TIME #8: PSORIASIS AND THE PHOTOELECTRIC EFFECT

In the last few blog series and member webinars, I gave you examples of how nnEMF, specifically excessive blue light, can cause different diseases. Today we are going to talk about a disease that is caused by a breakdown in optical signaling on our surfaces, that leads to alterations in our biochemistry at deeper levels to lead to other diseases. This occurs just by changing what happens in the mitochondria without involving the nuclear genome. The power to create this situation is linked to the amount and power of the nnEMF to shift the percentage of heteroplasmy in cells to cause the disease.

The rapidity and power cause massive shifts in heteroplasmy of our mitochondria. I'd strongly suggest that you listen to this video carefully. Heteroplasmy can be described as mitochondrial DNA damage that alter the respiratory proteins in the inner mitochondrial membrane. This radicals changes their ability, and the tissues ability, to tunnel electrons, reduce oxygen, and to make the proper free radical signals in mitochondria. As heteroplasmy percentage increases, **NAD^+ drops, pseudohypoxia develops, ROS/RNS profiles change, and the amount of ELF-UV increases in a cell. These are the fingerprints of heteroplasmy.**

Our maternally inherited mtDNA codes for essential energy genes on our inner mitochondrial membrane. You know these as the cytochrome proteins or the respiratory complexes. They are present in thousands of copies per cell, and they have an unusually very high mutation rate built in. It turns out this mutation rate links to how well energy can flow in a cell. As

heteroplasmy rates rise, this forms and inflammatory stimulus in the cell to stimulate regeneration programs in a cell to spring to action. Circadian signaling however, is what links the stimulus of inflammation to the cycles of regeneration. If they are altered, it leaves a cell and the tissues those cells are in, with poorly functioning mitochondria and no ability to engage the regeneration programs built into our cells. These mutations change the energetic profile in a tissue by changing the percentage of mutations in the respiratory mitochondria occur before any changes that happen in the nuclear genome. Normally this is used as the stimulus for change. Mitochondrial senescence = % heteroplasmy. Diseases that have *massive acute shifts* in mitochondrial heteroplasmy however, cannot engage regeneration programs; an example of this is Kawasaki's disease (KD). I covered this disease in a recent member webinar. In KD, the incident non native electromagnetic light source is very powerful because its frequency is higher powered than the visible UV spectrum, and this disease can kill a healthy child in 24 hours over a large swath of a hemisphere. The same is true of many prion diseases, except its timescales are in weeks and months. In recent months, I showed members how ***electromagnetic hypersensitivity syndrome (EHS)*** can come on more slowly than KD or a prion disease, *but just slow enough, that most clinicians do not and would not be able to attribute these patients symptoms to the the true etiology of the syndrome driving this increased heteroplasmic % in the respiratory proteins*. This same process is behind other misunderstood diseases like fibromyalgia, lyme disease, and mycotoxin illnesses like Sick Building Syndrome.

Today's blog is about another disease that comes on over even slower times scales that EHS, because of alterations of the photoelectric interactions at the surface of our skin, eye, and gut. This disease is really a gateway disease to many

other diseases linked to heteroplasmic amplification of badly performing mitochondrial DNA. Psoriasis is the disease topic for today's blog and it is a gateway illness to epi-oncogenesis because of its tie to an altered light spectrum and its lack of interaction of light at the surface of the skin. Light that hits our surfaces has to be be solar derived, and not artificially driven by blue color temperature light. Our skin is specifically evolved to slow specific frequencies in a very sensitive and specific fashion by the atomic lattice on our surfaces. This process is quantized by both photochemical and photoelectric interactions. Light has to be slowed down by atoms in specific layers of our skin to be used properly to control cellular growth in that level of the epidermis.

Today's blog take home: No one can stop a ticking atomic clock……..but the best of us figure out how to slow its mechanism down. Your skin layers are designed to do just that; slow light to create more time to live the life your choices allow. When the skin is altered with "an emergent autoimmune condition" you cannot do this well.

Psoriasis is an autoimmune disease of the basal skin layers that is undergoing hyper-growth. Another way to put this, is it appears the basal skin is growing at more rapid rates than the surface skin. The cause of this hypergrowth is due to higher powered light penetrating to the deeper layers of the skin. The deeper layers of the skin become thicker. People with psoriasis get patches of thickness that appear red and scaly. Our surface skin is designed to grow slower in relation to the basal levels. How does AM sunlight limit surface growth? The human daytime photoelectric chemicals that are created are Vitamin D3, histamine, cortisol, nitric oxide, and sulfhydryl groups. The sulfhydryl groups are added to cholesterol and other proteins in the skin I mentioned in Tensegrity 7 blog post.

What does full spectrum sunlight destroy naturally? UVA and B light naturally lowers adrenalin (the stress hormone of the sympathetic nervous system) while its photons re-zip collagen that cortisol release causes at 4AM that wakes us up by allowing water flows to occur between glial cells and neurons via the aquaporin 4 gates in the brain. AM sunrise light has no UVA or UVB present initially. It has blue, green, and red present. It has more blue than any other part of the spectrum and this is stimulatory to waking us up. Naturally, UVA light shows up later in the AM, depending upon your location in latitude, and this frequency of UVA light acts to begin to re-zip the collagen in our skin and eyes. That initial blue light stimulus from the sun's rise is used to unwind our collagen to increase water flows to stretch the interspaces in neurons to wake our body and mind up at dawn from sleep. The aquaporin 4 gates are what are destroyed in Multiple sclerosis, which is another autoimmune condition.

