

Entry Level Clinical Nutrition Part X

Micronutrient imbalances:
Vitamin D

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***Quality of life issues are the
major concerns more than
ever now.***

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Entry Level Clinical Nutrition – Part 10

Dr. Jeff Moss

Summer of Work Exposes Medical Students to System's Ills - NYTimes.com Page 1 of 3

The New York Times

September 9, 2009

Summer of Work Exposes Medical Students to System's Ills

By KEVIN SACK

SEATTLE — This summer, medical students from the University of Washington took a long look under the hood of the health care system they are about to inherit, and many returned to campus last week with their eyes wide open and their idealism tempered.

Jacob R. Olgard, a third-year medical student, said: "You have to be realistic about the system. It's not perfect, but it's not as bad as you think it is."

Summer of work exposes medical students to system's ills, *The New York Times*, September 9, 2009

The students learned not only to deliver babies and suture wounds, but also to order unnecessary tests as protection against lawsuits, to hector specialists into seeing Medicaid patients, to match patients with prescriptions on Wal-Mart's \$4 list. And they saw firsthand what Mr. Olgard called "a tidal wave of chronic disease" — diabetes, hypertension, obesity, depression — that left many questioning how much any one physician could really accomplish.

"I often wondered what we were actually doing to help people," Mr. Olgard said.

Not surprisingly, the students also saw a "tidal wave of chronic illness..."

"You have to be realistic about the system. It's not perfect, but it's not as bad as you think it is."

For 10 years, the University of Washington School of Medicine has dispatched its rising second-year students across the Northwest to provide primary care in rural or underserved areas. The monthlong program is elective, but a record number of students took part this year — 117 from a class of 216.

The school has a cooperative agreement to train medical students from Alaska, Idaho, Montana and Wyoming, none of which has a medical college of its own. The summer program is one of several vehicles, along with loan forgiveness endorsements, designed to lure students back to their home states to practice in areas with a shortage of primary care doctors.

This year, the students set out as negotiations were intensifying in Washington over wholesale changes to the health care system they will soon be joining. Whether there will be improvements to the system remains to be seen, but few students returned thinking there was little to fix.

http://www.nytimes.com/2009/09/09/health/policy/09medschool.html?_r=1&th=&mc=th&... 9/9/2009

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**Nestlé Nutrition Workshop Series
Clinical & Performance Program, Vol. 8**

**Nutrition and
Critical Care**

**Luc Cynober
Frederick A. Moore**

**Baracos VE. Overview on
metabolic adaptation to
stress, pp. 1-13.**

"An understanding of the nature of stress is fundamental to the rational design of nutrient mixtures to feed patients whose homeostasis has been altered by one or more stressors."

"All stresses may be presumed to be associated with characteristic modifications in the metabolism of lipids, carbohydrates, amino acids, and micronutrients."

