Independent Research & Further Reading

Guest: Dr Stasha Gominak

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Endocrine Society and Vitamin D

> *“Physicians are being told that they shouldn't be interested in vitamin D. They're actively being told that by the Endocrine Society, which is supposed to be the expert about all hormones.”*

The 2024 Endocrine Society guideline advises empiric vitamin D supplementation for children and adolescents ages 1-18, adults 75 years and older, pregnant people, and people with high-risk prediabetes; it advises against routine supplementation above current dietary reference intakes for healthy adults younger than 75 and against routine 25(OH)D testing in the general population without established indications.

References 1-3.

REM-Only Sleep Apnea Pattern

> *“Most of them have delayed rapid eye movement sleep, no rapid eye movement sleep, or they have rapid eye movement sleep apnea, meaning they only stop breathing... in rapid eye movement sleep.”*

A REM-related sleep apnea pattern usually means apneas or hypopneas occur predominantly or exclusively during rapid eye movement sleep, not throughout the whole night. REM sleep apnea can be described as REM-predominant or REM-isolated, but there is still no standard definition, so prevalence and diagnosis vary across studies.

References 4-7.

Sunscreen, Computers, and Air Conditioning in the 1980s

> *“This is actually a chemical deficiency state that happens when you move indoors.”*

Chemical and neurochemical factors can modify severity in some patients, and they are more central in some central sleep apnea syndromes, but they do not explain sleep apnea as a whole. Obstructive Sleep Apnea usually reflects upper-airway collapse from anatomical compromise plus impaired neuromuscular compensation during sleep, rather than a simple chemical deficiency.

Potential deficiency states such as vitamin D deficiency or alpha-1 antitrypsin deficiency are associated with OSA risk or severity in some studies, but the evidence frames them as contributing factors or comorbid associations, not the core definition of sleep apnea.

References 8-14.

Chronotypes, Genetics, and Night-Shift Work

> *"Although there are genetic mutations that lead to certain timing of sleep, it turns out everybody I was doing this vitamin stuff with would revert back to fall asleep at 10:00 and wake up at 6:00, even the people who were working nights their whole life."*

Genetic differences help shape sleep timing and chronotype, with rare mutations in core clock genes sometimes causing very early or late sleep phases and common variants usually shifting timing by smaller amounts across the population.

Vitamin D receptors are present in brain regions involved in sleep regulation, including the suprachiasmatic nucleus, and vitamin D is implicated in melatonin production pathways that regulate circadian timing. Molecular work also suggests vitamin D signalling can regulate clock-related genes such as BMAL1, PER2, and CLOCK. Human transcriptomic work further supports a connection between vitamin D signalling and circadian biology, but not yet a clinical circadian benefit. In one small supplementation study, vitamin D target genes showed circadian expression patterns and interindividual variability, and the authors concluded there is still no consensus that vitamin D acts as a Zeitgeber.

What the current evidence shows:

- Direct circadian trials in humans are scarce. The strongest direct human study found no significant change in circadian parameters after 4 weeks of 4000 IU/day, despite changes in sleep and mood (Gray et al., 2025).
- Sleep benefits appear more reproducible than circadian benefits, including better sleep quality in meta-analysis and some special populations (Abboud, 2022; Cai et al., 2025; Vesković et al., 2026)

- Deficiency is associated with circadian disruption in observational and animal studies, but that does not prove supplementation reverses it in humans (Arabi et al., 2024; Jung et al., 2019)

- Evidence for specific circadian alignment in shift workers is emerging, but current summaries still emphasise limited long-term and population-specific clinical data (Cai et al., 2025).

References 15-27.

Non-Alcoholic Fatty Liver Disease First Described in the 1980s

> *"Some of them for the first time, fatty liver was first described, non-alcoholic fatty liver, first described in the '80s."*

Non-alcoholic fatty liver disease (NAFLD) refers to liver fat accumulation without significant alcohol use and spans simple steatosis through steatohepatitis, fibrosis, cirrhosis, and liver cancer. NAFLD was first introduced in 1986 by Schaffner. Non-Alcoholic Steatohepatitis (NASH) was described in 1980, when Ludwig defined the inflammatory progressive form of fatty liver in non-drinkers.

Earlier descriptions of fatty liver in people with obesity or diabetes, and without alcohol as the defining cause, appeared before 1980 and 1986. The formal histologic history of NASH begins in 1980 and the term NAFLD in 1986, but reviews state that relevant observations go back to the 19th century.

References 28-30.

Walter Stumpf and Vitamin D Receptors in the Brainstem

> *"He's been writing about vitamin D for 30 years, who has actually published that there are vitamin D receptors in this little stripe along the back of the brain stem that paralyzes us."*

Stumpf found vitamin D targets in brainstem-related circuits, not just calcium tissues, using receptor autoradiography. Stumpf's central finding on this structure was that 1,25-dihydroxyvitamin

D3 showed specific nuclear retention in neurons of the rat hindbrain and spinal cord, consistent with receptor-like binding rather than nonspecific accumulation. He extended that interpretation into a broader brain-pituitary axis, proposing central endocrine-autonomic actions for vitamin D beyond classical calcium regulation. He described vitamin D target neurons across autonomic, sensory, motor, and endocrine brainstem components, alongside forebrain and spinal targets. He argued that these brain targets were part of a wider allocortex-brainstem circuitry linked by the stria terminalis and extending into the midbrain and hindbrain. That framing fits his broader anatomical style, which mapped steroid-sensitive circuits rather than isolated spots.

Stumpf's interpretation was that brain vitamin D acts as a sunlight-related steroid signal, helping regulate seasonal and daily rhythms, neuroendocrine activity, and behaviour. He explicitly argued these central effects were largely independent of calcium homeostasis.

He also linked vitamin D targets to the secretion of serotonin, dopamine, nerve growth factor, and acetylcholine and suggested relevance to psychiatric and neuroendocrine disorders, though these functional claims are more interpretive than the receptor-mapping data.

References 31-37.