When we are missing UV and/or IR light for any reason, these photoelectric and photochemical are not made. When full spectrum sunlight is absent in someone who is chronically stressed for any reason, sleep cannot be induced because the ocular melatonin cycle requires that these two frequencies of light be present to stimulate the regeneration processes in the eye during daytime. If you think about your childhood, when you spent the day at the park of the beach, you might remember how easy it was to fall asleep and get a sunburn. The reason is simple, sunlight induces sleep because the regeneration pathways that use melanopsin need daylight to regenerate. When you did fall asleep, the redness of your skin did not come from the thermal burn, but it was from the increased blood flow due to the release of nitric oxide that acted to bring the arterioles of the dermis layers to the surface. This is a photochemical change induced by sunlight to allow the skin to absorb the UVA and UVB light at the surface. UVA and UVB light does not penetrate deep. TO absorb the UV light we need the circulatory system to come

from the dermis and engorged the arterioles with RBC's. The RBC's are filled with hemoglobin and porphyrins that absorb both UV and IR frequencies. The sunburn is really an absorption of too much thermal IR energy. Deep sunburns can result from several factors: excessive sun, or thin skin, thick skin, or a poor adaptation to seasonal light due to chronic use of UV blocking makeup, clothing, or sunblock use in strong light cycles.

In fact, falling asleep in strong daytime sun, is a key clinical sign to the astute clinician your normal daily light environment really is. UV and IR sunlight normally lowers endocrine steroids (CT 7 blog) and **lowers the nucleic acids in the surface keratinocytes without any liberation of heat on the skin's surface.** This last photochemical change shows you how UV and IR sun working together helps dissipate surface photonic power by exposing more nucleic acids to the incident rays of the sun. This photoelectric action helps preventing aberrant optical scattering at deeper levels in the skin allowing for proper cell signaling in deeper skin levels. This is broken and lost in a disease like psoriasis because the surface non linear optics are broken by a serious deficiency in surface sunlight exposure. Many people fail to realize that the epidermis has no nuclear DNA, because as skin layers rise to the surface, DNA is exposed to a cells interior naturally as the cell sloughs away and dies a natural death. This exposure of the nucleic acids to incident sunlight allows the degenerating keratinocytes DNA to absorb all the extra UV light frequencies that the RBC's cannot tolerate. This photochemical and photoelectric dispersion of energy acts as a free chemical within the epidermis. The irony is, it acts as a natural sun protectant or sun blocker without altering the vitamin D3 cycle at deeper levels. The more photo-degredation our skin senses, the more skin cells show up in the epidermis and the skin thickens in summer months. This gives us an acute skin callus to sunlight. This also acts to lower our hemoglobin and hematocrit to lower UV assimilation.

If sunlight is a chronic stimulus, melanin production will be stimulated in the skin to protect the skin. Melanin also absorbs all UV frequencies and acts to serve as a storage protein for the power of UV light. It offloads these frequencies of light at night and not during the day. This provides photoelectric protection of other atoms in our skin lattice to protect deeper molecules in the skin from excessive growth. In this way your skin acts like a crystal. In psoriasis this protection system is broken.

As a skin cell rises to our surfaces epidermis, our DNA/RNA acts like a natural sunscreen for the absorption of surface UV light. What happens to if that DNA is not degenerated enough to perform this vital surface function? **More powerful blue and violet light energies get through to deeper levels to drive growth of the basal levels of the skin.** See the skin picture below for the details.

Dermatologist have struggled to treat this autoimmune condition for ages. One thing that had worked in the treatment of psoriasis in the older days of medicine was exposure of the surface skin to UV light. Phototherapy in the form of sunlight has long been used for psoriasis. Wavelengths of 311–313 nanometers have been shown to be most effective strategies. Here is more irony; how can UV light be helpful in a basal skin condition if dermatologist keep telling people that UV light is bad for humans and our for skin? You want even more irony in this tale? Why is that in the dermatology literature the amount of reportable skin cancer from the photo therapeutic treatments used in psoriasis are ridiculously low if UV light is so toxic to humans? How can these two contrary things be true? Does this make sense based upon what the dermatologists are reporting to you about sunlight?

PSORIASIS PHYSICS SIDE BAR: Tyrosine is the base amino acid for all pigmented proteins in the skin like melanin. All aromatic amino acids absorb UV and are excited by it. Photo-excited tyrosine can fluoresce, decay non-radiatively, or

undergo intersystem crossing to the triplet state, from which most of the photochemistry proceeds in the skin. The triplet state tyrosine is rapidly quenched by molecular oxygen that is found in the blood plasma but made by local surface dwelling mitochondria. This photoelectric change occurs adjacent to nearby residues like tryptophan or disulfide bridges in sulfated proteins (Bent & Hayon, 1975b). Here you begin to see where the mechanisms built into the Energy and Epigenetics 12 blog and the Tensegrity 7 blog show you how sunlight works with sulfated lipids and proteins.

An important photochemical mechanism that occurs using the photoelectric effect in proteins involves reduction of disulfide bridges (SS) upon UV excitation of Tryptophan and Tyrosine side chains (Kerwin & Rammele, 2007, Neves-Petersen et al., 2002 & 2009a). UV-excitation of tryptophan or tyrosine can result in their photoionization and to the generation of solvated electrons. The generated solvated electrons can subsequently undergo fast geminate recombination with their parent molecule, or they can be captured *by electrophillic species like molecular oxygen.* **Molecular oxygen decreases the chance of pseudohypoxia and this make formation of an exclusion zone (EZ) in water in skin cells**. This leads to coherent domains of H_3O+, (at a lower pH than normal) that allows photo molecular interactions to occur between cysteine and cystines (EE 12 blog on cysteine/cystine). In the eye, the surface cornea gets its oxygen directly from the air so if you wear a contact and have psoriasis you really are creating a massive mismatch for the mitochondria to deal with. This will spur massive heteroplastic growth in the deeper layers of the retina altering the central retinal pathways governing melanopsin and ocular melatonin regeneration cycles.