KARGER **Nestlé NUTRITION**

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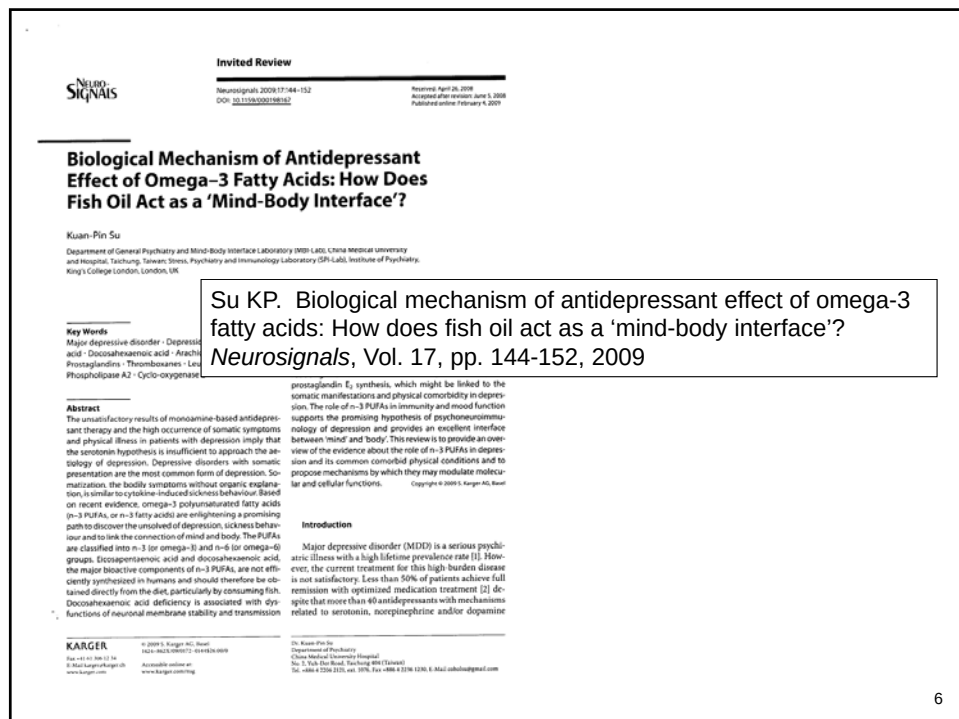
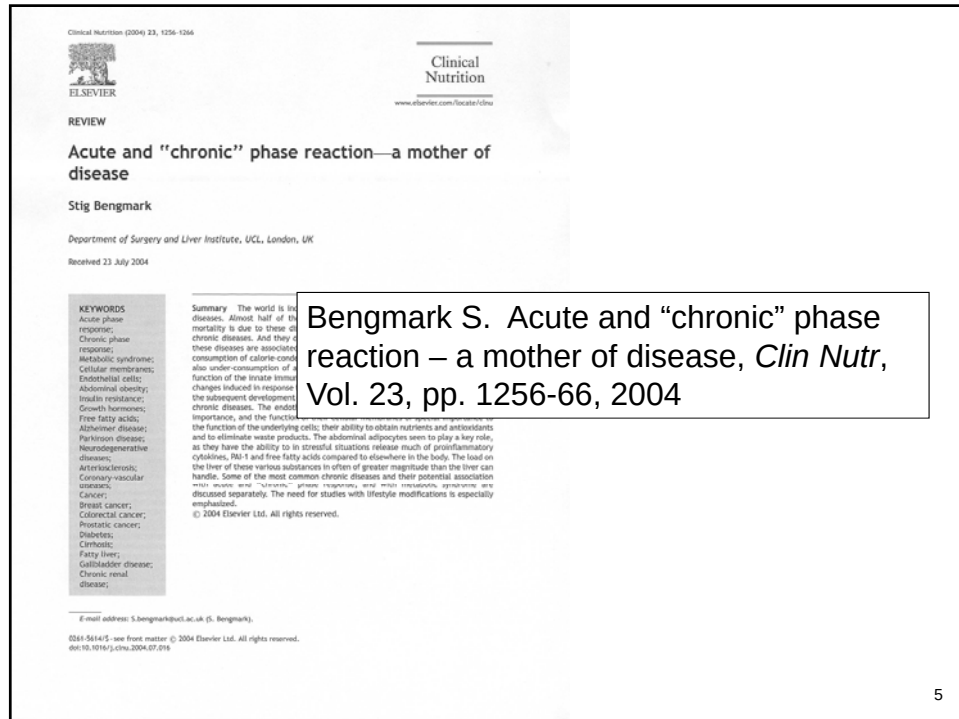


Table 1. Overlapping of symptoms of acute sickness behaviour associated with IFN- α therapy and the somatic symptoms in MDD

Symptoms	Prevalence in IFN- α therapy ^a , %	Prevalence in MDD ^b , %
Fatigue/asthenia	39–90	73
Headache	27–67	33 ^e
Gastrointestinal symptoms	50 ^{c, 1}	34–47 ^f
Psychomotor slowing	40 ^c	59–65 ^f
Insomnia	20–39 ^d	63
Irritability	35 ^d	50
Arthralgia	9–36	31 ^e
Musculoskeletal pain	26–32	62–80 ^{g, 2}
Abdominal pain	15–20	21 ^e
Anorexia	13–19	40
Anxiety	13–18 ^d	57
Poor concentration	14 ^d	51

^a [46], unless otherwise specified; ^b [99], unless otherwise specified; ^c [100]; ^d [101]; ^e [102]; ^f [103]; ^g [104].

¹ Nausea, vomiting, bowel problems.

² Result from depressed inpatient population.