Vitamin D as the "Hibernation Hormone"

> *"Walter Stumpf has already put together a beautiful belief system around vitamin D as the hibernation hormone. It allows animals to adapt their body to tolerate winter when there's no food."*

Stumpf repeatedly argued that vitamin D "soltriol" helps organisms adapt vital functions to daily and seasonal solar cycles, but the papers do **not** directly frame it as a hormone for surviving winter starvation or as a dedicated hibernation signal. Stumpf's core idea was a broad "endocrinology of sunlight and darkness" in which skin-derived vitamin D and pineal hormones together align biological activity with changing light conditions. He proposed vitamin D-regulated neural timing circuits in addition to the suprachiasmatic clock, with effects on seasonal and, to a degree, daily biorhythms.

References 38, 39.

Vitamin D Receptor Mechanism and Choline Acetyltransferase

> *"The protein that it makes is an enzyme that makes acetylcholine... it was an enzyme called choline acetyltransferase."*

Activated vitamin D receptor (VDR) does not induce just one protein; in most target cells it triggers networks of target genes that together drive tissue-specific biological responses.

Choline acetyltransferase (ChAT) is the enzyme that synthesises acetylcholine from choline and acetyl-CoA, and it is the most specific marker of cholinergic cells rather than a protein induced by VDR-like signalling. Direct evidence on choline acetyltransferase is narrow: one rat brain study found region-specific increases in ChAT activity after $1,25(OH)_2D_3$ repletion, while the rest of the literature shows that vitamin D signalling usually regulates different target genes in different cell types, so any ChAT effect is likely context-dependent rather than universal.

Across many systems, vitamin D signalling regulates hundreds to thousands of genes, and the overlap between cell types is small. The broader literature suggests that if ChAT is vitamin D responsive, that response probably depends on cell identity, differentiation state, and local vitamin D metabolism rather than being a general VDR output.

References 40-44.

American Academy of Pediatrics Sun Exposure Guidance for Infants

> *"The American Academy of Pediatrics says your baby's skin should not be touched by the sun until they're six months old."*

The American Academy of Pediatrics (AAP) states that infants younger than 6 months should be kept out of direct sunlight. Related pediatric guidance also advises no sunscreen under 6 months, prioritising shade, clothing, and avoidance of direct sun.

UV protection is emphasised because early-life sun exposure contributes substantially to lifetime UV damage. The current recommendation is to meet vitamin D needs through diet and fortified foods rather than sun exposure.

References 45-48.

Sunburn and Premature Skin Aging

> *"Every sunburn that's a bad sunburn that you peel from will age your skin, even if you're 10."*

Evidence supports that intense UV injury adds to cumulative photodamage that can show up later as premature ageing and also raises skin cancer risk. The strongest support is for UV damage accumulating over time, with childhood being a particularly vulnerable window because children receive a large share of lifetime UV exposure early and their skin is biologically more susceptible.

Peeling after sunburn reflects skin injury during healing, and sunburn can produce redness, tenderness, swelling, and blistering, then exfoliation or peeling, with reported long-term effects including premature ageing and pigmentation change.

References 49-58.

Vitamin D Report

> *"They know in 2004, when they gave the final recommendation for the doctors in practice, they know that the studies that have been done so far are rarely done in the way a hormone should be studied... Therefore, you should not do vitamin D levels and you should not give recommendations about vitamin D."*

The guest could be referring to the 2011 Endocrine Society Clinical Practice Guideline on vitamin D deficiency, titled *Evaluation, Treatment, and Prevention of Vitamin D Deficiency*. That is the Endocrine Society vitamin D document that appears in the record, while the newer 2024 guideline is about prevention of disease rather than deficiency.

However, she may also have been referring to the 2011 Institute of Medicine (IOM) vitamin D report, which shifted attention back to skeletal health, set intake targets at 600 IU/day for ages 1–70 and 800 IU/day for older adults, and argued that routine population screening with serum 25(OH)D was not warranted. It also pushed clinicians to stop treating vitamin D as a broad, disease-prevention hormone for healthy people without evidence-based indications.

References 59-61.

The 2012 Gominak-Stumpf Vitamin D and Sleep Hypothesis

> *"We publish an article with Walter in 2012 that says... the pandemic of sleep disorders that are chemically based...."*

Reference 62.

Pantothenic Acid, Coenzyme A, and Cortisol

> *"Pantothenic acid is a chemical that makes this thing called coenzyme A. Coenzyme A is responsible for making cortisol."*

- Pantothenate is repeatedly described as vitamin B5 and the key or obligate precursor for Coenzyme A (CoA) biosynthesis. CoA is then built from pantothenate in five conserved enzymatic steps in many bacteria and eukaryotes, with pantothenate kinase catalysing the first step.

- CoA's role in cortisol production is mainly upstream metabolic support: it is required for lipid and cholesterol synthesis, while cortisol itself is produced by adrenal steroidogenic machinery once cholesterol reaches mitochondria. In the adrenal, the immediate control point for steroid output is cholesterol availability and transfer, not CoA as a direct cortisol-making factor.

Pantothenic acid does make coenzyme A, but coenzyme A is not itself responsible for making cortisol. It supports upstream metabolism needed for cholesterol and steroidogenesis, while cortisol is produced by adrenal steroidogenic machinery.

References 63-70.

Prednisone and Rheumatoid Arthritis in the 1960s

> *"We talk about cortisol all the time, the timing of it, and it's huge in inflammation. So when we learned about prednisone in the 1960s, we got this drug, prednisone, and it made the pain of rheumatoid arthritis, which is terrible, go away in, like, two days.*
> *It looks like cortisol."*

Prednisone and prednisolone are analogues of cortisone and hydrocortisone, with a relatively small structural change in the steroid nucleus, so they were designed to act like endogenous adrenal glucocorticoids such as cortisol. Rheumatoid arthritis (RA) morning pain and stiffness are linked to a nightly rise in inflammatory cytokines, while cortisol levels appear insufficient to counter that inflammation, which is why exogenous glucocorticoids can function partly as a replacement-like anti-inflammatory signal.

Prednisone and prednisolone can improve rheumatoid arthritis symptoms within days, and older clinical studies reported that the effect can appear within hours to a few days rather than weeks. Low-dose nighttime prednisolone produced favourable effects after only 5 days on morning stiffness, joint pain, and inflammatory indices, while morning dosing had smaller effects.

References 71-78.