In the skin, in the case where the electron is captured by the cysteine, the result can also be the breakage of the disulfide bridge in proteins like glutathione (Hoffman & Hayon, 1972). I mentioned this in the last few blogs. ***Here you begin to see***

how full spectrum UVA light and the aromatic AA begin to work to create energies that can modify sulfhydryl groups in proteins and lipids in the skin to control non linear optical gating of signals from sunlight's frequencies. Activated electrons from the incident UV light is where the process begins. This is a photoelectric process. The UV light also creates thiol free radicals in this process.

LAMI is the first way to measure this process in mitochondria. Our beta testers using the Quantlet will also be able to create bio-hacks to test these photochemical changes as they develop

The resultant free thiol radicals/groups can then subsequently react with other free thiol groups in lipids and proteins in skin to create a new disulfide bridge that can act to quench the photoelectric excitation. Reduction of disulfide bridges with UV excitation of aromatic residues has been shown in the literature for proteins such as cutinase and lysozyme (Neves-Petersen et al., 2009a, 2006 & 2002), bovine serum albumin (Skovsen et al., 2009a; Parracino et al., 2011) prostate specific antigen (Parracino et al., 2010), and antibody Fab fragments (Duroux et al., 2007). These ***photoelectric phenomenon*** have actually led to a new technology for protein immobilization called light assisted molecular immobilization (LAMI).

LAMI is being used now to design drugs using UV light to PREVENT cancer. This stands in counter distinction of the modern view point that UV is the major cause of cancer. The UV light creates thiol groups that can bind thiol reactive chemicals on surfaces leading to oriented covalent protein immobilization. This is precisely what sunlight does naturally in our skin as I mentioned in Tensegrity 7.
In LAMI, pulsed UV illumination and the interaction of aromatic amino acids can halt activation of cancer cell

membrane receptors. HERCEPTIN is such a membrane receptor present in the breast and epidermal derived growth factor (EDGF). **It is the main cell membrane receptor involved in the skin associated with cancers.** The UV illumination affects all downstream photochemical reactions that could lead to cancer, by shutting down the cells' biological functions, thereby stopping oncogenic transformations. LAMI uses the same process to halt cancers that is present in normal cells that is designed to keep us from cancer generation. This new treatment is based upon the same findings that all stressed cells release ELF-UV light in response. This light appears to be used in "stressed cells" to activate the cell's own cell death program called apoptosis. Apoptosis also lowers heteroplasmy in a cell and lowers the percentage found in a tissue. This has already been documented on two human epidermal cancer cell lines (Olsen B.B. et al., 2007). The photonic dosage necessary for therapeutical results in LAMI has additionally been determined. It turns out, this dose is pretty low frequency, matching the experimental data we see published in Russian experiments of ELF-UV release. This is also published in Roeland van Wijk's recent book, Light Sculpting Light.
This pathway is how cells use UV light from the sun to activate apoptosis pathways and prevents all cancers by deactivating disulfide bridges in the cell membrane proteins. HOW does this occur?

Throughout 4.5 billion year of molecular evolution, proteins have evolved in order to maintain the spatial proximity between aromatic residues (Trp, Tyr and Phe) and disulfide bridges (SS) (Petersen et al, 1999). There is a very special spatial geometric relationship that exists because the process is quantized to light frequencies. This has not been well appreciated by modern healthcare. The interaction of the most powerful part of the solar spectrum of light (UV) measures the collisions in the aromatic amino acids and in the disulfide bridges. The aromatic amino acids location becomes

the first step in determining where the position and geometry of residues to act as nanosized antennas in the protein world that can capture UV light (from ~250-298nm). This is determined by nuclear DNA coding and protein folding. Once excited by the incident ELF-UV light these amino acids can enter photochemical pathways likely to have harmful or beneficial effects on protein structures by affecting specific bonds like disulfide bonds in cysteine/cystine. These two amino acids are the most rare amino acids in our proteins, and as such can acts as the ideal photo-optical switch or gate for signaling. *It turns out cysteine/cystine disulfide bridges in proteins are known to be excellent quenchers of the excited state of aromatic residues by UV light in the literature.* This means these disulfide residues naturally decrease the power present in UV light created in the nearby excited aromatic amino acids of the skin. In this way, they contribute to protein stability and activity in the skin, thereby, stabilizing ubiquitin rates and lowering cancer risk. UV light excitation of the aromatic residues is known to trigger electron ejection from their side chains (Bent & Hayon, 1975a; Bent & Hayon, 1975b; Bent & Hayon, 1975c; Creed, 1984a; Creed, 1984b; Kerwin & Rammele, 2007, Neves-Petersen et al., 2009a). These electrons can be captured by disulfide bridges in things like glutathione, leading to the formation of a transient disulfide electron adduct radicals, which will dissociate photoelectrically, leading to the formation of free thiol groups in the protein. This photochemical change then leads to non optical signaling at deeper levels in the skin. Once disulfide bonds are broken in this way, we can inactive detrimental cell membrane receptors that cause epidermal cancers like EDGF and herceptin. The irony in all these details, is that UV frequencies prevent, and do not cause cancer, by these mechanisms. More irony for the skin and eye docs: This mechanism is now being used by big-pharma to develop drugs using nanotechnology and the ability of cells to make ELF-UV light. **END SIDE BAR**

Our modern healthcare perspective skews most to the beliefs that the sun is fundamentally bad for us. Based upon the side bar above you have to wonder if it is time to replace these beliefs. Based upon this new data, I have changed my own behavior to fit in line with what is published in this realm. The biophysics of the skin is new, but it is not unknown by science. It is just not well known that this science is at the edge of what we now know.