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Key metabolic imbalances seen with the acute phase response

- Metabolic acidosis
- Loss of lean body mass (sarcopenia)
- Insulin resistance
- Inflamm-aging (Increased innate immunity and decreased adaptive immunity)
- Suboptimal caloric intake and carbohydrate:protein ratio (Refeeding syndrome)
- Gastrointestinal dysfunction/gut atrophy
- Deficiencies of key micronutrients such as zinc, selenium, and vitamin D

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Underlying hypotheses of Entry Level Clinical Nutrition:

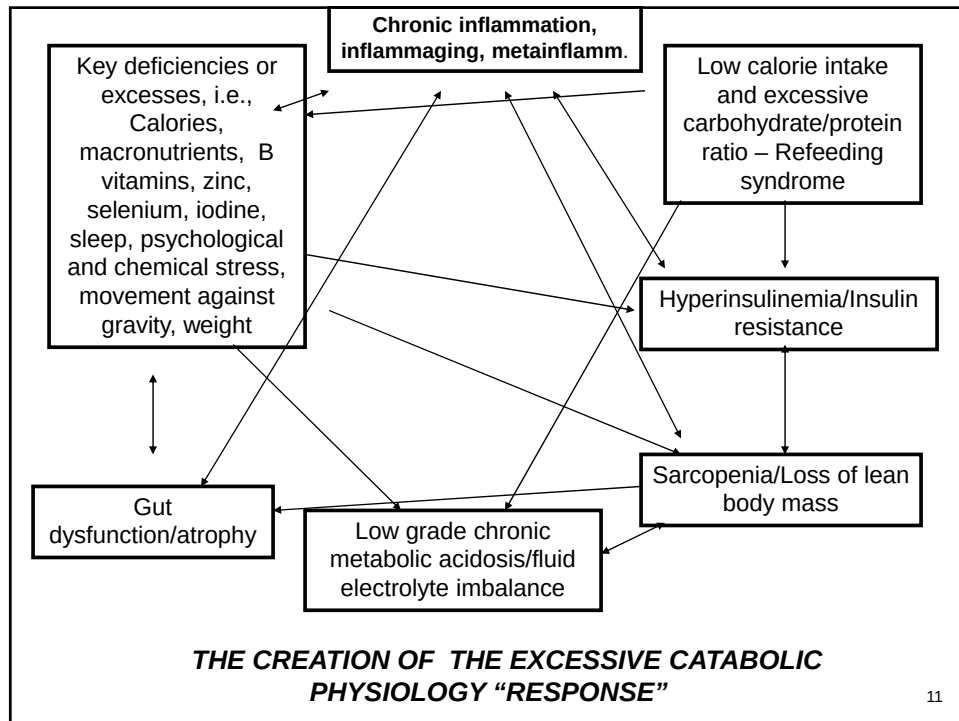
- Chief complaints in chronically ill patients are not diseases but responses that have gone on too long (Allostatic load).
- The metabolic imbalances that combine to form this response have been well defined by critical care nutritionists.

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Entry Level Clinical Nutrition:

A new model of functional
medicine that incorporates
allostatic load and the “chronic”
acute phase response

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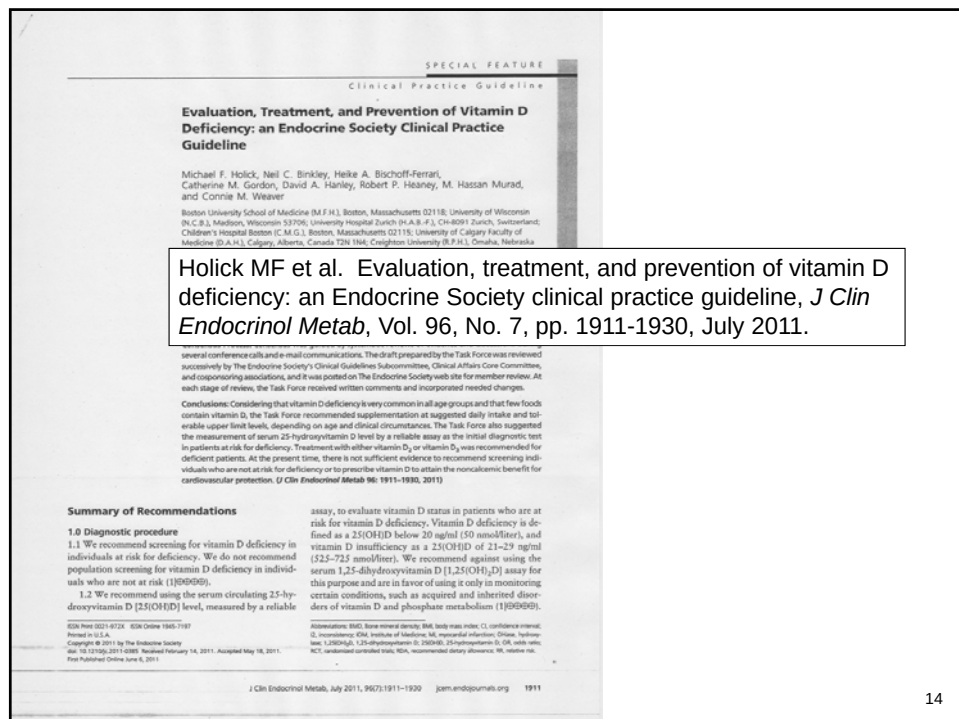
Vitamin D controversies