1950s Pantothenic Acid Deprivation Studies on Prisoners

> *"The articles that she references are from the 1950s in this... lab next to the Iowa State Prison, where they're doing these... experiments on prisoners... if you block pantothenic acid for two weeks, you see four things.*
>
> ***They can't sleep**. Their belly is, they have **belly complaints, bloating**, and all sorts of things. They have a funny **puppet-like gait**, and they have **burning in their hands and feet**. And I go, "This is wonderful." So that was one of the motivations.*
>
> *So the **cortisol link** to pain and the burning in the hands and feet. "*

There were human pantothenic acid (vitamin B5) depletion studies in the 1940s and 1950s, including research conducted at the Iowa State College (now Iowa State University). Some experiments involved volunteers housed in facilities associated with the nearby Iowa State Men's Reformatory in Anamosa, Iowa. At that time, it was not uncommon for nutritional studies to recruit prisoners as research participants, although such practices would not meet today's ethical standards. Researchers induced pantothenic acid deficiency by feeding volunteers a diet low in pantothenic acid and, in some studies, administering a pantothenic acid antagonist (such as ω-methylpantothenic acid).

The 1955 paper describes somnolence (drowsiness) rather than insomnia. At the beginning of the 1959 paper, there is a summary of the established Pantothenic Acid Deficiency symptoms:

- fatigue
- headache
- weakness
- emotional lability
- impaired motor coordination
- paresthesias
- muscle cramps
- nausea
- vomiting
- abdominal cramps

- tachycardia (some subjects)
- orthostatic hypotension (some subjects)

Sleep and Pantothenic Acid Syndrome

Human deficiency studies and reviews support a narrower claim: induced pantothenic acid deficiency can produce sleep disturbances or insomnia as one symptom among several, but the **evidence base is old, small, and not focused on sleep duration itself**.

Gait and Pantothenic Acid Syndrome

The **evidence is still limited to animal models**, and the two species emphasise somewhat different phenotypes: mice showed dragging and dystonic posturing, whereas pigs showed a spastic, goose-stepping gait.

Burning Feet Syndrome and Pantothenic Acid Syndrome

The literature links pantothenic acid deficiency to burning feet syndrome, but direct causal evidence in humans is limited and contested. The strongest controlled human trial found no significant benefit of pantothenol over placebo, which **argues against pantothenic acid deficiency as the main cause in that patient group**.

Cortisol and Pantothenic Acid Syndrome

Pantothenic acid is described as an “anti-stress vitamin” and a precursor of coenzyme A, which supports a biological connection to stress-hormone pathways. Pantothenic acid deficiency is thus reported to reduce cortisol production, while its role in the burning symptom itself remains unproven in the evidence.

References 79-89.

Vitamin D and Vitamin B Interaction

> *“Now I've got it in my head that I've made them use more of the B vitamins in this general way that's isn't that helpful, but I've, uh, I've made them use more B vitamins.”*
>
> *(...)*
>
> *“Vitamin D deficiency changes the intestinal microbiome, reducing B vitamin production in the gut. The resulting lack of pantothenic acid adversely affects the immune system, producing a pro-inflammatory state associated with atherosclerosis and autoimmunity.”*

The guest has published a paper which argues vitamin D deficiency alters the gut microbiome, which then reduces B-vitamin supply. **This is a hypothesis paper** that proposes a mechanistic chain linking vitamin D status, intestinal bacteria, B vitamins, sleep, pain, and inflammatory disease, rather than proving the mechanism in a controlled trial.

The core argument is that humans normally have a commensal relationship with the intestinal microbiome: humans supply vitamin D conditions, and gut microbes supply B vitamins. The paper specifically argues that the normal microbiome includes four major groups and that these groups mutually support one another by excreting B vitamins needed by the others. Vitamin D deficiency is proposed to create an intestinal environment that no longer favours this “healthy foursome", leading to a persistently altered microbiome. The paper then argues that this microbial shift reduces gut production of B vitamins, with a special emphasis on pantothenic acid.

Pantothenic acid is presented as central because it becomes coenzyme A, which the paper describes as necessary for cortisol and acetylcholine production. The proposed chain is that better sleep and cellular repair after vitamin D replacement gradually deplete pantothenic acid stores, creating a secondary deficiency if B vitamins are not replenished. The paper links lower cortisol production to increased arthritic pain and a broader pro-inflammatory state associated with autoimmunity and atherosclerosis. It also links lower acetylcholine availability to reduced parasympathetic activity and relatively unopposed sympathetic tone, which it associates with hypertension, tachycardia, atrial arrhythmias, and cardiovascular risk.

The intervention in this paper combined vitamin D plus B100, so it cannot isolate whether any benefit came from vitamin D, B vitamins, placebo effects, or regression to the mean.

More recent work also points to a bidirectional relationship, where microbiota may influence vitamin D metabolism as well as the reverse, which weakens the paper's mostly one-way narrative.

References 90-93.

Peanut Allergies Before the 1980s

> *"Peanut allergies did not exist before the 1980s. These are all allergy related."*

The earliest definite reference identified here is Blackfan in 1920, describing a 10-year-old whose reactions to nuts included throat burning, vomiting, diarrhea, lip and ear edema, and urticaria. That means peanut or nut allergy did not first appear in the 1980s; the literature in this set places documented allergic reactions to nuts at least six decades earlier.

A later longitudinal study shows those early clinically confirmed peanut cases were being evaluated between 1973 and 1985, using double-blind, placebo-controlled food challenges in 114 children. By the late 1980s and early 1990s, peanut allergy had become more prominent because reports linked peanut to fatal and near-fatal food anaphylaxis.

References 94, 95.

B Vitamins as Bacterial ("Poop") Products

> *"Thiamin, which is B1, has a poop bacteria source and a food source. B2, poop bacteria and a food source... All of them come from the poop."*

Humans primarily obtain thiamine (B1) from food, especially protein-rich foods, fortified grains, meat, eggs, cereal sprouts, rice bran, and beans. Dietary thiamine is absorbed mainly in the small intestine after phosphate groups are removed, although absorption can also occur in the large intestine. Riboflavin (B2) is also an essential nutrient that humans must obtain exogenously from

foods or supplements. Mammals cannot synthesise most B vitamins themselves, so they rely on diet and microbes together.

