, particularly in mild infections, and is shorter-lived than virus-reactive T-cells …*
>
> *Strong antibody response correlates with more severe clinical disease while T-cell response is correlated with less severe disease; and antibody-dependent enhancement of pathology and clinical severity has been described.*

> *Indeed, it is unclear whether antibody production is protective or pathogenic in coronavirus infections. Early data with SARS-CoV-2 support these findings. Data from coronavirus infections in animals and humans emphasize the generation of a high-quality T cell response in protective immunity."*

The authors go on to state that epitopes associated with SARS-CoV2 have been identified on CD4 and CD8 T-cells in the blood from patients who have successfully recovered from COVID-19, and that these epitopes "are much less dominated by spike protein than in previous coronavirus infections."[15]

As a refresher, aside from SARS-CoV-2, there are six other coronaviruses known to cause respiratory disease in humans:[16]

- **Types 229E, NL63, OC43 and KHU1** are quite common and cause mild to moderate respiratory infections such as the common cold.
- **SARS-CoV** (Severe Acute Respiratory Syndrome coronavirus), associated with severe respiratory illness.[17,18]
- **MERS-CoV** (Middle East Respiratory Syndrome coronavirus) which, like SARS, causes more severe respiratory infections than the four common coronaviruses.[19]

Understanding the Role of Epitopes

What do they mean by "epitopes associated with SARS-CoV2 have been identified on CD4 and CD8 T-cells"? Epitopes[20] are sites on the virus that allow antibodies or cell receptors in your immune system to recognize it. This is why epitopes are also referred to as "antigenic determinants," as they are the part that is recognized by an antibody, B-cell receptor or T-cell receptor.

Most antigens — substances that bind specifically to an antibody or a T-cell receptor — have several different epitopes, which allow it to be recognized by several different antibodies. Importantly, some epitopes can cause autoimmunological pathogenic priming if you've been previously infected with SARS-CoV-2 or exposed via a COVID-19 vaccine.[21]

In other words, if you've had the infection once, and get reinfected (either by SARS-CoV-2 or a sufficiently similar coronavirus), the second bout has the potential to be more severe than the first. Similarly, if you get vaccinated and are then infected with SARS-CoV-2, your infection may be more severe than had you not been vaccinated.

For this reason, "these epitopes should be excluded from vaccines under development to minimize autoimmunity due to risk of pathogenic priming," a recent paper[22] in the Journal of Translational Autoimmunity warns.

One of the reasons why mRNA gene therapy "vaccines" are causing so many problems may in fact be because they have failed to "screen out unsafe epitopes to reduce autoimmunity due to homology between parts of the viral protein and the human proteome," according to that Journal of Translational Autoimmunity paper.[23]

Natural SARS-CoV-2 Infection Induces Broad Epitope Coverage

The authors of the Vaccine: X paper point out that while most COVID-19 gene therapy "vaccines" focus on the SARS-CoV-2 spike protein as a natural antigen, "natural infection by SARS-CoV-2 induces broad epitope coverage, cross-reactive with other betacoronaviruses."

Indeed, this has been demonstrated in a number of studies, including a Singaporean study[24,25,26] that found common colds caused by the betacoronaviruses OC43 and HKU1 might make you more resistant to SARS-CoV-2 infection, and that the resulting immunity might last as long as 17 years.

In other words, if you've beat a common cold caused by a OC43 or HKU1 betacoronavirus in the past, you may have a 50/50 chance of having defensive T-cells that can recognize and help defend against SARS-CoV-2. What the Vaccine: X authors are basically warning about is that the so-called vaccines are unlikely to provide the same level of immunity as natural infection does, and may even cause pathogenic priming.

Vitamin D Speeds Viral Clearance

Other research,[27] published in November 2020 in the Postgraduate Medical Journal, shows oral vitamin D supplementation also helps speed up SARS-CoV-2 viral clearance. This study included only asymptomatic or mildly symptomatic SARS-CoV-2-positive individuals who also had vitamin D deficiency (a vitamin D blood level below 20 ng/mL).