I have had patients with psoriasis come to me before their spine surgery and ask me about their low Vitamin D3 level and their altered bone density. They want to know what is smart sun exposure in their case. What I tell them today, is very different from my past, because my perspective now has been updated to reflect this quantum view point of how purple light and aromatic amino acids work. Psoriasis is believed to have a large "genetic component" like most cancers, but this new work shows, that these ideas maybe outdated too. I personally believe this is why the psoriasis, cancer, and autoimmunity is so misunderstood. Dermatologists and oncologists have been looking in the nuclear genome for the smoking gun for these diseases, when all along, the etiology was staring them in the face when they looked out a window. The lack of full spectrum sunlight is the missing piece. It is supposed to shines on the largest organ in the body, which is the skin, and the skin's thickness is determined by the amount of light that hits it and how effective the underlying circulatory system is in assimilating UV and IR light. These initially photoelectric interactions determines ultimately, how the atomic lattice of the skin interacts or did not interact, with specific frequencies of light we allowed to hit our skin and eye. If you share the same light environment in your family, it begins to makes sense why you might think there is a genetic component. The interesting aspects that links both cancer and psoriasis is how light plays a deep role in both diseases.

Light works via the photoelectric effect. This means light can be both a wave and a particle at the same time. **The key to which affect dominates in our tissues, seems to be the type and the arrangement of the atoms in our protein lattices that interacts with light first. Light interacts with the electrons of atoms and not its neutrons or protons.** This implies that the surface chemistry of where light first interacts with any electrons determines what can happen deeper in a tissue. Light dramatically alters how electrons are handled in plants and animals, but too few people in biology see the homology between them. In plants, photosynthetic capacity is controlled by a few variables. It turns out in humans, the same thing is true. The first step in both photosynthesis and in light's interaction with our blood plasma is charge separation of water into positive and negative charges to build a battery. This battery is the true source of cell power and not ATP as we currently believe. Gilbert Ling tried 55 years ago to point this out but was ignored. Today Gerald Pollack's work shows this effect clearly in experiments and published in his recent book.

However, when the surface layers of the human skin do not face full spectrum sunlight they cannot be atomically arranged properly. The key is the exact relationships of aromatic amino acids and sufhydryl groups in the skin. This environmental sun stimuli is need to gain proper geometric organization of the skin to work properly. A covered surface of artificial lit one, alters and varies the protein lipid raft arrangements. this changes the cytosocial behavior of the skin to other photochemicals, like nitric oxide, melanin, cortisol, DHA, and carotenoids. When these cytosocial changes occur over time so do sun light's photoelectric interactions. This causes mitochondrial heteroplasmy at deeper levels to alter the physiology of the skin. This alteration is what leads to diseases like psoriasis, in my opinion. Light can

drive growth or slow growth, simply by its interaction with the circadian clock timing mechanism in the skin.
In psoriasis, more highly powered sunlight gets through the surface layers into the deeper layers of the skin to drive growth rates higher. This interaction is important to understand. Light, especially higher powered low frequency light, (UV) is capable of increasing ubiquitin marking. This increases protein turnover because free radical signaling in mitochondria at deeper levels is altered. Singlet state radicals predominate over the triplet state. This explains how thickening of skin in psoriasis can occur with a lack of UV light exposure. Moreover, and more concerning, is that the development of psoriasis is also linked to future cancer's in other organs. Psoriasis is also associated with an increased risk of psoriatic arthritis, lymphomas, cardiovascular disease, Crohn's disease, and depression. All of these disease are linked to poor redox markers, increased mitochondrial heteroplasmy, and diminished DC electric currents at surfaces when measured.

Many of these diseases are associated with alterations of melanin, sulfated cholesterol, Vitamin D3 synthesis, RBC mass, anemia, and skin thickness. Patients with psoriasis get thicker skin in patches, and this mimics what we see in diabetics skin all over their body. Diabetics skin globally gets thicker because it undergoes glycosylation of the surface skin. This changes the optics of the skin in different ways.
People with psoriasis and diabetes get more cancers than those without these diseases further linking them both. Both conditions are associated with an inability to properly absorb sunlight into their plasma and RBC's. *Using a Quantlet to bio-hack these conditions might be a wise thing to consider longer term.* Since people with diabetes and psoriasis both have higher incidence of cancer, might this have something to do with the thickness of the skin or how light interacts

within their plasma? These findings link a lack of full spectrum light to an optically altered surface chemistry photoelectrically to both oncogenesis and metabolic syndrome. These linkages are what you saw me tease out and explain over ten to twelve blogs in the ubiquitination series.

Shocking links for psoriasis:

1. The odds of having hypertension are 1.58 times higher in people with psoriasis
2. A similar association was noted in people who have psoriatic arthritis—the odds of having hypertension were found to be 2.07 times greater when compared to odds of the general population
3. The incidence of the heart rhythm abnormality atrial fibrillation is 1.31 times higher in people with mild psoriasis and 1.63 times higher in people with severe psoriasis.
4. There may be a slightly increased risk of stroke associated with psoriasis, especially in severe cases
5. The real shocker: compared to individuals without psoriasis, **those affected by psoriasis are more likely to satisfy the criteria for metabolic syndrome irrespective of their weight.**

When your skin thickens and you have an altered melanin cycle, lower amounts of sulfated cholesterol and sulfated vitamin D in your skin, you become unable to properly absorb certain light frequencies because of surface changes to the skin. Once the skin changes then the plasma cannot be fully loaded with energy from the sun to charge separate water. This leads to mitochondrial heteroplasmy at deeper levels in tissues to change their physiologic profiles before any changes can be induced in the genome.

Want more shocking links: The rates of Crohn’s disease and ulcerative colitis are increased when compared with the general population, by a factor of 3.8 and 7.5 respectively.