Many gut microbes can synthesise B vitamins, including thiamine, while others cannot and must import them or obtain them from neighbouring microbes. For thiamine specifically, about half of gut microbes encode de novo synthesis, and predicted B1 producers include Bacteroides, Prevotella, some Clostridium, Lactobacillus, Bifidobacterium, and Fusobacterium. Riboflavin-producing microbes also include lactic acid bacteria and other fermentation organisms, which is why fermented foods can be enriched in B2.

Bacterial production is real for B1 in the colon and for B2 in multiple gut- and food-associated microbes. Gut communities include both prototrophs and auxotrophs, so vitamin cross-feeding occurs between microbes. Some major gut bacteria cannot make B1 and depend on diet or other microbes instead.

Most microbe-mediated vitamin production occurs in the large intestine, and those vitamins can feed other microbes or be absorbed by the host. For B1, bacteria in the colon produce both free thiamine and TPP, and the colon has transport systems that can absorb these forms.

References 96-104.

Fecal Microbiota Transplants for C. diff

> *"As people are dying of things like Clostridium difficile... it's resistant to everything, and they're just dying. They're doing poop transplants and they live."*

For recurrent *Clostridioides difficile* infection (CDI), fecal microbiota transplantation (FMT, "poop transplant") is not fringe care: randomised trials, meta-analyses, and a Cochrane review show it resolves infection more often than standard antibiotics, especially after repeated recurrences. The best-supported claim is for recurrent CDI. Across systematic reviews, overall clinical resolution clusters around 85%–93%, and high-quality evidence supports repeat FMT as more effective than antibiotics alone.

One double-blind randomised trial found donor FMT cured 90.9% versus 62.5% with autologous stool, and every patient who failed autologous FMT and then received donor FMT cleared

subsequent CDI. In severe refractory CDI, a randomised trial found multiple colonoscopic FMT infusions plus vancomycin cured 100% vs 75% with a single infusion plus vancomycin. After an inpatient FMT programme began, CDI-related mortality fell from 10.2% to 4.4% overall and from 43.2% to 12.1% in refractory severe/fulminant cases.

Safety looks favourable in the short term, but serious-event estimates remain imprecise in trials, and long-term risks still require surveillance and strict donor screening. In immunocompromised or transplant populations, overall cure still appears high after repeated treatment, but single-FMT failure is more common, and close monitoring matters.

References 105-113.

The Four Phyla of the Human Microbiome

> *"There are four big groups of bacteria that is the normal human microbiome... we only know about 2% of them... we know there are 200."*

Across human-associated bacteria, the phyla most repeatedly reported as dominant are Firmicutes/Bacillota, Bacteroidetes/Bacteroidota, Actinobacteria/Actinomycetota, and Proteobacteria/Pseudomonadota.

In the gut specifically, the most common phyla are usually Firmicutes, Bacteroidetes, and Actinobacteria, with Proteobacteria, Fusobacteria, Cyanobacteria, and Verrucomicrobia encountered less often. Healthy digestive-tract communities also separate into four compositional groups, but those four groups are body-site clusters, not four bacterial phyla: one stool cluster plus three oral clusters.

Older culture-based work recovered more than 300 gut bacterial species, and later reviews reported more than 400 species by culture and more than 3,000 by molecular methods. A typical intestinal microbiota contains roughly 100–1,000 species, and individuals differ greatly in which species dominate. Large genome-reconstruction studies now report far beyond 200 species: one study recapitulated 4,930 species, with 77% never described before, and another identified 3,796 unknown species-level genome bins lacking any reference genome. Other gut metagenomic studies

found 1,952 uncultured candidate species and 2,058 newly identified species-level OTUs, expanding known gut phylogenetic diversity by 281% and 50%, respectively.

References 114-124.

Vitamin D Deficiency and COVID-19 Mortality

> *"they actually started to realise that the people who were D deficient died in COVID."*

Vitamin D deficiency was consistently associated with higher COVID-19 mortality in observational studies and meta-analyses, with pooled mortality odds ratios around 1.8 to 2.6, and some hospital cohorts reporting larger adjusted hazards or odds ratios. The mortality association becomes less robust after bias-sensitive analyses, and some meta-analyses found adjusted or higher-quality subsets no longer statistically significant.

Lower 25(OH)D was also associated with greater disease severity, including ICU admission, respiratory failure, or advanced radiologic stage, across pooled analyses and hospital cohorts.

Evidence that vitamin D supplementation improves COVID-19 outcomes is more mixed: meta-analyses suggest lower ICU admission or severity, but mortality benefit is clearer in deficient patients than in unselected populations.

This evidence is mostly observational, heterogeneous, and vulnerable to confounding and reverse causation, especially because vitamin D can behave as a marker of illness severity or acute inflammation.

References 125-130.

Vitamin D Effects on Microbiome

> *"And they started to actually look at, oh, is the microbiome playing a role in our immune system? Yes. If I give vitamin D, will the bacterial makeup of my stool change in a positive way? Does the poop bacteria makeup change in a positive way? We know we've seen people who don't have inflammatory disorders, and they have these – this bacterial makeup. Will giving vitamin D change the population? And yes, that's the answer."*

In humans, vitamin D supplementation often changes fecal taxonomic composition or specific taxa, but findings vary by population, dose, and study design. The most repeatable signal is taxon-level change without major diversity change, including shifts in *Lachnospira*, *Blautia*, *Bifidobacterium*, *Ruminococcus*, and related groups. Null fecal findings also occur, especially in small healthy-adult trials, suggesting effects are not universal or may be transient.

The strongest direct RCT evidence is mixed but leans yes. In vitamin D-deficient overweight or obese adults, 16 weeks of cholecalciferol caused no α-diversity or clustering change, yet did shift genus-level composition, increasing *Lachnospira* and lowering *Blautia*. A newer placebo-controlled trial in healthy adults found 12 weeks of 4,000 IU/day altered β-diversity, reduced microbiota stability as serum 25(OH)D rose, changed acute and persistent taxa, and rewired metabolite networks.

Other human studies support shifts in selected taxa more than broad restructuring. In healthy vitamin D-deficient women, supplementation increased microbial diversity, raised the *Bacteroidetes*:*Firmicutes* ratio, and enriched *Akkermansia* and *Bifidobacterium*.

