Mitochondrial-Metabolic Theory of Cancer:

Implications for Sun Exposure

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Emerging Revolution in Cancer Research

In recent decades, the somatic-mutation theory of cancer has predominated, insisting that cancer arises as the result of nuclear genetic mutations (Boddy, 2022; Fujimura, 2013; Soto & Sonnenschein, 2014;). From this belief, a significant and misguided *conflation* of DNA damage, mutations/mutagenicity, carcinogenicity, and the risk of developing malignant cancer has prevailed (Adjiri, 2017; Finn, 2018; Okwan-Duodu et al., 2025). Recent, more robust theoretical work has suggested cancer may be a *metabolic* disease, arising from derangements in the number, structure, and function of mitochondria, resulting in altered energy metabolism and *downstream* effects on nuclear DNA via oxidative stress (Gaude & Frezza, 2014; Hsu et al., 2016; Seyfried et al., 2014; Seyfried 2015; Srinivasan et al., 2017). The dysfunction of mitochondria seen in cancerous cells reduces their ability to generate ATP through *oxidative phosphorylation*, the radically productive pathway relied on by multi-cellular animals for over 90% of their ATP, but which requires the complex machinery of mitochondria (Lodish, 2008; Luo et al., 2020). They instead must resort to increased substrate-level phosphorylation, that is, methods of generating ATP using simpler chemical reactions on fuel substrates. In potentially *all* cancers, energy production relies predominantly on 2 main substrates: glucose, through glycolysis and anaerobic fermentation to lactate, and glutamine (an abundant, non-essential amino acid), through aberrant usage of the mitochondria's TCA cycle (Chinopoulos & Seyfried, 2018; Schwartz et al., 2017; Seyfried et al., 2014).

This has led to the development of emerging metabolic therapies which target these fuel substrates, such as the Press-Pulse method, which employs a very low-carbohydrate (glucose-reducing) ketogenic diet consistently *pressing* blood glucose levels down, while

applying targeted, *pulsed* pharmacology for glutamine (because it is a necessary amino acid and cannot be suppressed chronically), alongside other drugs intended to disrupt fermentative substrate-level phosphorylation (Seyfried et al., 2017).[1] This in theory leaves only the cells exhibiting this deranged metabolism starved for energy, while healthy cells remain capable or even glad to be using ketone bodies, as a result of reduced blood glucose and insulin, within healthier mitochondria, whose health is further enhanced by ketone bodies (Kolb et al., 2021).

In the face of the theoretical and empirical strength of the mitochondrial metabolic theory of cancer, we are forced to reevaluate the corpus of mutogenic and carcinogenic stimuli, and consider that when their association to actually developing cancer is real, this may be mediated more by cellular and DNA *damage*, inflammation, oxidative stress etc., than actual DNA mutation. DNA damage, like these other influences, has a series of intelligent systems in place within the body, specifically designed to govern it (Chatterjee & Walker, 2017). In fact, DNA damage may have been unjustly emphasized as a purely deleterious phenomenon, and might warrant alternative framing, in some contexts, as a normal hormetic stressor, for which the body is not just prepared, but may even conditionally benefit from (Shibamoto & Nakamura, 2018). Indeed, exposure to the DNA-damaging challenge of solar irradiation might be *necessary* to cultivate optimal, *cancer-protective* levels of long-term DNA repair proficiency (Mägdefrau et al., 2019; Latimer et al., 2020).

There are different biochemical types of damage, some of which are easier to repair than others, and some are more common than others. Damage which manages to make a functional but altered encoding, and not be detected and reverted, is termed a “mutation.” Ultraviolet (UV)

[1] Incidentally, intestinal helminths are reliant on anaerobic energy production while residing in the gut. As a result, some anti-parasite drugs target the exact fermentation pathways in question, and are being repurposed for cancer.