Few studies have evaluated the association of multiple sclerosis with psoriasis, but the relationship has been questioned in the literature. Both diseases are linked by a blood plasma that cannot carry enough light energy to deeper tissues and to mitochondria. This causes rises in heteroplasmy percentages. Psoriasis has been associated with a 16% increase in overall relative risk for non-skin cancer. People with psoriasis have a 52% increased risk cancers of the lung and bronchus, a 205% increase in the risk of developing cancers of the upper gastrointestinal tract, a 31% increase in the risk of developing cancers of the urinary tract, a 90% increase in the risk of developing liver cancer, and a 46% increase in the risk of developing pancreatic cancer. The risk for development of non-melanoma skin cancers is also increased. Psoriasis increases the risk of developing squamous cell carcinoma of the skin by 431% and increases the risk of basal cell carcinoma by 100%. All of these diseases also show alterations in the skin thickness when you look for it……..few do.

It is my firm belief, with time it will be shown that alterations in surface chemistry will be more important to patients and clinicians because the initial surface interactions will determine the biochemistry that is possible below the surface. Psoriasis has markedly altered growth below the skin's surface and no one seems to be able to explain it. They continue to blame the nuclear genome, but is that really likely given what we know about how the photoelectric effect works upon surfaces? What about plants? Leaves have photoelectric surfaces don't they? Do they offer us clues to this mystery too? They do.

LETS LOOK AT PLANTS:

Leaf thickness tends to increase with decreasing rainfall, humidity and soil fertility, and increasing light exposure.

These are all stressors. In plants with specialize leafs that have very high photosynthetic capacity, thickness is related to pulsing of nutrients from its environment. For example, evergreen leaves are invariably thicker than deciduous leaves, and this has been interpreted as a means of nutrient storage (Mooney and Rundel 1979) or a response to pulsing of resources from their environment. Might the same "***pulsing of nutrients***" be why our skin thickness changes? We know in shady plants, their leaves are thinner as a rule. We also know that sun loving plants have thicker leaves. Any of you who have visited my house in New Orleans have seen how thick a magnolia leaf is off of my tree in my front yard. This tree is in sub tropical sun 24/7. UVA and UVB light is always present.
Plants can increase leaf thickness by altering how they utilize water. Strong light frequencies in the purple and blue ranges are known to dehydrates plant leaves faster, so protection of the leaf by increasing its thickness is a way to conserve water. Could artificial light alter our skin thickness, in some aspects we have yet to consider? Yep. This is why diabetics are dehydrated and why they urinate a lot and have thicker skin. The blue light environments they allow penetrates deep into the dermis and causes dehydration and calcium efflux. My members recently heard that in the webinar on EHS, the same mechanism was in play. Calcium efflux and dehydration change size and shape in cells don't they? They do.
In plants, leaf specific mass (LSM) is a useful index of sclerophylly which reflects the combined effects of density and thickness of leaves. Jurik (1986) has reported in the literature that LSM can also be used as a measure of carbon balance properties of the plant as it integrates various factors such as canopy structure, leaf area index, light environment and leaf photosynthetic performance. Photosynthetic performance = quantum yield for us. This is a plasma effect in the blood and this is one of the reason why the Quantlet can drive performance gains. Might you use it to bio-hack other aspects of diseases. That is what beta testers

will have the opportunity to do. It appears the environment dictates LSM. Could surface light some how determine animal skin thickness in similar fashion? After all, skin cancers like all cancers are tied to an altered carbon/nitrogen balance. This was the basis of the ubiquitin series of blogs. Psoriasis is believed to be disease of the basal cells and not the apical ones today. Today's blog will make you question that belief. Might it be a combination of both simultaneously? Yep. Let's jump back to leaves and their position on the tree for a lesson in nature. Net photosynthesis (Pn) and stomatal conductance (Gs) increases with leaf age. This is particularly true in the early stage of tree development. Pn reaches its maximum value when the leaves are completely expanded and exposed to sun. Leaves at different developmental stages in the tree's canopy from the apical position (top) to the base performed quite differently photoelectrically. Does leaf position and age of the leaf act like the skin layers mentioned above? Yes they do.

In plants, the transpiration rate and the vapor pressure deficits of older leaves on the base of the branch were higher than those of the younger leaves at the apex of the branch. Leaves at the bases of branches and tree have higher saturation vapor pressure and transpiration rates so this lowered their growth rates. In psoriasis, this is reversed. The basal levels show explosive growth. Why is that? It must mean that somehow powerful light is gaining access through the canopy of those surface cells to ignite this growth. Might the penetrating power be so high that the UVB light present in the middle of the skin, where vitamin D3 is made, be too powerful to make sulfated Vitamin D3? Yep. This is why Vitamin D3 is low in most autoimmune conditions. Is this powerful (purple frequency light) light stimulus delivered to the deeper levels in the skin than would be expected? Yep. Would this make the deeper level grow faster? Yep. How do plant surfaces look in comparison?

In plants and trees, both the apical and basal leaves have higher stomatal resistance and lower net photosynthesis, than leaves in an intermediate position. The intermediate position is where chlorophyll is found to drive photosynthesis in leaves. In your skin, this is where sulfhydryl groups are added to cholesterol and Vitamin D3. When the surface is altered because of a lack of UV and IR stimulus you lose this ability in the middle layers of the skin. Might the quantlet help this aspect of disease considering it can bypass a surface defect? Beta testers will want to bi0- hack this aspect for sure.