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Major vitamin D controversies

- What is the optimal level of supplementation?
- D₂ versus D₃: Which is best?
- Is serum 25 (OH) D measurement ideal?

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PART 2A • VITAMINS

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Vitamin D

Nomenclature and biochemistry

Vitamin D is a fat-soluble vitamin that functions as a prohormone (hormone precursor). Vitamin D₃ (also known as cholecalciferol) occurs naturally in fish and in small amounts in a few other foods (e.g., cheese, egg yolk, and beef liver), and is synthesized after exposure to light. Vitamin D₂ (also known as ergocalciferol) is found only in irradiated ergosterol.

Vitamin D itself is biologically inactive, and must undergo two hydroxylation reactions to become active. Vitamin D is first hydroxylated in the liver to form 25-hydroxyvitamin D (25(OH)D). It is further hydroxylated in the kidney to 1,25-dihydroxyvitamin D (1,25(OH)₂D), the biologically active form of the vitamin, which functions as a steroid hormone.

Vitamin D enhances the intestinal absorption of calcium and phosphorus, promotes bone mineralization and remodeling, and is involved in regulating serum calcium and phosphorus levels. Vitamin D also plays a role in neuromuscular function and influences cellular growth and differentiation. As a modulator of immune function, vitamin D may help prevent both infectious and autoimmune diseases. In addition, vitamin D appears to enhance the secretion and action of insulin.

Throughout most of human history, vitamin D was obtained almost exclusively through cutaneous biosynthesis, since typical diets contained little or no vitamin D. Moreover, people who are exposed to adequate amounts of sunlight do not require a dietary source of the vitamin. For these reasons, it has been debated whether vitamin D should be classified a nutrient. However, there is no question that vitamin D becomes an essential nutrient when sunlight exposure is inadequate.

Clinical indications

The increase in the serum 25(OH)D concentration in response to vitamin D supplementation becomes less pronounced. This blunting of the serum 25(OH)D response may be due to an age-related decrease in vitamin D absorption, but it could also reflect a decrease in 25-hydroxylation by the liver.¹ Studies in infants suggest that iron deficiency leads to impaired absorption of vitamin D, and that correction of iron deficiency improves vitamin D absorption.² Vitamin D malabsorption may also occur in individuals with chronic liver disease, pancreatic insufficiency, and diseases of the small intestine (e.g., celiac disease, Crohn's disease).

Vitamin D is excreted primarily in the bile, and some vitamin D metabolites are excreted in the urine.

Table 28.1. Vitamin D may be useful for preventing and/or treating the following conditions:

Bone pain	Stomach loss
Respiratory distress	Influenza
Burns	Myopathy
Cancer	Osteomalacia
Chilblains	Osteoporosis
Cold	Paget's disease
Congestive heart failure	Polycystic ovary syndrome
Critical illness	Rickets
Dermatitis	Seasonal affective disorder
Diabetes	Tuberculosis
Fracture	

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Basic information

- “Vitamin D is a fat-soluble vitamin that functions as a prohormone (hormone precursor).”
- “Vitamin D₃ (also known as cholecalciferol) occurs naturally in fish and in small amounts in a few other foods (e.g., cheese, egg yolk, and beef liver), and is synthesized in the skin from 7-dehydrocholesterol after exposure to sunlight or other sources of ultraviolet light.”
- “Vitamin D₂ (also called ergocalciferol) is produced by irradiation of ergosterol, a sterol present in fungi.”

Basic information

- “Vitamin D itself is biologically inactive, and must undergo two hydroxylation reactions to become active.”
- “Vitamin D is first hydroxylated in the liver to form 25-hydroxyvitamin D (25[OH]D).”
- “It is further hydroxylated in the kidney to 1,25-dihydroxyvitamin D (1,25[OH]₂D, the biologically active form of the vitamin, which functions as a steroid hormone.”

