November 2019 - Mid-Month Bonus Newsletter

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Nutrient of the Month: Vitamin D

We once thought vitamin D really was a vitamin that was all about bones—but it’s neither. Endocrinologist Michael Holick, MD, PhD reminds us that just a century ago, most children in industrialized Europe experienced how overt deficiency of this sunlight-triggered, liver-converted, mainly kidney-activated, tissue receptor-gobbled essential hormone caused bone pain, deformity, and fractures, until sunbathing, cod liver oil, and, most especially, milk fortification with “vitamin” D virtually eradicated rickets. We now recognize that it plays a multitude of roles throughout the body, yet a billion or more people are probably deficient or insufficient in it, leading to calls for a more thoughtful approach to food fortification.

Those most at risk for deficiency/insufficiency include the following, particularly in combination: obese, post-bariatric surgery, older, darker-skinned, with chronic inflammation, living at higher latitudes, during winter, pregnant and lactating women, infants, children, teens, young adults, those taking anticonvulsant, glucocorticoid, or AIDS-related drugs, and those with gene variants negatively affecting vitamin D metabolism (such as in the DHCR7, GC, and CYP2R1 genes, among others). Major food sources of vitamin D include cod liver oil, mushrooms, fish (especially cod, carp, mackerel, catfish, trout, and halibut), dietary supplements, and fortified foods.

The sunshine hormone deserves its own encyclopedia: it’s a derivative of a metabolite of cholesterol, a seasonal hormone having receptors throughout the body, a drug in certain forms, a modified steroid, a transcription factor, an epigenetic modulator, and a nutrient. Vitamin D is a bit of a virtuoso micro-manager of genetics regulation, and via its generous body distribution of receptors, it affects a broad swath of physical, metabolic, cognitive, and mood-related functions. As a transcription factor, it influences the reading
of many genes, while as a post-translational epigenetic regulator, it additionally fine-
tunes how genes are silenced or unsilenced as well as the molecular shapes of their
gene products (regulatory and structural proteins). It also initiates its own catabolism,
thus even regulating itself.

Dr. Holick has described how exquisitely crucial vitamin D is around pregnancy due to
developmental and trans-generational effects, and one of his studies found that even
among healthy, D-supplemented new mothers and their infants, most were deficient.
Vitamin D directly or indirectly regulates up to 5% of the human genome (up to ~1250
genes), and blood levels of vitamin D can significantly impact:

- how well muscle mass and strength are maintained, especially during aging (and
  thus frailty and falls)
- insulin secretion, blood lipid levels, weight gain, and cardiometabolic function
- cell life cycles—cell growth, proliferation, differentiation, cellular aging processes,
  and cell death
- immune balance, cytokine production, incidence of infectious diseases, and
  whether or not an immune response is resolved or is perpetuated, potentially
  encouraging chronic inflammation or autoimmunity
- cardiovascular disease and events, and parathyroid hormone levels, which
  influence their development
- all-cause mortality (and thus longevity)
- risks for asthma, inflammatory bowel disease, and several types of cancer
- mood and neurocognitive function
- (mothers’ levels) telomere length in newborns
- endothelial function and control of chemical mediators of vascular tension
- bone building, remodeling, resorption, and many bone conditions
- intestinal calcium absorption, renal reabsorption of calcium, and blood calcium and
  phosphate levels
- autoimmune conditions like multiple sclerosis, type 1 diabetes, rheumatoid
  arthritis, and thyroiditis

The Vitamin D Council has prepared dozens of helpful research summaries on how
vitamin D status relates to specific health conditions, and a 2014 Lancet study suggests
that a low vitamin D blood level may itself constitute a biomarker for overall poor health.
Preliminary research revealed that vitamin D levels may relate to gut microbiome
composition, though further work is needed to characterize interactions in healthy and
unhealthy populations.

Concerns about the safety of spending time in the sun and of taking active, preformed
vitamin D have a lot to do with its continued widespread lack, but the work of Dr. Holick
and colleagues clarifies that incidence of vitamin D intoxication (blood 25-hydroxyvitamin
concentrations >150 ng/ml) is much rarer than insufficiency (now defined as blood levels
<30 ng/ml, though Dr. Holick and others consider 40-60 ng/ml preferable), and mostly
occurs as result of drug/supplement misuse or formulation errors; blood levels of up to
100 ng/ml are considered safe. Supplementation with 2000-4000 IU (50-100 mcg) of
vitamin D daily will generally increase D-deficient adults’ blood levels to over 30 ng/ml,
while 1000 IU (40 mcg) daily will fail to do so for many adults at risk for deficiency; taking
1500-2000 IU (37.5-50 mcg) daily may therefore represent a reasonable supplemental
level for adults, though specific recommendations depend on individual lifestyle, health,
and genetic and risk factors. For children, recommendations are additionally influenced
by weight and age.