What we are all missing today in healthcare, is that these photo-chemical changes in the skin are made in quantized fashion because of the photoelectric effect. When they are off, they limit the time we get in our life. The incoming light energy has to match precisely, the proteins ability to absorb and assimilate the correct incident frequencies the sun emits, to get the desired results at deeper levels. All these effects require ideal thickness of the surface to use the sun correctly. If any of these variables are off, we lose the quantized ability of the photoelectric effect, as well as our ability to make Vitamin D3 in the middle layers of our skin. This is why Vitamin D3 is associated with so many circadian environmental mismatches. It also explains why all autoimmune conditions are linked to altered light environments. The specific matching of incoming light to skin thickness cannot occur properly in psoriasis because the surface skin lets in too much highly powered light to the deepest levels. The surface skin is dying and degenerating exposing the DNA to sunlight. DNA is an ideal absorber and dissipative chemical of UV frequencies. When this does not occur, UVB light, bypasses the middle layers of the skin because it only responds to tight light frequencies in the UVB range. Look at the picture of the skin again below in this blog. The answer is right there staring at you. This is why UVB radiation wavelengths of 311–313 nanometers have been shown to be most effective in treatment of this disease. Blue and violet light

penetrates deeply to stimulate growth. The altered frequency of this incident light changes the atomic lattice above and below and shows us how changing the skin thickness can manifest into a huge problem longer term. The more new autoimmune diseases a person with psoriasis gets the closer they get to cancer and to a loss of time. Normal skin makes sulfated vitamin D3 in 290-320 nm range.

Synthesis of pre-vitamin D3 in the skin involves UVB radiation which effectively penetrates only the epidermal layers of skin. 7-Dehydrocholesterol absorbs UV light most effectively at wavelengths between 290-320nm and thus the production of vitamin D3 will only occur at those wavelengths. The two most important factors that govern the generation of pre-vitamin D3 are the quantity (intensity) and quality (appropriate wavelength) of the UVB irradiation reaching the 7-dehydrocholesterol in the ***middle of the skin layers in the stratum basale and stratum spinosum.***

Note how UVB light only affect the most superficial layers in the skin. UVA goes deeper but blue light penetrates all the way to the dermis layers where the arterioles and blood plasma are located.

The skin consists of two primary layers: the inner layer called the dermis, composed largely of connective tissue, and the outer thinner epidermis. The thickness of the epidermis ranges from 0.08mm to more than 0.6mm (0.003 to 0.024 inches). The epidermis consists of five strata; from outer to inner they are: the stratum corneum, stratum lucidum, stratum granulosum, stratum spinosum, and stratum basale. The highest concentrations of 7-dehydrocholesterol are found in the epidermal layer of skin, specifically in the middle epidermal layers, the stratum basale and stratum spinosum. These are located in between the apical and basal layers. The arrangement parallels what we see in leaves where chlorophyll

is. The production of pre-vitamin D3 is therefore greatest in these two middle layers of skin, whereas production in the apical and basal layers there is reduced amounts of Vitamin D3 production. Do you think this is a coincidence or homology, due to the mechanism built into photoelectric effect and our surface photochemistry?

PHYSICS GEEKS PART 1: 7-Dehydrocholesterol is a *fluorophore protein* that acts to absorb UVB light and power down sunlight by changing its frequency. The energy associated with fluorescence emission transitions is typically less than that of absorption, the resulting emitted photons have less energy. As a result in coming light and are shifted to longer wavelengths and this slows light down and reduces its power. **This is how the photoelectric effect works**. This phenomenon is generally known as Stokes shift and occurs for virtually all fluorophores commonly employed in hydrated solution experiments. All human fluorophores require hydration to work accurately. A fluorophore is a dipole, surrounded by water molecules which is also a massive dipole. **When a fluorophore enters an excited state, its dipole moment will change, but water molecules will not be able to adapt this quickly. Only after vibrational relaxation, there will be a realignment of their dipole moments.**
What is the Stokes shift? In many cases, excitation by high energy photons leads to the population of higher electronic and vibrational levels (S(2), S(3), etc.), which quickly lose excess energy as the fluorophore relaxes to the lowest vibrational level of the first excited state. Because of this rapid relaxation process, emission spectra are generally independent of the excitation wavelength (some fluorophores emit from higher energy states, but such activity is rare). In this way fluorophore proteins in us, release energy to the surrounding water. The water can react to this change and it acts like a molecular mirror of the memory of this collision. In normal skin, we follow the mirror image rule, but in psoriasis skin we follow a Stokes shift, and the resultant

emission of light is what drives the growth of the basal layers of cells in skin.

PHYSICS GEEKS PART 2: Einstein says, if you shine light and ultraviolet light on a piece of metal, the light can knock electrons out of the metal. This was covered in the last blog in detail. In a lab this data is easily collected and proven. Back in the early 20th century, the photoelectric effect made science uncomfortable just like the story of Vitamin D3 does today for dermatologists. The wave theory of light suggested as you turned up the brightness of the light, you should eject more electrons, because brighter light has more energy in it. Why did they had trouble with Einstein's ideas initially? It was because in many experiments completed this did not happen. If you shine light onto a piece of an element, no electrons were ejected, no matter how bright the light was made. But when you changed the light to violet in the UV range, you could turn it down to the lowest intensities possible, and electrons will still be ejected. This is why all living cells emit ELF-UV. It is also why the Quantlet has some purple light within its design. This is how it optimizes performance gains. Einstein's genius was that he took the experimental data that others created and made sense of them, without getting caught in their dogmatic beliefs that light was only wave like. He realized that if light was made up of small particles called photons, the photoelectric effect became easy to explain. Max Plank had shown in years prior to Einstein's ideas, that it was the frequency of light, and not the intensity of the light, that determines its energy to do work. All that had to occur is somebody to prove Einstein's insight correct. Time 7 showed you somebody did just that.

NON GEEKS: The brain integrates and pays attention to the frequency of light in our retina, pineal gland, and our entire visual system; these systems are indirectly linked to the skin by the blood plasma and by sulfated cholesterol levels in our blood plasma and to sulfated Vitamin D3 levels; moreover,

they are linked to our gut surfaces via the vagus nerve. Is this also a funny coincidence or is it where form meets function at surface where light interacts? Is everything quantum at its core? Yep.