induced damage primarily results in bulky cyclobutane *pyrimidine dimers* (Barnes et al., 2018), as well as 6-4 photoproducts, which are repaired by a several protein, broad-spectrum, multi-armed mechanism called Nucleotide Excision Repair (NER) (Budden & Bowden, 2013; Kusakabe et al., 2019). NER is employed by all insects, arachnids, reptiles, birds and mammals—all eukaryotes, and predates the 350 million years of life on land (Schärer, 2013; Selden & Kliman, 2016). Sun exposure is a metabolic stressor, to be sure, but one for which it is implausible that normally functioning repair mechanisms would not be well adapted. It is accumulated *unrepaired damage* that poses functional risk. This should lead us to be concerned not with sun exposure, but with dysfunction in reparative capacity. NER is a "highly efficient and accurate pathway" (Kuper & Kisker, 2023) but is also involved with handling the DNA damage caused by excessive oxidative stress (Lee & Kang, 2019), and is multiply dependent on ATP availability. In fact, in non-cancerous cells, genotoxic stress causes upregulation of fatty acid oxidation and oxidative phosphorylation in order to increase ATP generation in direct response to its depletion by DNA repair (Turgeon et al., 2018). To optimally repair UV damage, the cell should have ample ATP from normal, healthy mitochondrial oxidative phosphorylation, and should not have abnormally large amounts of oxidative stress, damaging DNA as well as NER proteins themselves. Indeed, it is plausible that developing cancer causes the sun to induce DNA mutations.

Oxidative stress seems to be a larger component of the challenge of sun exposure than simply DNA damage. Seed oils high in poly-unsaturated fatty acids are prone to lipid peroxidation, which cascades oxidative stress across the cell. Removing these from the diet will allow the DNA repair mechanisms of the skin cells to not be overwhelmed with excess oxidative stress. Healthy levels of the mammalian anti-oxidant glutathione, as a result of sufficient intake

of cysteine (meats) and glycine (skin and bone), have been shown to have preposterously positive effects on multiple metabolic markers (Kumar et al., 2023). Glutathione provides multi-faceted support for the skin's tolerance of sun exposure, not least by protecting the mitochondria from oxidative stress, ensuring ATP output for DNA repair remains ample. In fact, multiple studies have shown that higher levels of glutathione, apparently by direct virtue of its anti-oxidant effect, cause a set dose of sun exposure to lead to less sunburn (Hanada et al., 1990; Hanada et al, 1997). Walshe et al. (2007) found that problems with glutathione seem to precede the formation of UV-associated cancer.

We should not forget that for every mechanism we can describe now, the strange, intelligent engineering that evolution has engaged in constantly surpasses our expectations. Instead of relying solely on currently available biochemical details, we ought to bear in mind that basic evolutionary theory is constantly capable of foreseeing ideal interventions before they are explicable at a biochemical level.

Sun Exposure Under a New Light

We need to reinterpret the findings of the dangers of sun exposure away from a somatic-mutation understanding of cancer, which demonizes all DNA damage and therefore *any* sun exposure, to a mitochondrial-metabolic understanding of cancer, which suggests a lengthy process of progressive, *systemic* derangement of mitochondrial health, influenced by a multitude of controllable lifestyle factors. Indeed, evolutionary theory is brilliantly clear here: solar irradiation is the last stimulus on this list we should expect to need to modify, that is to say, we should expect the normal, healthful, systemic state to be one where detoxification, repair, and mitochondrial function are so secured that the burden of solar irradiation is either totally benign, or even beneficial. For sun exposure to be problematic, you probably need to be very unhealthy,

first.

The conditions for vitamin D synthesis are exactly synonymous with the conditions for DNA damage (Bouillon, 2017; Webb & Engelsen, 2006). Even research which is enthusiastic about the benefits of vitamin D remains coldly foreboding about sun exposure, limiting their recommendations from what the vitamin D observations *actually* imply, due to fears arising from the mistaken somatic-mutation theory of cancer. Hoel et al. (2016) state unequivocally: "The message of sun avoidance must be changed to acceptance of non-burning sun exposure sufficient to achieve serum 25(OH)-D concentration of 30 ng/mL or higher in the sunny season and the general benefits of UV exposure beyond those of vitamin D." But research on vitamin D has consistently shown that serum vitamin D should probably be about 50 ng/mL, where physiologic function is optimized, and levels mimic those found in ancestrally living populations (Cannell & Hollis, 2008; Heaney, 2015).

Findings of the danger of sun exposure are usually conflated and confounded with the extreme prevalence of metabolical-illness (Noubiap et al., 2022; Saklayen, 2018). Infrequent and extreme jumps in sun exposure are not only paired with occasional sunburns, but titrated into an altogether dysfunctional mitochondrial-metabolic context, a stage arguably set for cancer even prior to the sun exposure (Rezaiian et al., 2022; San-Millán, 2023), making the solar irradiation a kind of health-shibboleth which the modern person fails, revealing not the danger of the sun, but the metabolic incapacitation of the population. Skin health rests upon mitochondrial health (Sreedhar et al., 2020). Sunburns are damaging, and damage affects the mitochondrial health of the cell (Rong et al., 2021).