A pilot trial in healthy deficient adults found no lasting fecal change after 8 weeks of vitamin D3 or calcifediol, despite biochemical repletion. In ulcerative colitis, supplementation lowered inflammation but did not change overall diversity, while *Enterobacteriaceae* increased. In *C. difficile* infection, high-dose intramuscular vitamin D increased *Bifidobacteriaceae* and *Christensenellaceae* and lowered *Proteobacteria* during recovery. Combined interventions and non-oral exposure also show modulation: fish oil plus vitamin D3 changed several fecal taxa, and NB-UVB-induced vitamin D increases changed fecal diversity mainly in previously unsupplemented participants.

Vitamin D supplementation does shift the fecal microbiome in many studies, but the effect is usually taxon-specific, context-dependent, and not uniformly large.

References 131-139.

Obesity in Wild vs. Domestic Animals

> *"There are no obese squirrels in my backyard. The only obese animals are cats and dogs that we keep inside."*

Domestic pets have the clearest and highest documented obesity burden, often affecting roughly one-quarter to one-half or more of animals depending on species and definition. In wild animals, obesity appears less commonly documented not because it is absent, but because most available evidence tracks human-influenced wildlife, captive animals, or body-mass shifts rather than true obesity prevalence in untouched wild populations.

In primates, captivity and provisioning are associated with markedly higher body mass than in the wild: across 40 non-human primates, captive-to-wild relative body mass averaged 1.28 in females and 1.24 in males, overlapping human values, and provisioned wild/free-ranging populations looked similar to zoo and research populations.

References 140-146.

Dinosaur Microbiomes and Brain Biochemistry

> *"For 250 million years, the dinosaurs had a microbiome in their intestines. The dinosaurs were very smart. They had the brain like we do. It was exactly the same biochemistry as ours."*

Dinosaurs were an extremely diverse group that lived for about 165 million years. Comparative evidence supports the idea that dinosaurs, especially herbivores, likely had gut microbiomes, but it is indirect. Farlow argued that plant-eating dinosaurs probably used symbiotic gut flora in a hindgut fermentation chamber to digest plant cell walls, with long gut residence times helping microbial breakdown of fibre.

Modern alligators strengthen that inference because they are crown archosaurs with organ-specific, compositionally distinct gut microbiomes, which may preserve archosaur-associated symbioses over time. But bone microbiome work on Centrosaurus shows that microbes recovered from fossil bone are active later colonisers and can obscure endogenous signals, so they do not demonstrate the dinosaur's original gut community.

One analysis argued some theropods had baboon-like neuron counts and therefore flexible cognition, tool use potential, and even culture-building capacity. That interpretation is disputed because revised modelling recovered lower neuron estimates for large theropods and judged neuron counts poor proxies for cognition. Several studies instead place many dinosaurs at avian-like, reptile-range, or uniquely dinosaurian cognition rather than human-like cognition.

Some dinosaurs show evidence of increased encephalisation, sensory specialisation, and complex behaviour, but that does not amount to a consensus for human-like intelligence. Ornithopod and hadrosaur studies report enlarged cerebral hemispheres, higher EQs, and behaviours consistent with herding, communal nesting, and juvenile care.

Theropod-focused work also documents large brains in some maniraptorans, grasping hands, stereoscopic vision, and well-developed sensory systems, which make elevated cognition plausible in some lineages. But multiple recent critiques argue that primate-like cognition in T. rex is not supported, that neuron counts are unreliable for fossil cognition and that dinosaurs were neither simply crocodile-like nor human-like but cognitively distinct animals.

References 147-158.

Gut Bacteria and Iron Regulation

> *"The bacteria have been a pivotal part in our intestine speaking to our body about whether or not our iron level is low or high in the liver or in the blood system, in our bone marrow or in our brain."*

The microbiome is not the primary regulator of iron homeostasis. Core control is attributed to host transporters, iron-sensing pathways and hormones such as IRE/IRP, HIF-2α, and hepcidin, with microbial effects acting through or alongside these systems.

The gut microbiome influences iron metabolism but is a **secondary modulator** rather than the primary regulator of systemic iron homeostasis. Gut microbes compete for dietary iron and produce metabolites that alter intestinal iron absorption and storage by suppressing HIF-2α signalling, reducing iron transporter activity, and increasing ferritin expression. Evidence from mouse and germ-free studies supports these mechanisms, showing that microbiota-derived metabolites limit intestinal iron uptake and tissue iron accumulation. This interaction is bidirectional and context-dependent: iron deficiency or overload reshapes the microbiome, while microbial composition influences iron absorption and storage differently under iron-rich and iron-poor conditions.

For the liver, the evidence is strong that gut microbes are associated with host iron stores and liver biology. However, evidence for the intestine and liver is strongest across mechanistic and animal-human studies. Higher serum ferritin was associated with lower microbial gene richness, specific taxonomic shifts, microbial iron-metabolism functions, and liver iron-related genes, and microbiota transfer from high-ferritin donors altered recipient mouse genes involved in iron metabolism and fat accumulation.

For the brain, the evidence is more mixed but increasingly supportive. Evidence for bone marrow and brain is earlier-stage and more inferential, except for the direct neuronal mechanism shown in worms. Human studies link gut microbiota, serum ferritin, microbial metabolites, and executive function, and iron overload in thalassemia is associated with dysbiosis and cognitive impairment.

References 159-171.

Lactobacillus reuteri and a Newly Named Metabolite

> *"If you do not have a specific bacteria called Lactobacillus reuteri, you will be missing a brand-new metabolite or chemical that was named after that bacteria just in the last three years, that's called reuteri."*

Limosilactobacillus reuteri is described as producing **reuterin**, a glycerol-derived antimicrobial metabolite or metabolite system. A **1988 paper** described reuterin as a newly discovered broad-spectrum antimicrobial substance produced by *Lactobacillus* (now *Limosilactobacillus*) *reuteri* during glycerol fermentation. A later review explicitly states that **reuterin was first discovered in the 1980s** and traces the finding back to that earlier study.