We know that the age of the leaf decreases photosynthetic capacity. That tells us light's interaction with Rubisco enzyme in chlorophyll must also have a time variable. Does it? Yep. How many times can incoming sun light be slowed down by the 4 nitrogens in a chlorophyll ring? Do cells have such a light timer? Yes, in fact, they have several. The SCN in the eye, neuropsin in the skin, and then we have peripheral clock mechanisms, and then something called the Hayflick limit in chromosomes. Are these all linked to telomere length's in cells in some fashion? Yep. In psoriasis, telomere lengths have been found to be altered in the basal skin. In fact, telomere length has been shown to be significantly reduced in all T cell subsets in psoriasis and in other skin disease like atopic dermatitis compared with normal individuals.
Telomerase is an enzyme-reverse transcriptase that protects chromosomes from degradation by stabilizing telomere lengths. Telomerase activity is increased and telomere length shortened in T cells from blood of patients with atopic dermatitis and psoriasis. HYPERLINK

WHAT ABOUT PLANTS?

Plants also regulate their photosynthetic balance to the presence or absence of light by altering the components involved in photosynthesis in quantum fashion. All plants are coupled to animal life by way of the CO_2 and O_2 cycles. Photosynthesis forms the basis of all food chains on the planet linking plants to animals. Photosynthesis operates most efficiently when the rate of CO_2 diffusion into the leaf matches the biochemical capacity of the leaf to fix CO_2. This is exactly what this entire blog is about. In psoriasis this linkage is lost. CO_2 is a gas that comes from plants and is

stored in our atmosphere. It can be thought of as a bio-plasma surrounding Earth that sunlight interacts with daily. The chemistry of the atmosphere varies day to night because of this interaction. We do the same things as plants do, using water and O_2 reduction in a mitochondria. Plants do it by altering stomatal conductance of their pores. We do it by how we construct our surface skin layers between sunlight and the arteriole system deep to the skin layers affected by psoriasis. The first step in both processes is the charge separation of water from sunlight. In psoriasis, this is lost. Might using a quantum performance device be helpful in a case like this? Beta testers of the Quantlet device might provides us an answer to this interesting question.

Does hot knife melt through butter, or does the butter melt from within when a hot knife encounters it? The answer seems obvious, until you realize that blaming the genome in psoriasis is akin to believing the latter is true about butter and the hot knife. Surface action is a huge factor, and biology is completely missing this today because they do not understand these photoelectric interactions. There is a reason for the global decline in Vitamin D3 and this blog is giving you a huge insight why it is occurring. The light humans have created is no sun and never will be.

Dermatologists have the privilege of examining the largest organ of the body. However, unlike other organs, there are hardly any tests of clinical significance that measure these critical skin functions besides a lack of Vitamin D3 in the blood. Might the Quantlet somehow help here? Bio hackers might want to pay attention to protocols that come down the pike. In dermatological practice, methods of evaluating the severity of skin diseases are often crude, subjective and not reproducible, which creates discrepancy in results and inter-individual variations. Hence, to maintain objectivity in

observations, scores are used to evaluate the severity of skin diseases. Most people have heard about the ABCDE criteria of melanoma, as an example. This is particularly important for monitoring the response to therapy and for evaluating the efficacy of new drugs. Over the years scoring systems have been developed for a number of skin diseases. This has greatly helped the cause of clinical practice and clinical research, but it has not helped patients outcome much at all. Why is this? Take a look at this hyperlink below of all the scoring mechanisms and review it carefully? What have they forgot to measure in all these scores? HYPERLINK

Skin thickness.

People with advanced HIV/AIDS often exhibit psoriasis. They too, have altered skin thickness. Here are some of the more common skin conditions related to HIV/AIDS. Molluscum contagiosum, herpes viruses, Kaposi sarcoma, Oral hairy leukoplakia, Thrush, Photodermatitis, Prurigo nodularis.
Do you know that HIV is also associated with a lot of new cancer diagnosis too? Do you think this trend with altered skin thickness and co-morbid altered solar spectrums will get anyone thinking better? Might cancer generation be linked to an alien sun? Is it a coincidence or might it be related to how light interacts photoelectrically with the atoms in our skin? Could this interaction change how cells deep to the surface layer work physiologically? Could this interaction drive elevated ubiquitin marking rates?

You bet your ass it does.

SUMMARY:

What is inflammation at its fundamental level? It is the excess of protons and/or loss of electrons at your surfaces. The surfaces being the eye, gut, and skin. pH is a function of proton concentration; low pH = high proton concentration = inflammation = lower EZ in water. Water is a chromophore for

red light in the solar spectrum. Once the EZ is formed by proteins filled with electrons touching water, the EZ grows massively when 270 nm UV light hits a surface. If you cannot build an EZ what kind of life do you get? It is optimal or suboptimal? Do you think a device designed to deliver purple and red light using surface cooling might have an effect on performance since it acts on this fundamental mechanism? I bet many Quantlet beta testers are already writing down bio-hacks.

Inflammation lowers our quantum yield to solar spectrum, just as putting a tarp on a tree lowers photosynthetic yield and slows growth. Thusly, inflammation sounds like the final common pathway of NRF2 and NFkappa beta pathways, which both tie back to calcium efflux, mitochondrial swelling and the increase percentage of heteroplasmy in a tissue. A lack of purple and red light exacerbate this situation in mitochondria because a chronic stimulus is given but no activation of the regeneration pathways are possible because of missing frequencies. Our species has acted to bury the sun over the last hundred years. This is a huge error because of a lack of understanding of the photoelectric effect. Without purple and red light we get dehydrated and we leak calcium. Calcium is the concrete for all of our cell membrane lipid bilayer. The more calcium we lose the more DHA we lose. The more DHA we lose the less we lose the ability to handle sunlight well. So we go inside under fake light with its massive array of blue and lack of purple and red light. We spend 95% of our lives under these lights. Calcium continues to leak out of cells, cells are stressed and chronically lose ELF-UV and our blood plasma cannot replace the losses. Calcium within the atomic lattices of our cell membranes keep vibrations to a minimum from our environment. Living under a microwaved Wifi world keeps those membranes vibrating like a concert piano being played 24/7. Normally, calcium keeps the lipid bilayers welded together to act as a native EMF antenna for the Earth’s magnetic field and the quantum waves of sunlight. All paths