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Basic information

- “Vitamin D enhances the intestinal absorption of calcium and phosphorus, promotes bone mineralization and remodeling, and is involved in regulating serum calcium and phosphorus levels.”
- “Vitamin D also plays a role in neuromuscular function and influences cellular growth and differentiation.”
- “As a modulator of immune function, vitamin D may help prevent both infections and autoimmune diseases.”

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Basic information

“In addition, vitamin D appears to enhance secretion and action of insulin.”

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Absorption

- “The absorption of vitamin D is enhanced by the presence of bile and fat in the intestinal tract.”
- “With advancing age, the increase in the serum 25(OH)D concentration in response to vitamin D supplementation becomes less pronounced.”
- “This blunting of the serum 25(OH)D response may be due to an age-related decrease in vitamin D absorption, but it could also reflect a decrease in 25-hydroxylation by the liver.”

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Excretion

- “Vitamin D is excreted primarily in the bile, and some vitamin D metabolites are excreted in the urine.”

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Definition of deficiency

- “Traditionally, the lower limit of normal for serum 25(OH)D (the most frequently used indicator of vitamin D status) was 10-15 ng/ml (25-37.5 nmol/L), depending on the laboratory.”
- “More recently, some investigators have recommended that vitamin D deficiency be defined as a serum 25(OH)D level <20 ng/ml (<50 nmol/L) and that vitamin D insufficiency (a milder form of vitamin D deficiency) be defined as a level <30 ng/ml (<75 nmol/L).”

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Is vitamin D deficiency only seen in cooler climate areas?

- “Low or suboptimal vitamin D status is common even in regions with abundant amounts of sunlight, such as Arizona and Florida.”

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Risk factors for deficiency

- “Risk factors for vitamin D deficiency include living above 35 degrees latitude or in areas with tall buildings or a lot of atmospheric pollution; being elderly or obese; having dark skin; avoiding sunlight exposure (by staying indoors, wearing protective clothing outdoors, or using sunscreen); having low dietary vitamin D intake; have a condition that causes malabsorption (such as primary biliary cirrhosis, Crohn’s disease, pancreatic insufficiency, small-intestinal bacterial overgrowth, or a history of gastrointestinal surgery); taking vitamin D-depleting drugs (such as anticonvulsants);...”

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Risk factors for deficiency

- “...being an ethnic minority living in Europe; and being an exclusively breastfed infant not receiving vitamin D supplements.”

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Assessment of vitamin D status

- “Although $1,25(\text{OH})_2\text{D}$ is the biologically active form of vitamin D, the serum $1,25(\text{OH})_2\text{D}$ level is not a reliable indicator of vitamin D status.”
- “That is because vitamin D deficiency results in a compensatory increase in the concentration of parathyroid hormone, which increases the renal production of $1,25(\text{OH})_2\text{D}$.”

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Assessment of vitamin D status

- “Consequently, serum $1,25(\text{OH})_2\text{D}$ levels are often normal or even elevated in people with vitamin D deficiency.”
- “The serum vitamin d concentration is also not a reliable indicator of vitamin D status, because its serum half-life is only about 24 hours.”

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Assessment of vitamin D status

- “Therefore, serum vitamin D levels are dependent largely on recent vitamin D intake and recent sunlight exposure.”
- “In contrast, the serum half-life of $25(\text{OH})\text{D}$ is about 3 weeks, which makes this metabolite more reliable than vitamin D itself as an indicator of long term vitamin D exposure.”

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Problems with serum 25(OH)D measurement

- “One problem with this test is that substantial variations have been encountered from one laboratory to another and with the use of different methods of assessment.”
- “Another complicating factor is that 25(OH)D is only one of more than 50 metabolites that have been identified.”

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Problems with serum 25(OH)D measurement

- “True vitamin D status (as it relates to the regulation of calcium and phosphorus metabolism and to various other biological roles of the vitamin) might therefore be a function of complex interactions between many different vitamin D metabolites.”

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Problems with 25(OH)D measurement

- “If that is the case, then different individuals could have different serum 25(OH)D ‘set points’ for adequate or optimal vitamin D status, based on factors such as individual differences in the synthesis and degradation of various vitamin D metabolites, and differences in the binding affinity of these metabolites to vitamin D receptors.”