The statement that the absence of *L. reuteri* makes gut reuterin production unlikely is plausible, but it needs nuance. Several papers describe *L. reuteri* as the primary or most important producer in gut-related contexts and show that both *L. reuteri* and reuterin are reduced together in colorectal cancer samples. But **the evidence does not support exclusivity**. Reviews state that other genera, including Bacillus, Klebsiella, Citrobacter, Enterobacter, Clostridium, and some lactobacilli, can also produce reuterin. Production is also strain-specific, with **some *L. reuteri* strains failing to produce reuterin** despite belonging to the species.

References 172-181.

Concurrent Vitamin D and B12 Deficiency

> *"People are very frequently both low in vitamin D and vitamin B12 at the same time, and this is because they share the same root cause like a poor gut absorption, aging, or a restrictive diet, not because one vitamin is destroying the other."*

Direct studies generally show that low vitamin D and low B12 do happen in the same people more often than expected by chance. Co-occurrence appears especially common in people with chronic disease, restrictive diets, older age, and some psychiatric populations. For instance, type 2 diabetes shows high vitamin D deficiency and clinically relevant B12 deficiency in the same patient population. Severe mental illness is marked by prevalent vitamin deficiencies, with D and B12 both

linked to worse metabolic or psychiatric outcomes. Older adults and GI disorders raise B12 risk through impaired absorption, while the same groups often carry a high combined-deficiency burden.

Some studies show coexistence without proving identical drivers. In West Bank hospital data, vitamin D insufficiency was extremely common at 88%, whereas B12 insufficiency affected about 19%, so overlap is plausible but not universal. In Indian urban-rural data, both deficiencies were common, but vitamin D was more deficient in urban participants, whereas B12 was more deficient in rural participants, showing that the two vitamins can diverge when their main exposures differ.

The evidence is mostly cross-sectional, so it shows co-occurrence and correlation better than cause.

References 182-191.

2016 Hypothesis on Sleep, Microbiome, and Vitamin D

> *"I hypothesize that the parallel epidemics of abnormal sleep and abnormal intestinal microbiome are linked to one another through vitamin D deficiency."*

The claim that vitamin D deficiency causes microbiome-driven B-vitamin depletion that then worsens sleep is a mechanistic hypothesis with uncontrolled clinical observations, not a tested causal trial. The 2016 paper proposes a mechanism in humans where vitamin D deficiency produces a persistently altered intestinal environment, reduces bacterial B-vitamin production, and, secondarily, lowers pantothenic acid needed for cortisol and acetylcholine synthesis. **That mechanistic chain remains speculative** because it comes from a hypothesis paper and practice-based observations rather than controlled microbiome trials. The paper supports a plausible link between abnormal sleep and abnormal intestinal microbiota involving vitamin D but does not prove that vitamin D deficiency is the primary explanation for the parallel epidemics.

Reference 192.

Maternal Vitamin D and Newborn Vitamin D Levels

> *“If you do this before you get pregnant, you keep the mom's D in the 60s. The baby's D is in the 40s when they're born.”*

Vitamin D supplementation during pregnancy raises both maternal and cord-blood 25(OH)D, and maternal and neonatal levels are strongly correlated. The evidence is mostly about supplementation during pregnancy, not starting before conception. Many trials were small, heterogeneous, or high risk of bias, which limits confidence in precise target numbers. Several newer cohorts still found high neonatal deficiency rates despite supplementation, especially with lower doses or in higher-risk settings. Personal decisions about testing, dose, timing, and pregnancy planning should go through a licensed clinician; this evidence does not support a universal target strategy. There is no single pregnancy-specific consensus target for serum 25(OH)D, but most authorities and reviews treat <20 ng/mL (50 nmol/L) as deficient and ≥30 ng/mL (75 nmol/L) as a more ambitious sufficiency target used by many experts and trials; some authors argue >40 ng/mL could be relevant for broader non-skeletal or neonatal goals. For babies, neonatal status tracks maternal status closely, and one review highlighted that when maternal 25(OH)D was at least 50 nmol/L, infants in a Danish cohort did not have cord levels below 30 nmol/L.

References 193-204.

Sulfated Vitamin D vs. Supplemental Vitamin D

> *“There is a lot of literature about, by a gal named Stephanie Seneff, who says that the vitamin D that we make on our skin is sulfated, which is different than the vitamin D we're taking as supplemented."*

Seneff is not a leading experimental author on measuring sulfated vitamin D metabolites in humans. Her direct contributions are mainly conceptual reviews and hypothesis papers.

Sulfated vitamin D is a real, abundant metabolite pool, but its physiological role remains unsettled. A 2021 study (Jenkinson et al.) showed that in a cohort of 170 older men, sulfate conjugates accounted for roughly 18% to 53% of total vitamin D metabolites, with 25OHD3 sulfate around 40% to 48% and glucuronides much lower. In lactating women, serum 25OHD3-S exceeded

25OHD3 at baseline, and VitD3-S in serum was similar to VitD3. Interestingly, 5000 IU/day vitamin D3 raised serum vitamin D3 and 25OHD3 but did not significantly raise sulfated metabolites over 28 days in lactation. What we do not yet know is how much these metabolites contribute to human vitamin D function, infant nutrition, or disease risk beyond serving as a substantial circulating reservoir.

References 205-215.

Supplemental Vitamin D vs Being Outdoors

> *“Stasha: "Supplementing with vitamin D is not the same as being outdoors."*

Skin-made vitamin D3 and supplemented vitamin D3 converge on the same hormone pathway, but they are not fully equivalent in delivery, kinetics, or practical use. The core molecule is the same when the supplement is vitamin D3: sunlight converts 7-dehydrocholesterol in skin to vitamin D3, and orally ingested D3 feeds the same downstream endocrine pathway.

They are not identical in pharmacokinetics: skin-produced vitamin D3 appears to persist longer in circulation, while oral D3 peaks rapidly and returns toward baseline sooner. They are not equally practical or controllable: oral supplementation is easier to titrate and is described as the safer way to increase vitamin D status because UV exposure is hard to dose and carries skin-damage risk.

Outdoor time still matters independently of supplements. Outdoor time also offers mental and social benefits, among others (which go beyond the context of this claim and are, thus, not included here), that supplements do not inherently provide.

References 216-220.

