Is it Mast Cell Activation Syndrome or Parasitic Infection? A Cycle of Chronic Illness

One visual illustration of the ways environmental toxicants impact biochemical pathways and the human body.

The Graphic Key (see illustration below)

- ➢ Environmental and industrial toxicants, radioactive elements, and poisons disrupt the microbiome, cause bacteria to pleomorph, alter the inner terrain, and make way for pathogenic infections
- ➢ Organisms pleomorph and become pathogenic and infectious, and alter themselves and the inner terrain to evade the immune system and release metabolites
- ➢ For example, Cestodes, Nematodes, and Trematodes produce and release metabolites that upregulate TH2
- ➢ Elevated TH2 upregulates IL-4, IL-5, IL-13 and IgE cytokines
- ➢ Elevated IL- 4, IL-5, IL-13 and IgE cytokines downregulate TH-1
- ➢ When TH-1 is downregulated, 1) interleukins are unable to regulate immune responses, and 2) IgE reactions increase: patients experience a rise in allergies, histamine, and Mast Cell responses
- ➢ Bring TH-2 into balance: remove parasites and poisons, to reduce inflammation, sensitivity, Mast Cell responses
- ➢ Support mitochondria and drainage, remove pathogens and upstream triggers; chemicals and poisons to support whole body healing
- ➢ The Answer: The Roadmap to Health

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disrupt the microbiome, cause pleomorphism, alter the inner terrain, and make way for pathogenic infections.

Within a disrupted terrain, pathogenic organisms release metabolites that promote their pathogenic cycles, including evading the immune system.

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The Roadmap to Health

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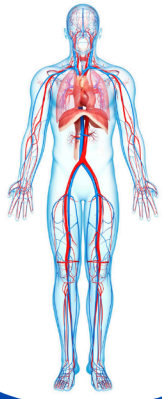

Is it Vitamin D supplement deficiency or chronic toxicity?

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Medical literature shows that those with Vitamin D deficiency are at risk of cardiovascular, respiratory, gastrointestinal, neurological, musculoskeletal, metabolic, cancer, and skin disease.

Medical literature also shows that Vitamin D supplementation can help many of these problems, including treatment of cystic fibrosis and prostate cancer, management of multiple sclerosis and prevention of cancer, and improvement in the mood in women with Type 2 Diabetes and heart function.

We question this belief, and here's why:

For example, someone presents with health issues. We run a Vitamin D lab test. The lab comes back low. What do we do? We give them a Vitamin D supplement. Then we rerun the lab. Their level now shows normal, or we might need to adjust it a little. But do we take them off the Vitamin D supplement now that their level is normal? No. And if we do, what happens? You know, you've seen this before, their Vitamin D level drops. That is, their Vitamin D level is good if they take the supplement, but it drops back to where it was if they go off the supplement.

I call this icing-on-a-mud-pie mentality.

We ask, what's the real problem? What's causing this rise in Vitamin D deficiency?

We believe the Vitamin D deficiency we're seeing in our clinics is not a supplement problem, but the accumulation of chronic toxicity in the body, downstream infections pleomorphism causes, organ dysfunction, and not being able to produce vitamin D metabolites. This biochemical breakdown and physiological dysregulation cause Vitamin D and chronic mineral deficiency, and high body fat.

Let's remember, Vitamin D is synthesized in the skin, and then it is transported to the liver. The liver then takes up the Vitamin D and converts it to 25-hydroxyvitamin D (25OHD). The liver then releases the modified vitamin D, which then goes to the kidneys, where it is further modified to 1,25 - Dihydroxyvitamin D (1,25OH2D).

The 25OHD is the inactive form of Vitamin D. The 1,25OH2D is the active form of Vitamin D. In this active form, Vitamin D acts like a hormone that influences cellular metabolism. Both the liver and the kidneys use enzymes called cytochrome P450 enzymes (CYP enzymes) to modify vitamin D.

But what does medical literature show us about poor Vitamin D production and conversion?

We found the real causes of vitamin D deficiency to be rooted in high body fat mass, mineral deficiencies, metabolic dysfunction caused by chronic infections, lack of daily sunshine, a decrease in UVBs from air pollution, medications, and toxicity.

Research shows

- A very clear correlation between fat mass and low serum 25OHD concentrations. Once it is synthesized in the skin or ingested, ***Vitamin D can be sequestered in body fat stores, making it less bioavailable to people with higher body fat mass.***

- Magnesium deficiency causes Vitamin D deficiency – Magnesium is required for vitamin D synthesis and metabolism.

  Recent findings suggest that high intakes of magnesium may reduce the risk of vitamin D deficiency. Magnesium regulates the activity of critical enzymes in vitamin D metabolism, ***but taking large doses of vitamin D can cause severe depletion of magnesium.***

- Some authorities now believe that low 25OHD is a consequence of chronic inflammation rather than the cause. Research points to a bacterial etiology pathogenesis for an inflammatory disease process, which results in high 1,25OH2D and low 25OHD. ***When immunotherapy is directed at eradicating***

intracellular pathogens, dysregulated Vitamin D metabolism is corrected, which resolves inflammatory symptoms.

- Chronic kidney disease can lead to Vitamin D deficiency in patients with impaired renal function due to reduced synthesis of 1,25OH2D and an increased loss of 25OHD.

- Vitamin D deficiency is common among people with cystic fibrosis and both cholestatic and non-cholestatic liver disease due to decreased absorption of dietary Vitamin D and impaired conversion of Vitamin D to 25OHD.

- Glyphosate suppresses Cytochrome P450 (CYP45) enzymes and amino acid biosynthesis via the gut microbiome – Vitamin D3 synthesis and degradation depend upon various CYP enzymes. Two CYP enzymes in the liver catalyze 25-hydroxylation of vitamin D3 to its active form, and two other CYP enzymes catalyze the breakdown of vitamin D3 in the liver.
    - There is a growing epidemic of vitamin D deficiency in the United States. While this problem is in part due to overaggressive sun avoidance practices, glyphosate's interference with CYP proteins may play a role in disrupting vitamin D3 activation in the liver.
    - Glyphosate's impairment of liver CYP enzymes may interfere with zinc absorption, further depleting the Vitamin D3 getting to the tissues.

- Metabolic Dysfunction – Vitamin D deficiency has been implicated in various diseases, including metabolic syndrome (MetS), which is clinically defined by a set of metabolic and vascular disorders.

- Accumulating evidence on the health benefits from daily sunshine suggests that insufficient exposure is a significant public health problem – Past studies show that insufficient sun exposure may be responsible for 340,000 deaths in the

The United States and 480,000 deaths in Europe per year, and an increased incidence of breast cancer, colorectal cancer, hypertension, cardiovascular disease, metabolic syndrome, multiple sclerosis, Alzheimer's disease, autism, asthma, Type 1 diabetes, and myopia.

Oral vitamin D research shows supplementation does not prevent the above conditions; serum 25OHD as an indicator of vitamin D status may be a proxy for and not a mediator of the beneficial effects of sun exposure.

In summary, there are several causes for Vitamin D deficiency, but it's not a lack of supplementation. Vitamin D deficiency's upstream cause is found in the accumulation of chronic toxicity in the body, downstream infections pleomorphism causes, organ dysfunction, and not being able to produce vitamin D metabolites. This biochemical breakdown and physiological dysregulation cause Vitamin D deficiency, chronic mineral deficiency, and high body fat.