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Problems with serum 25(OH)D measurement

- “Serum 25(OH)D may be a less reliable indicator of vitamin D status when large doses of vitamin D are being given than when smaller doses are being used.”
- “That is because at dosages below about 2,000 IU/day, the rate of conversion of vitamin D to 25(OH)D is proportional to the amount of vitamin D administered.”

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Problems with serum 25(OH)D measurement

- “However, at higher dosages, hepatic 25-hydroxylases become saturated, and the conversion of vitamin D to 25(OH)D does not increase linearly with increasing vitamin D doses.”
- “Instead, large quantities of vitamin D are stored as the native compound, presumably in body fat, and are slowly released to be converted to 25(OH)D.”

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Problems with serum 25(OH)D measurement

“This increase in body stores of vitamin D is not reflected in serum measurements of 25(OH)D.”

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The problem with using 25(OH)D as the “gold standard” for chronic illness

- “With regard to the concept of optimizing 25(OH)D levels in order to reduce the incidence of certain chronic diseases, the supporting evidence has been derived largely from observational studies, in which associations were found between serum 25(OH)D levels and health outcomes.”

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The problem with using 25(OH)D as the “gold standard” for chronic illness

- “Four different cytochrome P₄₅₀ enzymes are thought to be capable of hydroxylating vitamin D.”
- Since cytochrome P₄₅₀ enzymes also play a role in detoxifying xenobiotic chemicals, people who achieve high 25(OH)D levels in response to vitamin D supplementation may have more robust detoxification mechanisms than those whose serum 25(OH)D response is less pronounced.”

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The problem with using 25(OH)D as the “gold standard” for chronic illness

- “Moreover, hydroxylase enzymes play a role in the synthesis of dehydroepiandrosterone and estriol, both of which may have beneficial effects on human health.”
- “Therefore, the achievement of a high serum 25(OH)D level in response to vitamin D supplementation may be just a marker for the presence of other health-promoting biochemical capabilities.”

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Adverse effects of supplementation

- “Since vitamin D is fat-soluble, it may accumulate in tissues and produce toxic effects after prolonged administration of large doses.”

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Adverse effects of supplementation

- “Most of the symptoms of vitamin D toxicity are due to hypercalcemia, although one investigator stated that signs of toxicity can occur in the absence of hypercalcemia.”
- “According to a review article, all published cases of vitamin D toxicity with hypercalcemia involved an intake of at least 40,000 IU/day.”
- “However, there is one case report in which vitamin D toxicity occurred at a dosage of 25,000 IU/day.”

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Tolerable upper intake levels

- “The ULs for vitamin D are based on levels of intake that can apparently be consumed indefinitely by healthy people without causing hypercalcemia.”
- “In establishing the ULs, the Food and Nutrition Board (FNB) noted a study in which supplementation of healthy adults with 3,800 IU/day of vitamin D for 3 months resulted in a mean serum calcium concentration above the normal range.”

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Tolerable upper intake levels

- “A dosage of 2,400 IU/day for 3 months also significantly increased the mean serum calcium concentration but the values remained within the normal range.”
- “The FNB therefore established 2,400 IU/day as the no-observed-adverse-effect level.”

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Tolerable upper intake levels

“Dividing by an ‘uncertainty factor’ of 1.2 resulted in a UL of 2,000 IU/day.”

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Are dosages above the ULs safe?

- “Some investigators have argued that the ULs are excessively conservative, and that 10,000 IU/day is a safe level of intake for most adults.”
- “The argument that 10,000 IU/day of vitamin D is generally safe is based on 2 main points.”

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Are dosages above the ULs safe?

- “First, hypercalcemia has not been observed in studies in which doses up to 10,000 IU/day were given (the study cited above in which 3,800 IU/day produced hypercalcemia was dismissed as not credible, because it was not consistent with the preponderance of the evidence.”

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Are dosages above the ULs safe?

- “Second, whole-body sunlight exposure results in the production of at least 10,000 IU/day of vitamin D; and people who obtain large amounts of sun exposure do not exhibit signs of vitamin D toxicity or other long-term adverse effects (other than photoaging of the skin and skin cancer).”

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Are dosages above the ULs safe?

- “However, the contention that the UL for adults should be raised to 10,000 IU/day has 4 important weaknesses.”
- “First, studies of high-dose vitamin D supplementation were of relatively short duration.”
- “Second, the absence of hypercalcemia from a given dosage of vitamin D is not proof of safety.”