Indoor Work and Cardiovascular Risk in Vietnam

> *“They'd shown this in Vietnam. When Vietnam started to really be successful, the guys who were inside the office but still had the same Vietnamese diet, they developed hypertension, heart disease, and early stroke and heart attack... because they weren't outside.”*

Among nearly 58,000 Vietnamese employees, metabolic syndrome affected 16.0% unadjusted and 21.8% after age-sex adjustment, with abdominal obesity and high triglycerides as major contributors. In rural Khánh Hòa, higher occupational physical activity was associated with lower hypertension prevalence, but **that association became non-significant after BMI adjustment**. In another study in Vietnamese adults, greater occupational activity was linked to lower hypertension prevalence, which indirectly supports harm from more sedentary work, but BMI attenuated the association.

Less time outdoors is a plausible contributor because greener and more natural environments are consistently associated with lower blood pressure, less hypertension, and lower cardiovascular risk, while the proposed pathways include more physical activity and lower stress. But “less time outdoors” is not uniformly protective or harmful by itself: one large UK Biobank study found more outdoor light time was associated with a slightly higher hypertension risk, so the relevant exposure is probably not just being outside but what kind of outdoor environment, activity, light, and pollution exposure someone has. Therefore, the evidence does not support a simple rule that “being outside lowers blood pressure", because sunlight, nighttime light, air pollution, and the quality of the outdoor setting can push risk in opposite directions.

Sunlight’s benefits are not explained by vitamin D alone in the cardiovascular literature. Reviews and cohort analyses consistently report that greater sun or insolation exposure is associated with lower blood pressure or cardiovascular risk, while oral vitamin D supplementation has generally not reproduced those benefits. The mechanisms are broader than vitamin D. The strongest evidence points to UVA-triggered nitric oxide release from skin, with additional support for circadian effects through light–melatonin signalling and for greener outdoor exposure through lower stress, more activity, and less environmental strain.

References 221-236.

The Redhead Gene and Vitamin D Synthesis

> *"The redhead gene usually comes with freckles, which means that there's only little tiny parts of the skin that have melanin, and the redhead gene came about in the far north Viking Scotland, and those places don't get much sunlight.*
>
> *That means it cleared out the blocker of the thing that collected that energy and allowed people who lived in very overcast environments to make D because the melanin didn't block the conversion to vitamin D and all these other things that it does. So redheads actually have a bit of a superpower when it comes to their relationship with the sunshine."*

Red hair is commonly caused by variants in the MC1R gene, which are especially common in northern European populations. However, red hair is not explained by MC1R alone, and penetrance is incomplete. These variants reduce the amount of protective dark eumelanin in the skin, allowing more UVB light to penetrate and likely facilitating vitamin D biosynthesis under low-light conditions. This may have been advantageous in regions with limited sunlight, although the evolutionary reasons remain a hypothesis. The trade-off is that red-haired people burn more easily and have a higher risk of skin cancer, so it is not simply a "sunshine superpower".

References 237-244.

Americans' Time Spent Indoors (USA Today Statistic)

> *"90% of Americans spend close to 22 hours inside every day. That was from USA Today. And roughly 20% of Americans never go outside, regularly spending 24 hours indoors."*

Americans spend, on average, about 90% of their time indoors (equivalent to roughly 22 hours per day). This is a population average, not evidence that 90% of Americans individually spend 22 hours indoors each day. This estimate originates from the National Human Activity Pattern Survey (NHAPS), a nationally representative survey of 9,386 U.S. residents, which found that participants spent approximately 87% of their time in buildings and 6% in enclosed vehicles. The U.S. Environmental Protection Agency (EPA) summarises these findings as Americans spending "about 90%" of their time indoors.

Reference 245.

UVA Tanning Beds and Vitamin D Production

> *“If you're gonna use a... tanning salon, if they're using UVA, you are not making vitamin D from that.”*

The World Health Organization (WHO) states:

> *"Sunbeds emit UVA and UVB radiation. In general, sunbeds predominantly emit UVA radiation. However, in recent years, sunbeds have been manufactured that produce higher levels of UVB to mimic the solar spectrum and speed the tanning process."*
>
> *"The UV spectrum emitted by tanning machines has evolved in recent years towards higher UVA irradiance", although the UVA/UVB mix varies between machines.”*

The WHO explains that commercial sunbeds are predominantly UVA, whereas medically supervised phototherapy uses specific UVB wavelengths. Because vitamin D synthesis requires UVB, predominantly UVA sunbeds are a poor source of vitamin D.

References 246-249.

UV Penetration Through Cloud Cover

> *“Up to 80% of UV radiation still penetrates light cloud cover... When it's thick, dark, and overcast... that can block up to 70 to 90% of UVB rays.”*

Most empirical studies here report roughly 30% to 60% lower vitamin D–effective UV under heavy cloud, with larger losses possible in some conditions but not as the typical value. Broader modelling and observational studies show clouds reduce UVB relevant to vitamin D status, but they do not support a single universal percentage because cloud effects vary with region, season, and other atmospheric factors.

References 250, 251.

Beriberi in Japanese POW Camps and the Bataan Death March

> *"There are two incidences from the 1940s where the first vitamin deficiency states were described, and both of them turn out to be purely carbohydrate diets. One of them was in the Japanese prisoner of war camps and the Bataan Death March in World War II, where they had rice, rice, rice."*

Beriberi was recognised long before World War II, with descriptions in East Asia centuries earlier and clear medical attention from Western physicians in Asia by the late 1880s. By the late 19th and early 20th centuries, research had already linked beriberi to polished white rice and to loss of an "anti-beriberi" factor in rice bran, rather than to infection alone. Japan itself had major beriberi epidemics before WWII, including high mortality in the late 1870s and large military outbreaks in 1904–1905, and army use of rice-barley mixtures reduced disease by restoring vitamin B1.

Japanese prisoner of war camps in WWII strongly confirmed, under extreme conditions, that predominantly polished-rice diets produced beriberi and other B-vitamin deficiency syndromes, but they were not the first setting to reveal that relationship. These camps were important because physicians documented prolonged human deficiency syndromes in large captive populations eating mainly highly milled rice, often with edema, neuropathy, pellagra, riboflavin deficiency, and severe calorie deprivation layered together. Bataan appears as part of a broader wartime malnutrition history in the Philippines that later fed directly into postwar beriberi control efforts such as the 1946 Bataan Rice Enrichment Project.