Sunburns should be desperately prevented, but not by sun avoidance, but rather extreme efforts to ensure that skin tanness is maintained at sufficient levels to match the seasonal strength

of local sun (de Gruijl, 2017; ElObeid et al., 2017; Hauser, 2006; Shah, 2015). Tanness results from an adaptive increase of the production of UV-blocking melanin, which reduces DNA damage (Barker et al., 1995; Fajuyigbe et al., 2018), and provides anti-oxidant, cancer-protective effects (Brenner & Hearing, 2008; Solano, 2020; Upadhyay, 2022). Consequently, the danger of sun exposure is enhanced by its avoidance, and reduced by consistent exposure (Lindqvist et al, 2016; Swope, 2018; Van der Rhee et al., 2016a; Van der Rhee et al., 2016b). If we suddenly jump sun exposure from a UV 0 to a UV 6, it's no wonder it isn't tolerated well. Maintaining synchronized tanning with the seasons, through far more sun exposure, is the top priority.

The Health Benefits of Vitamin D Sufficiency

Vitamin D is a hormone previously emphasized for providing systemic effects on calcium homeostasis, whereas more recent research has come to understand that it plays vital roles across a wide variety of tissues, many of which contain their own enzymatic machinery to activate the storage form and produce a paracrine signal (Holick, 2003). It is involved in the regulation of cell differentiation, proliferation, and apoptosis, innate and adaptive immunity, adiposity, energy expenditure, and inflammation (Bikle, 2009; Grant 2025).

When research is done properly, the benefits of vitamin D show up much more clearly, showing *strong* beneficial effects on cardiovascular disease, stroke, type 2 diabetes mellitus, hypertension, irritable bowel syndrome, autism, Alzheimer's, multiple sclerosis, bone health, kidney health, oral health, autoimmunity, pregnancy and birth outcomes, and cancer prevention and survival (Alfredsson et al., 2020; Chong et al., 2022; Grant et al., 2025). Grant et al. state "... [W]omen with 25(OH)-D concentrations ≥ 60 ng/mL had an 80% lower risk of breast cancer than those with concentrations < 20 ng/mL." Vitamin D findings demonstrate a reduced risk of incidence and mortality for 23 types of cancer (Muñoz & Grant, 2022). Indeed, vitamin D

reduces oxidative stress and inflammation, augments mitochondrial health and cellular antioxidant capacity, and is “crucial for mitochondrial oxidative phosphorylation capacity” (Latham et al., 2021; Reddy et al., 2022).

Generally these effects have been artificially produced by vitamin D supplementation, but normally over 90% of vitamin D is produced from the skin, not diet (Glerup et al., 2000). The amount of sun exposure necessary to reach these levels is reflective of the outdoor lifestyles of ancestral populations. Sun exposure required to reach actual optimal levels around 50 ng/mL far exceeds usually recommended levels, which are generally much less than an hour, and is on the order of *several hours per day*, particularly once skin tanning adapts to local UV (Binkley et al., 2007; Webb & Engelsen, 2006). Research which advocates dosages around 10–30 minutes is based on vitamin D goals about 10% as large as they should be (Kralova et al., 2025; Papadimitriou, 2017; Webb & Engelsen, 2006).

The Health Benefits of Sun Exposure

Health benefits of sun exposure *independent of vitamin D* are becoming evident.

UV radiation is used to convert nitrogen oxides in the skin to nitric oxide (Weller, 2016), which is a “key regulator of cardiovascular function, metabolism, neurotransmission, immunity and more” (Lundberg & Weitzberg, 2022), with complex effects requiring further research. Nisoli et al. (2004) explain long-term nitric oxide exposure “stimulates mitochondrial biogenesis, both *in vitro* and *in vivo*, and that this stimulation is associated with increased mitochondrial function, resulting in enhanced formation of ATP.”

UV radiation has immunological effects beyond the skin, penetrating to the circulation and modulating leukocyte activity, providing anti-inflammatory, antipruritic, antifibrotic

immunomodulatory effects (Hart & Norval, 2021). UV irradiation of blood provides an anti-bacterial effect even upon antibiotic resistant strains (Hamblin, 2017).

Other mechanisms of UV benefit apart from vitamin D include circadian effects, photoadaptation, serotonin, endorphins (Van der Rhee et al., 2016b). Benefits of sun exposure are detectable *despite* the prevalent metabolic illness in these populations which would make them less prepared for that very sun exposure.