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Are dosages above the ULs safe?

- “Third, it is not clear whether human skin really can synthesize as much as 10,000 IU/day of vitamin D.”
- “And fourth, the physiological effects of sunlight exposure differ substantially from those of orally administered vitamin; consequently, it may be inappropriate to compare the safety of these different methods of obtaining vitamin D.”

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More concerns about long term safety

- “Furthermore, the absence of hypercalcemia from a particular dosage of vitamin D does not necessarily indicate that that dosage is safe.”
- “The human body tightly regulates serum calcium levels by several different mechanisms, and it is likely that hypercalcemia occurs only after all calcium-regulating mechanisms have been overwhelmed.”
- “One way in which the body prevents hypercalcemia is by increasing urinary calcium excretion. High vitamin D intake might therefore increase the risk of developing kidney stones, even if serum calcium levels remain normal.”

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More concerns about long term safety

- “While vitamin D-induced hypercalciuria appears to be uncommon, it would be prudent to monitor both serum and urinary calcium levels in patients being treated with large doses of vitamin D for long periods of time, particularly if they are also taking a calcium supplement.”

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More concerns about long term safety

“Atherosclerosis and arterial calcification might also result from long-term consumption of large amounts of vitamin D.”

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“Can human skin produce 10,000 IU/day of vitamin D?”

- “One study used to support that assertion found that, in elderly vitamin D deficient volunteers, exposure of 5% of body surface area to ultraviolet B irradiation 3 times a week for 12 weeks increased serum 25(OH)D levels about the same amount as oral administration of 400 IU/day of vitamin D.”
- ***“However, there is no clear evidence that one can extrapolate the effects of irradiating 5% of the skin to the effects of irradiating the entire body.”***

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- “Another study found that a one-time exposure of young adults to 1 minimal erythral dose of simulated sunlight (i.e., the minimum amount that produces redness of the skin) was equivalent to oral administration of 10,000 – 25,000 IU of vitamin D₂, as determined by changes in circulating vitamin D levels.”

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- “However, results from a single-dose study are of doubtful relevance to long-term vitamin D homeostasis, because repeated exposure of the skin to sunlight results in photodegradation of the vitamin D that has not yet escaped into the circulation.”
- “Thus, the net amount of vitamin D produced on subsequent days of sun exposure may be substantially less than the amount produced on the first day.”

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“Sunlight exposure and oral vitamin D are not the same”

- “At least one of the photodegradation products of vitamin D (5,6-*trans*-vitamin D) has demonstrated biochemical effects similar to those of 1,25(OH)₂D in rats, although 5,6-*trans*-vitamin D is 20-40 times less potent than 1,25(OH)₂D.”
- “As a weak vitamin D agonist, 5,6-*trans*-vitamin D might compete with 1,25(OH)₂D for binding to vitamin D receptors, and thereby function as a regulator of vitamin D activity.”

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“Sunlight exposure and oral vitamin D are not the same”

- “In addition to producing vitamin D degradation products, sun-exposed skin synthesizes corticotropin-releasing hormone (CRH). CRH, which is better known as a hypothalamic hormone, has a wide range of physiological effects, some of which might modulate the actions of vitamin D.”

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“Sunlight exposure and oral vitamin D are not the same”

- “Some of these sunlight-induced responses in the skin...might decrease the deleterious effects of vitamin D.”
- “Since there is no evidence that any of these ancillary effects of sunlight exposure occur with oral administration of vitamin D, one cannot draw conclusions regarding safety of ingested vitamin D from data on the safety of vitamin D synthesized by the skin.”

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“Are the ULs too high?”

- “...it would seem premature to assume that almost all healthy individuals (particularly those with abundant sunlight exposure and those who are taking calcium supplements) can safely ingest 2,000 IU/day of vitamin D.”
- ***“This point does not negate clinical observations that some patients fare best with vitamin D in dosages of 2,000 IU/day or more.”***

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“Safe and effective vitamin D levels: what to make of it all”

- ***“The available evidence suggests that currently recommended vitamin D intakes are not sufficient to promote optimal health.”***

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“Safe and effective vitamin D levels: what to make of it all”

- “I frequently recommend 800-1,200 IU/day of supplemental vitamin D, and sometimes more, depending on age, body mass index, skin color, and amount of sunlight exposure.”