During summer, increases in D blood levels average around 10-20 ng/ml in white-skinned
individuals, peaking at an increase of ~35 ng/ml for those spending ≥300 hours/month in
sunlight, but D blood levels decline considerably within 1-2 months after this sunlight
exposure, reinforcing the need for dietary sources to help maintain blood levels year-
round. For those with no specific reason for avoiding sunlight, some combination of
limited sun exposure and D supplementation may be the best means of achieving
optimal circulating levels, and since genetic variants can greatly influence vitamin D
usage (and may even have different effects in men and women), periodic blood testing
is most reliable for assessing functional D status, though marked individuality in
molecular responsivity to vitamin D has led some researchers to suggest development of
a Vitamin D Response Index to reflect low, medium, and high response patterns. A
mobile application named dminder is available to help gauge individuals’ sun exposure
and track overall D intake in order to improve D status while minimizing risk of sunburn.
Resource: Volume One of the Encyclopedia of "Vitamin" D

Vitamin D is many things to many cells—especially to their genes. But it doesn’t act as a cofactor for essential reactions, as vitamins do; it is actually a hormone that binds to “vitamin” D receptors found in cells throughout the body, where it exerts direct, multilevel effects on gene regulation.

The Vitamin D Council is a non-profit organization whose founder, John Cannell, MD, has been communicating significant research findings on vitamin D since 2003. Dr. Cannell has personally written dozens of helpful blogs that increase awareness about this often-misunderstood hormone/nutrient, such as how the body metabolizes it, which ultraviolet light wavelengths trigger skin D production, the rationale behind his personal intake recommendations for adults and children, and others; it is good to see the multifaceted topic of autism examined from numerous angles.

The site presents a multitude of facts and research organized into many health conditions related to vitamin D status, like Parkinson’s disease, asthma, cognitive impairment, and osteoporosis, in addition to many others. It also features a growing catalogue of informative podcast interviews and talks and even a forum for discussing related issues and directing questions to Dr. Cannell. A generous set of valuable offerings worth checking into regularly!

A Different Strain of the Same Skin Microbe May Render a Commensal Beneficial

One square centimeter of skin is home to up to 1 billion microbes, and the relative balance among these species may have much to do with infectious and non-infectious skin disease and wound healing. Staphylococcus species are among the most common on the skin’s surface, and commensal species include Staphylococcus epidermidis and Staphylococcus hominis. S. epidermidis is known to produce a number of antimicrobial substances that curb Staphylococcus aureus, Streptococcus species, and other pathogens, and it also helps shape a host’s immune response through influencing development of skin T-cells and expression of antimicrobial peptides.

In a 2018 study investigating the metabolites of skin commensals, the MO34 strain of S. epidermidis was found to block an important step in the development of some skin cancers. Given to animals internally, the strain inhibited the growth of melanoma, and externally, it reduced the incidence of skin tumors induced by ultraviolet (UV) light exposure. This strain was discovered to produce a purine that seemed responsible for these findings, as S. epidermidis strains that did not produce the inhibitor did not show these same protective effects. The researchers found two other S. epidermidis strains on human skin that produce the same purine metabolite, especially on the hands, between fingers, between toes, and heels.

In those with atopic dermatitis, Staph. aureus is often found on skin while S. epidermidis is relatively rare; an August 2019 review describes atopic dermatitis as form of skin dysbiosis with greatly reduced microbial diversity having S. aureus as an opportunistic colonizer that encourages chronic inflammation. The research team that discovered the properties of S. epidermidis MO34 previously identified other S. epidermidis and S. hominis strains that limit the growth of S. aureus, and subjects with atopic dermatitis given topical application of five of these strains showed significant reductions in S.
*Staphylococcus aureus* colonization.

Other skin commensals also show interesting metabolic relationships in atopic dermatitis, and demonstrate the degree to which **commensal species may compete with each other** as well as with potential pathogens. Though proliferation of *Cutibacterium acnes* (previously known as *Propionibacterium acnes*) is associated with acne, it is normally present on the skin at low levels. In those with atopic dermatitis, the abundance of *C. acnes* is inversely correlated to that of *S. aureus*, and *C. acnes* metabolites were found to inhibit the growth of not only *S. aureus* but also *S. epidermidis*. In turn, *S. epidermidis* metabolites were found to inhibit the growth of not only *S. aureus* but also *C. acnes*. Even the serum of those with atopic dermatitis limited *S. aureus* growth more than that of healthy persons, demonstrating the systemic nature of the immune response in this “skin condition.”

While these *S. epidermidis* and *hominis* strains that produce anti-*Staph. aureus* substances are not in the majority of their respective species and *C. acnes* is not a major skin commensal, these studies note that some do commonly occur on human skin, and their presence has an outsized influence on skin health. Exercise, diet, medication, and skin products all affect the skin’s moisture, barrier function, and microbiome, and there is increasing interest in oral as well as **topical probiotic use** in skin issues. Greater understanding of how skin microbes interact and produce particular metabolites under particular circumstances will likely aid personalization of therapies in addressing systemic skin conditions.

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