Sun avoidance's contribution to all-cause mortality risk was found to be of similar magnitude to smoking, while active sunlight exposure improved mortality rate (Lindqvist et al., 2016). Alfredsson et al. (2020) state "insufficient sun exposure may be responsible for 340,000 deaths in the United States and 480,000 deaths in Europe per year."

Sun exposure has multiple mechanisms for the improvement in mitochondrial-metabolic health. Infrared light, which comes paired with UV in natural sunlight but is rare in modern artificial lighting, is known to increase mitochondrial ATP production across species (Powner & Jeffery, 2022; Jeffery et al., 2025). Gapeyev et al. (2017) demonstrates that studies investigating the harmful effects of UV are *too decontextualized to apply to human exposure to sunlight*, by showing that the DNA-damaging effects of UV are protected against when the UV is paired with red light ranges, which would be present in the full-spectrum irradiation of natural sunlight. This effect may well be mediated by the enhanced ATP availability for DNA repair, further corroborating the notion that the mitochondrial dysfunction of cancer causes the sun to induce mutations, not the other way around. Infrared light exposure produces improvements in insulin resistance across cell types, and ameliorates mitochondrial dysfunction, reduces inflammation, and modulates oxidative stress (Magalhaes & Ferraresi, 2022).

The mitochondria-metabolic theory of cancer not only shows promise for effective

interventions for all types of cancer, it also, less frequently discussed, directly implies that cancer might be possible not just to effectively treat, but to *definitively* prevent as well, via lifestyle cultivation of the health of the mitochondria and of the insulin-glucose system. Exposure to full-spectrum sunlight and acquiring optimal levels of vitamin D appear to be powerful tools for cultivating the health of mitochondria and insulin signaling. Apart from this, increased circulation of ketone bodies, the hallmark intent of a well-formulated ketogenic diet, have wide ranging effects in the body, promoting mitochondrial health (Kolb et al., 2021), powerfully reducing inflammation (Youm et al., 2015), reducing genomic damage, improving ROS handling in the body, and improving genomic repair (Kolb et al., 2021). Altogether, ketone bodies should be seen as powerful anti-cancer fuel and signaling substrates, regardless of the theoretical model used.

Implications for Practice

All patients should be recommended to quadruple sun exposure above current recommendations, alongside the below recommendations to prevent sunburn. Patients with any reason, such as family history, to fear developing cancer, skin or otherwise, should be recommended a ketogenic diet immediately. An abundance of caution suggests that these latter patients should be expected to maintain a very low-carbohydrate diet *before* engaging in this sun exposure regimen, since a genetic predisposition for cancer does indicate a genetic profile *more* reliant on good mitochondrial health than others.

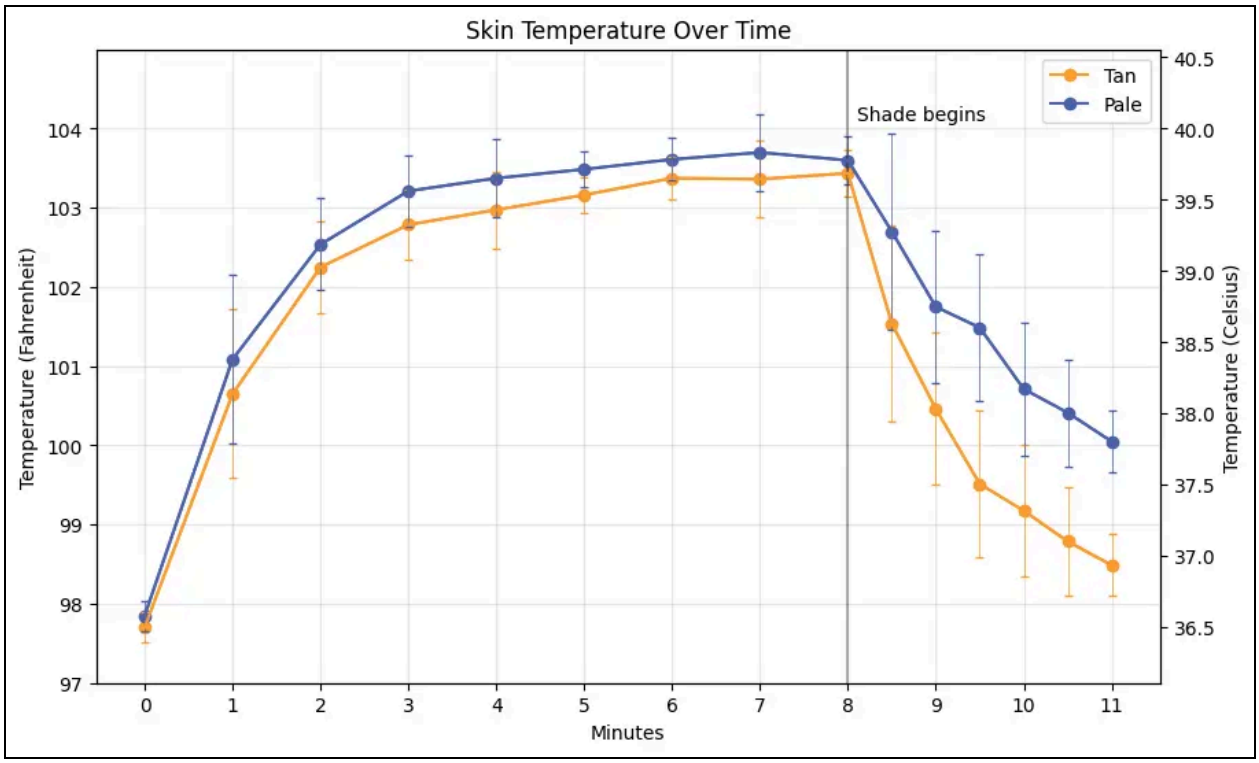
Of primary importance is that the tanness of skin be maintained in concert with seasonal variations in UV strength, via consistent *exposure*, not avoidance. Alongside this, supporting the health of mitochondria and reducing oxidative stress are crucial to restoring the safety of sun exposure, as well as preventing the metabolic conditions for cancer. Avoidance of seed oils is