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“Safe and effective vitamin D levels: what to make of it all”

- “However, the safety and efficacy of giving healthy people large doses of vitamin D (such as more than 2,000 IU/day) for the sole purpose of reaching a target serum 25(OH)D level have not been established.”
- ***“For patients with vitamin D-responsive diseases and for those who are at increased risk of developing these diseases, the potential benefits of high-dose vitamin D supplementation should be weighed against the risks.”***

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“Vitamin D₃ versus vitamin D₂”

- “Vitamin D₃ has been found to be more potent than vitamin D₂. In one study, both forms of vitamin D raised serum 25(OH)D concentrations by the same amount during the supplementation period.”
- “However, 25(OH)D levels continued to rise for 14 days after vitamin D₃ supplementation was discontinued.”

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“Vitamin D₃ versus vitamin D₂”

- “In contrast, after vitamin D₂ was discontinued, 25(OH)D levels returned to the pretreatment values within 14 days.”

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“Vitamin D₃ versus vitamin D₂”

- “A second difference between vitamin D₃ and vitamin D₂ is in their metabolism.”
- “Vitamin D₂ is metabolized to 25(OH)D₂, which does not bind as efficiently to vitamin D-binding proteins in plasma as does the respective vitamin D₃ metabolite, 25(OH)D₃.”
- “Consequently, vitamin D₂ may be less available to some tissues than vitamin D₃.”

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“Vitamin D₃ versus vitamin D₂”

- “Vitamin D₂ is also less stable than vitamin D₃, particularly when exposed to variations in temperature and humidity.”
- “Consequently, vitamin D₂ has a shorter shelf-life than vitamin D₃.”
- “Because of its poor stability, vitamin D₂ preparations might contain relatively high concentrations of potentially toxic degradation products.”

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“Vitamin D₃ versus vitamin D₂”

- “When switching from high-dose vitamin D₂ to vitamin D₃, it is important to remember that vitamin D₃ is more potent than vitamin D₂.”
- “A 50,000-IU dose of vitamin D₂ may be roughly equivalent to 5,000-15,000 IU of vitamin D₃.”

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“Vitamin D₃ versus vitamin D₂”

- “Because the relative potency of these forms of vitamin D may differ from one person to another, patients being switched from vitamin D₂ to vitamin D₃ should have their serum 25(OH)D level monitored until the optimal dosage is determined.”

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“Vitamin D from sunlight”

- “According to one investigator, exposure of the arms and legs or the hand, arms, and face to sunlight for 5-15 minutes 2-3 times a week between 10 a.m. and 3 p.m. during the spring, summer, autumn usually results in adequate vitamin D production by individuals with skin type II (fair skinned) or III (darker Caucasian).”
- “That amount of sunlight exposure is 25% of what would cause a minimal erythema response.”
- “After the initial 10-15 minutes of sunlight exposure, application of a sunscreen with a sun protection factor (SPF) of at least 15 is recommended.”

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The relation between acute changes in the systemic inflammatory response and plasma 25-hydroxyvitamin D concentrations after elective knee arthroplasty¹⁻⁴

David Reid, Barry J Toole, Susan Knox, Dinesh Talwar, Johann Harten, Denis St J O'Reilly, Scott Blackwell, John Kinneir, Donald C McMillan, and A Michael Wallace

ABSTRACT
Background: Studies indicate that low plasma 25-hydroxyvitamin D (25(OH)D) is associated with a range of disease processes, many of which are inflammatory. However, other lipid-soluble vitamins decrease during the systemic inflammatory response, and this response may confound the relation between 25(OH)D and inflammation.

Objective: The objective was to determine the relation between acute changes in the systemic inflammatory response and plasma 25(OH)D concentrations after elective knee arthroplasty.

Design: Patients had venous blood samples taken preoperatively (day 0), and at days 1, 2, 3, 4, 5, and 6 postoperatively. Plasma concentrations of 25(OH)D, C-reactive protein (CRP), and interleukin-6 (IL-6) were measured.

Results: Preoperative plasma 25(OH)D concentrations were significantly lower (20% and 30%, respectively; $P < 0.01$) in patients with CRP concentrations ≥ 10 mg/L and IL-6 concentrations ≥ 1 pg/mL, respectively.

Conclusion: Plasma concentrations of 25(OH)D decrease after an inflammatory insult and therefore are unlikely to be a reliable measure of 25(OH)D status in subjects with evidence of a significant systemic inflammatory response.

INTRODUCTION
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Thank you!!

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