lead to low plasma volume, loss of RBC mass, a decreasing the zeta potential of the plasma, with massive ongoing dehydration, altered skin thickness, and loss of all divalent atoms (Mg and Ca) in cell membranes and the blood plasma all at once. Do you think a device that can alter that balance might have a role? That change in blood plasma volume and chemistry is sensed by the 7 areas in the brain with no blood brain barrier. The most important one, and first one affected is PVN. This drives a stress response from the retina and the eye clock. The second one is in the area postrema that connects the vagus nerve to the brainstem to control the parasympathetic outflows of the brain. Might the photoelectric effect wave particle duality be represented by the two arms of the autonomic nervous system in how they function within us? Consider the following example: when someone gets a Candida infection in their aero-digestive tract, the area postrema is activated by a stimulus because the amount emitted light emitted by the gut and microbiome is lowered and altered. Are candida plaques in the aero-digestive system associated with increased thickness? Yes. This change alters surface interaction with light emitted from the microbiome of the gut or lung. The microbiome is made of bacteria which emit 5000 times more light than our own cells. The thickness of candida plaques lowers the amount of bacteria and its diversity to emit light while also causing a thickening of the mucosa below. These things lead to surface inflammation. What connects the gut or lung surfaces with area postrema? The branches of the vagus nerve do. This is the nerve that controls parasympathetic flows throughout the body. Stress from any stimuli cause increase firing of the PVN in the brainstem and this acts to break down all surface barriers to alter photo-chemistry at deeper levels. What is deeper to the gut surface? The gut associated lymphatic tissue layer (GALT). This is where T regulators cells control both arms of the immune response. This alters the interaction and direction of energy flows from the environment to the cells below where mitochondrial heteroplasmy increases

and autoimmune conditions manifest. This is how autoimmune conditions progress and cause other conditions. The surface chemistry of the eye, skin, gut, and lung, dictates what happens in the deep layers where mitochondrial are.

You can't out supplement, out hack, out exercise, or out eat a bad environment……that is the story of the time and ubiquitin series. Might the Quantlet help these condition's? Time will tell. It is nature's axiomatic rules of engagement that we understand the precise nature of photoelectric interactions in our tissues. Today's blog gives a small sliver of my current understanding of this process. So until that time does occur, then you the patient or the clinician must realize that buying supplements or good food, is only trading wellness for time. Psoriasis is a disease that shows you their is a deeply embedded quantum process of this interaction built into our surfaces.

Autoimmunity is not about food and never was. There is no illness set point for metabolism; too many patients and clinicians, however, believe otherwise. This is why diet is their first tool to engage a change. Growth and Metabolism are on a sliding scale of how the frequencies of light interact with the natural selection a proteins atoms to lead to stable protein cores under solar light. This is built into ubiquitin cycles. Ubiquitin marking monitors the photoelectric interactions of light with atoms in proteins. Its boundaries are limited only by the how we account for, assimilate, and contain the native frequencies of photons and electrons from our environment. Set points are creations of concrete reductive minds. That is not the work of evolutionary biology, nor is it following nature's law's.

If you believe the nonsense that food and metabolism are the ultimate controllers of your life ask yourself this: At what age do you hit your set point? The concept is vacuous. It confuses negative and positive feedback loops of stressful stimuli to regeneration cycling with an attempt to attain a

steady-state outcome. There is nothing steady state about life until one dies. I spoke about this in member webinars and in many series at length on this blog. Loss of negative feedback (ocular melatonin cycle) is deadly in a far from equilibrium state, why? In that scenario when control is lost, both prey and predator die. For example, when a patient gets a gut infection like candida from any stressor…….you've lost predator and prey in your microbiome, the biome gets smaller in number and species, because your surface chemistry is destroyed. You saw above how thrush, a candida infection, can also alter mucosal thickness. Candida, like psoriasis and diabetes, is also associated with an immunocompromised state and thickening of a surface. People think candida is something you catch or infects you. I think it manifests from an alien sun environment that we allow on several surfaces at once. That change above, ruins mitochondria below, and heteroplasmy increases and tissue function slowly crawls to the edge of an abyss. Obesity, heart disease, cancer, or autoimmunity are the diseases that await you next if you allow the light environment to drive the mitochondrial process further. Excessive artificial light, with a subtraction or purple and red light from light is the most common mechanism we see, but we remain blind to it because we are ignorant of surface chemistry induced by the photoelectric effect.

In a non-linear system, there may be no equilibria or there may be many. Either one is possible. That makes the process of life fundamentally quantum. All possibilities exists in this realm until the waveform is collapsed by the environment you allow. The light we allows is the measuring stick for how our mitochondria can react to build the reality we get. I think the 900 pound plus people we have in the USA and OZ have gone into a zone of the non-linear dynamics where the negative feedback loops in our cells ceases to limit fat mass or autoimmunity as a protective mechanism of a toxic environment to our surface chemistry.

I am quite confident that with time, surface chemistry will be shown to be more powerful than the biochemical pathways in deeper tissues at causing disease in humans. Why? Psoriasis shows you why I feel as I do. It is an uncoupling or light from your atomic lattice in your skin that cause hetroplasmy in mitochondria to manifest and lead to the phenotype skin disease it is today.

The take away from the blog: QED predicts that allostasis > homeostasis at every level. Everything is quantum and nothing zen.

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8.www.youtube.com/watch?v=KwbIR2yUziw (Doug Wallace talk on heteroplasmy in mitochondria)