likely to radically reduce the (lipid peroxidation induced) oxidative damage of DNA (Bartsch & Nair, 2006; Jenkinson et al., 1999; Łuczaj & Skrzydlewska, 2003), reducing competition for ATP-throttled NER repair capacity. The incorporation of glycine-rich foods, like skin or bone broth, exceeds the throughput of endogenous glycine synthesis, a rate-limiting step for the production of antioxidant glutathione, sufficient quantities of which will reduce oxidation stress and inflammation (McCarty et al., 2018; Ruiz-Ramírez et al., 2014).

Advocacy of sun exposure in clinical practice has been thwarted by uncertainty and fear. 43% of general practitioners reported that they were ‘not at all confident’ about their vitamin D knowledge. As described above, research claiming danger of sun exposure is based on multiple faulty extrapolations, such as the unlikely somatic-mutation theory of cancer, as well as decontextualized research employing isolated UV spectra. Meanwhile, vitamin D research has frequently used doses too small, pursued vitamin D levels too low, used poor design, poor conduct, and poor analysis (Grant et al., 2025; Heaney, 2015). Practitioners are faced with a morass of biochemical claims, but evolutionary theory provides practitioners a strong signal through the noise.

My own research provides anecdotal and experimental data supporting a method of thermal biofeedback for preventing sunburn during exposure to UV.