References 252-261.

Pellagra and Corn Gruel Diets in the 1930s-40s South

> *"Pellagra, which is actually a niacin deficiency in adults and children that were institutionalised in the South of United States in the '30s and '40s and fed corn gruel as their only food."*

Pellagra in the South was fundamentally a dietary niacin deficiency disease, and heavy reliance on corn was a major contributor when diets were otherwise poor. The evidence also shows corn itself was not sufficient by itself; poverty, low dietary diversity, low animal-protein intake, and food processing determined who became deficient.

References 262-266.

Paleolithic Human Lifespan

> *"There were people that lived to be 75 years old 20,000 years ago."*

Some Upper Paleolithic people survived into older adulthood, but it does not let anyone confidently identify a 75-year-old individual living exactly 20,000 years ago. The main reason is methodological: adult skeletal ageing often underestimates old age, especially past 50–60 years, so the absence of many very old skeletons is not strong proof that such people did not exist.

Hunter-gatherer mortality profiles indicate that post-reproductive longevity is a normal human pattern, not just a modern medical phenomenon. In some modern small-scale populations with low adult mortality, the modal age at adult death can be in the late 60s to 70s. In these populations, at least one-fourth of people who survive childhood likely live long enough to be grandparents for 15–20 years. Grandparenthood and extended post-reproductive survival are documented in many human populations, but rates vary depending on mortality conditions.

Around 20,000 years ago falls within the Last Glacial Maximum, when Upper Paleolithic groups persisted despite climatic stress and population bottlenecks, so humans were certainly present then, but that does not itself resolve exact ages at death.

References 267-275.

Nicotine as a Substitute for Acetylcholine in Narcolepsy

> *"There is only one drug that duplicates acetylcholine completely, and it's nicotine... she stopped smoking, and she uses nicotine patches."*

Nicotine acts on nicotinic acetylcholine receptors rather than replacing all acetylcholine signalling, and those receptors exist in many subtypes with different pharmacology and functions. Acetylcholine normally has rapid, transient synaptic actions because it is broken down within milliseconds by acetylcholinesterase, whereas nicotine is not hydrolysed that way and produces more prolonged receptor activation.

Because nicotine persists longer, chronic exposure commonly causes desensitisation and upregulation of nAChRs, which can make receptors less responsive to both nicotine and acetylcholine rather than restoring normal cholinergic function. Several reviews explicitly describe brain nAChRs as mainly modulatory, with only a minority contributing directly to fast excitatory transmission, so replacing acetylcholine globally is not what nicotine does.

Narcolepsy-specific evidence here is very limited. The only directly relevant narcolepsy paper is a brief report noting small-dose nicotine as a stimulant and describing signals of possible benefit for narcoleptic seizures, but it does not show that nicotine substitutes for acetylcholine across the brain or establishes an effective mechanism in narcolepsy.

For narcolepsy, the evidence provided does not show that nicotine completely replaces acetylcholine in the brain. It shows that nicotine can stimulate selected nAChRs and produce distinct, prolonged, and adaptive effects, while direct evidence for narcolepsy remains preliminary, of low quality and insufficient to support its use as an evidence-based clinical treatment.

References 276-286.

Smoking and Parkinson's Disease Outcomes

> *"The guys with Parkinson's disease who smoked throughout the last 30 years have been shown to do better because Parkinson's disease is an acetylcholine deficiency state first."*

The evidence is that long-term smoking is associated with a lower incidence of developing Parkinson's disease, while established Parkinson's disease remains primarily defined by nigrostriatal dopamine loss with important cholinergic abnormalities that contribute to some motor and non-motor symptoms. The association appears biological rather than simple bias, because it persists across tobacco products, is stronger in current than former smokers, declines after quitting, and is not well explained by selective survival or shared genetics.

Nicotine-based treatment in people with established Parkinson's has mixed clinical results and has not clearly slowed disease progression, despite supportive animal and mechanistic data. Proposed nicotine mechanisms include dopamine release, α-synuclein effects, and anti-inflammatory

signalling, but these mechanisms are largely preclinical and not proof that smoking improves diagnosed Parkinson's Disease.

As a summary, smoking is associated with a lower observed risk of Parkinson's disease, but the reason remains unclear and does not outweigh the substantial, well-established harms of smoking. Additionally, nicotine-based therapies have not demonstrated convincing clinical benefit for Parkinson's disease.

References 287-295.

Sleep Disorders and the Microbiome

> *"Are many of the sleep disorders caused by the microbiome?"*

The literature supports a **bidirectional link**, not a simple one-way cause. Microbiome effects look most plausible as one contributor among many, with stronger evidence for association than for broad causation. Sleep disorders have multiple causes, and several reviews explicitly frame the microbiome as one factor among neural, immune, metabolic, circadian, dietary, psychiatric, and environmental influences rather than the main cause.

Some specific taxa show causal signals for insomnia or related sleep phenotypes in Mendelian randomisation studies, but these are limited to selected microbes and outcomes rather than most sleep disorders overall.

The clearest consensus is that sleep disruption changes the microbiome and that altered microbiota commonly accompanies poor sleep, insomnia, sleep fragmentation, or short sleep duration. Human observational studies repeatedly find microbiome differences in insomnia or poor sleep, including shifts in diversity, beta diversity, inflammatory taxa, *Firmicutes*/*Bacteroidetes* balance, *Clostridia*, *Faecalibacterium*, *Bacteroides*, *Prevotella*, and *Akkermansia*, but these designs do not establish that microbes are the primary cause.

Mechanistically, the proposed pathways are consistent across reviews: microbial metabolites, immune-inflammatory signalling, vagal and enteric neural pathways, serotonin, GABA, SCFAs, tryptophan-related metabolism, and circadian interactions could influence sleep regulation.

Causal evidence exists, but only for selected microbes and phenotypes such as insomnia, snoring, sleep duration, excessive daytime sleepiness, obstructive sleep apnea, sleep-wake rhythm disorder, and isolated REM sleep behaviour disorder. However, some effects are indirect, mediated by pain, obesity, smoking, or adenosine-related pathways rather than direct microbial causation alone. Microbiome-targeted treatments appear promising, but reviews still say effectiveness is not established and longer, **better-controlled trials are needed**.

References 296-301.

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