Participants were randomly assigned to receive either 60,000 IUs of oral cholecalciferol (nano-liquid droplets) or a placebo for seven days. The target blood level was 50 ng/mL. Anyone who had not achieved a blood level of 50 ng/mL after the first seven days continued to receive the supplement until they reached the target level.

Periodically, all participants were tested for SARS-CoV-2 as well as fibrinogen, D-dimer, procalcitonin and CRP, all of which are inflammatory markers. The primary outcome measure of the study was the proportion of patients testing negative for COVID-19 before Day 21 of the study, as well as changes in inflammatory markers. As reported by the authors:[28]

> *"Forty SARS-CoV-2 RNA positive individuals were randomized to intervention (n=16) or control (n=24) group. Baseline serum 25(OH)D was 8.6 and 9.54 ng/mL, in the intervention and control group, respectively.*
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> *10 out of 16 patients could achieve 25(OH)D>50 ng/ml by day-7 and another two by day-14 … 10 (62.5%) participants in the intervention group and 5 (20.8%) participants in the control arm became SARS-CoV-2 RNA negative. Fibrinogen levels significantly decreased with cholecalciferol supplementation unlike other inflammatory biomarkers.*
>
> *[A] greater proportion of vitamin D-deficient individuals with SARS-CoV-2 infection turned SARS-CoV-2 RNA negative with a significant decrease in fibrinogen on high-dose cholecalciferol supplementation."*

More Evidence Vitamin D Impacts COVID-19

If you haven't already gone to the free website I created to educate the world about vitamin D, please do now. It's **www.stopcovidcold.com**. You can download the free condensed version of the paper I had published last year that is easier to read and full of graphics to illustrate the information.

October 31, 2020, my own vitamin D review,[29] co-written with William Grant, Ph.D., and Dr. Carol Wagner, both of whom are part of the GrassrootsHealth expert vitamin D panel, was published in the peer-reviewed journal Nutrients. You can **read the paper for free on the journal's website**.

As noted in that paper, dark skin color, increased age, preexisting chronic conditions and vitamin D deficiency are all features of severe COVID disease and, of these, vitamin D deficiency is the only factor that is readily and easily modifiable.

You may be able to reverse chronic disease, but that typically takes time. Optimizing your vitamin D, on the other hand, can be achieved in just a few weeks, thereby significantly lowering your risk of severe COVID-19.

In our paper, we review several of the mechanisms by which vitamin D can reduce your risk of COVID-19 and other respiratory infections, including but not limited to the following:[30]

- Reducing the survival and replication of viruses[31]
- Reducing inflammatory cytokine production
- Maintaining endothelial integrity — Endothelial dysfunction contributes to vascular inflammation and impaired blood clotting, two hallmarks of severe COVID-19
- Increasing angiotensin-converting enzyme 2 (ACE2) concentrations, which prevents the virus from entering cells via the ACE2 receptor — ACE2 is downregulated by SARS-CoV-2 infection, and by increasing ACE2, you also avoid excessive accumulation of angiotensin II, a peptide hormone known to increase the severity of COVID-19

Vitamin D is also an important component of COVID-19 prevention and treatment for the fact that it:

- Boosts your overall immune function by modulating your innate and adaptive immune responses
- Reduces respiratory distress[32]
- Improves overall lung function
- Helps produce surfactants in your lungs that aid in fluid clearance[33]
- Lowers your risk of comorbidities associated with poor COVID-19 prognosis, including obesity,[34] Type 2 diabetes,[35] high blood pressure[36] and heart disease[37]

Data from 14 observational studies — summarized in Table 1 of our paper[38] — suggest that vitamin D blood levels are inversely correlated with the incidence and/or severity of COVID-19, and the evidence currently available generally satisfies Hill's criteria for causality in a biological system.[39] Our paper[40] also details several features of COVID-19 that suggest vitamin D deficiency is at play in this illness.

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