Figure 1

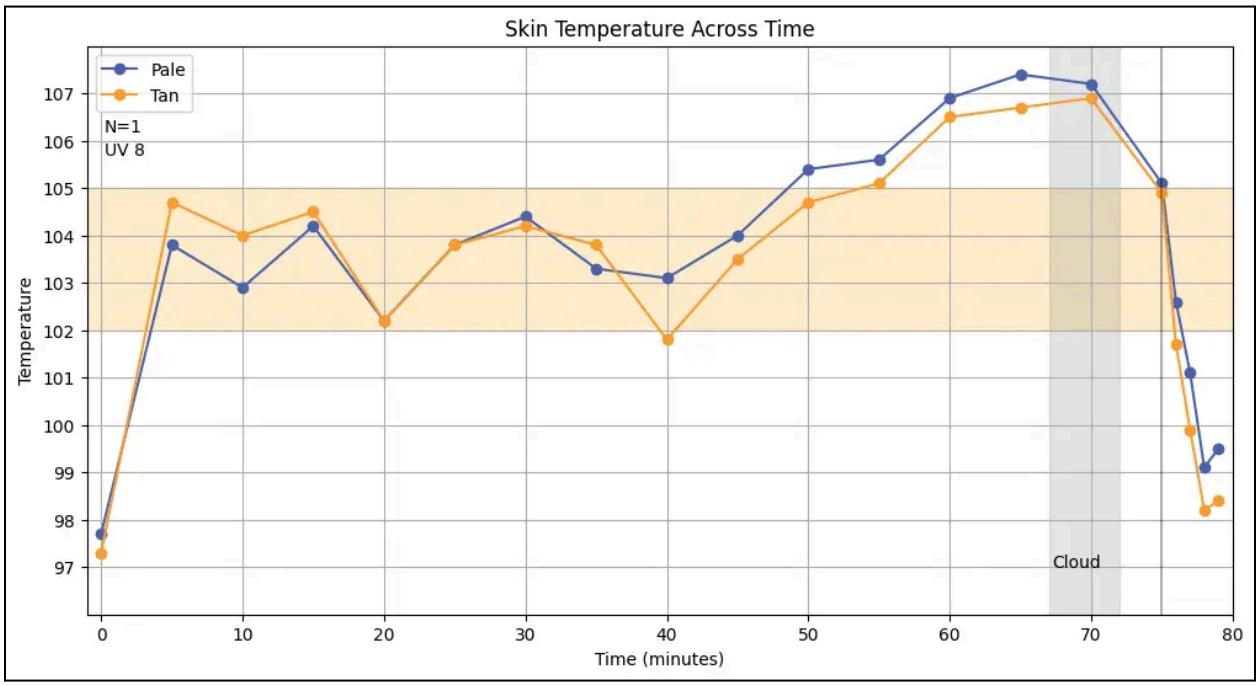


Note. Figure depicts averages at each time point across 12 trials, 1 outlier (pale wrist) excluded, various areas of skin, matched in pale and tan pairs, collected across multiple consecutive days of UV 9-12, zero-wind conditions. Data collection used an infrared measuring device, which importantly required shade to be applied momentarily to acquire a valid sample. This and other procedural challenges likely reduced the strength of findings, despite their apparent ease at detecting with one's hand following minor training, especially on the abdomen.

From this research I have found the following means for preventing sunburn to be efficacious: (1) Skin temperature will rise from 98° (all measurements in Fahrenheit) all the way to 103-104° (very warm to touch) following sun exposure longer than 2 minutes; (2) Skin that is insufficiently tan for the UV index will reach this level faster, and ride higher; (3) Skin will stay at this temperature, and will not burn, until the exposure length / UV exceeds the preparedness (tanness) of the skin, which will cause this steady level to be breached, and skin will rise to

106-107° (slightly painful to touch), (4) never allow sun exposed skin to reach 106° for longer than a minute, instead finding full body shade, and allowing skin to cool back to 98°, which takes just a few minutes when skin is far from burning, and occurs noticeably slower when skin is nearing or past burn. If skin does not cool below 100° in 3 minutes of shade, cease sun exposure for the day.

Figure 2



Note. Temperature in Fahrenheit. Figure depicts a single burn trial, comparing immediately adjacent skin areas, closely sharing circulation, likely causing the similarity in temperature. Pale skin exhibited in a full erythemal reaction and peeling. Tan region reddened slightly for a day, with no other symptoms.

Interestingly, there is evidence that other animals may use this thermal proxy to calibrate sun exposure (Yin et al, 2018). Use shade breaks interspersed among periods of sun exposure, when skin is not sufficiently tanned. Fairer-skinned individuals have a genetic approach which *defaults*

to a lower bottom-end of tanness than darker-skinned individuals, when solar stimulus is absent, but both are capable of tanning to withstand sun of UV 12, while the former must take more caution to work through accruing the necessary tanness. UV is linear, meaning that a fair skinned person, who has defaulted to being as pale as possible under conditions of UV 0, experiences the same challenge with a UV 6 (Northern US Summer), as this person would after adapting to a UV 6 and entering a UV 12 (Southern US Summer), with some caveats that increase the difficulty slightly.

My own cursory calculations, based on extrapolating from research findings, suggest the actual requirements, at 50% skin exposure to sun (higher than most research) , are ~120 minutes/day at UV 3, 60 minutes/day at a UV of 6 to maintain the optimal serum 25(OH)-D levels, at 50 ng/mL. These are for sun exposure recurring every single day of the week, although several complex dynamics across UV, skin type, and vitamin D and nutrient status are not factored in. These rough calculations may be overestimates for *maintaining* that level of 25(OH)-D (although Blinkley et al. (2007) suggest otherwise), but may apply better for increasing from the ubiquitous state of deficiency seen in sun avoidant cultures like the US. Sun exposure is safe and necessary, when sunburn is prevented effectively, by keeping up year round, or starting as early in spring as possible.

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