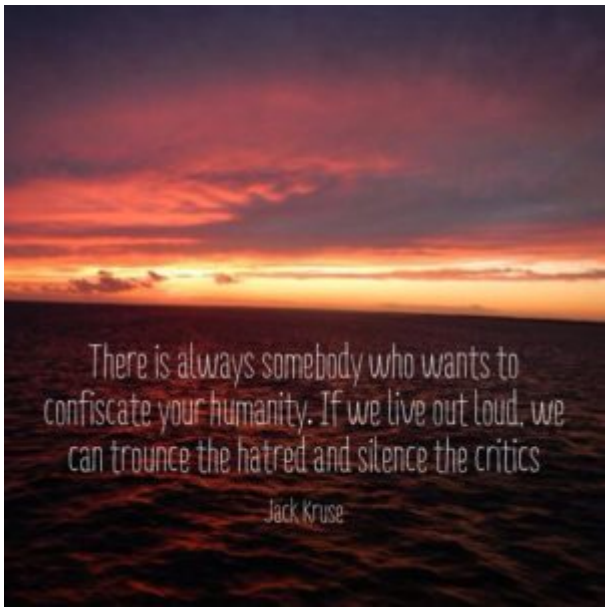
TIME #9: THE "DARK KNIGHT" OF REGENERATION?

BLOG TAKE AWAY: **Every morning you need the sun to charge separate water in your body to create the battery that creates your life. That life is created by a cadre of photochemical that do things we all rely on. You need to expose all your surfaces to this signals to make sense of the world around you. The food and exercise guru's that keep bathing in blue light and never see this message……..and blame foods and a lack of exercise for their ills. This blog shows you what you might miss when you focus only on food. Make like the Sphinx……While meditating in the morning sun, look to the East and ground and remain as connected to this environment as you can and food will never be your main driver. Light water and magnetism works to limit food influence at the confluence of your mitochondria. It's that simple a Rx to regenerate.**



Melatonin needs the sun's signal via neuropsin to regenerate our tissues. The second messenger's in this system are triplet state free radicals created from mitochondrial respiratory

proteins. These limit ELF-UV release from cells.

IS MELATONIN REALLY A SOLAR STORY TOO?

Most people think melatonin is our “dark hormone”. That is true, but few know that UV light is what regenerates the melatonin cycle in us. Melatonin teaches us a great many things about all the proteins in us that are biogenic amines that also work *with sunlight in novel ways*. Those proteins are *melatonin, serotonin, melanin, and dopamine*. They all use the sun in some very complex and counterintuitive ways. The end result of this transaction for all four is to deliver energy to mitochondria in a novel way. How it happens is the unfolding story in this blog, but it is based around how life works at the smallest levels in a cell. When a photon interacts with a particles in our cellular components on our globe, it lifts one electron from an electron pair in atoms in our molecules to a higher excited level. This excited state as a rule has but a short lifetime and the electron drops back within 10^{-7} to 10^{-8} seconds to the ground state giving off its excess energy in one way or another. Life has learned to catch the photon or electron in its excited state, and then act to uncouple it from its partner and let it drop back to the ground state, through its biological semiconductors utilizing its excess energy for life processes. Melatonin is a chemical that makes sure this process is orderly. In this sense, you can begin to see how living organism are organized. This makes sure that all forms of life are never at the mercy of their environments, on account of the coherent energy stored within them. Plants and animals use the power of the sun in different subtle ways. This explains why animals don’t have to eat constantly, leaving plenty of time for many other useful activities of daily living. It also explains why fat evolved in animals as a store for the sun energy so they could uncouple their metabolisms and burn fat to liberate heat when

the sun quality was poor. This is why people are getting fatter today. We are creating a low quantum yield world and we just do not see it. This allowed animals to live disconnected from the Earth and sun for period of times. This benefit was not afforded to trees and plants who are 100% connected to the Earth by their roots and the sun with their leaves and canopies. Fat mass allows animals to return entropy back to the environment while providing energy when food is not present. Food is ONLY IMPORTANT TO LIFE WHEN IT IS DISCONNECTED FROM EARTH, SUN, and WATER FOR LONG STRETCHES.

The sun excites electrons to allow life to move. Nothing happens in life until something moves. It turns out melatonin slows many things in nature down. Slowing atoms down allows melatonin cleans up the mess that living makes in sunlight to help it regenerate. Melatonin is highly fat soluble while also being soluble in water making it the ideal soldier for free radical scavenging anywhere in a cell. Things that interact with sunlight tend to be fat soluble and those that interact in darkness tend to be water soluble.

When life is organized to store energy, no part of the system needs to be pushed or pulled into action, nor is it subject to

“mechanical regulation” and control. Instead, it allows for coordinated action of all the parts. It is subject to timing, and it depends on rapid intercommunication throughout the system. The cell can thereby be thought of as a system of ‘excitable media,’ tissues, or organs called excitable cells poised to respond specifically and disproportionately to weak low electromagnetic signals. The excitable media is analogous to a dark mode plasma. Sunlight hitting tissues is how we are sensitized and animated to move electromagnetic currents in our dark mode state. Light is created from dark mode plasma only when resistance builds up to the solar plasma radiations. Since humans are made from a collection of semi conductive tissues this is why living things remain in the dark mode. When any living media is stressed it emits light and can be seen as ELF-UV light release via a GDV camera or a photomultiplier. Semiconductors by their very nature have very low resistance because they can accept current and move it very easily. This is why doctors do not see light when they cut you open.

Those excitable media are DHA, water, NADH and collagen are called “semiconductors” buried in tissues in many places. We have massive thin ones in our skin, gut, lung, and blood. Our brain and heart are loaded with them too because both organs are energy hogs. Anytime energy is transformed melatonin will be present locally. Energy in cells is neither created nor destroyed. It just changes shape under the power of sunlight and melatonin make sure the shapes of things are maintained when sunlight is absent. Water’s molecules shapes are changed by light to become the molecular transformer of lights power to electric signals during day and at night DHA changes the signal back to light when blue light is absent. Electromagnetic signals are the strongest forces in all of nature that bind the smallest particles in nature to innovate the most complex things in nature that live. Because large amounts of energy are stored everywhere in cells and tissues, they automatically amplify these weak electromagnetic signals

to often cause macroscopic actions in other atoms and molecules to control entropy to allow life to manifest. Melatonin, first made in our RPE, skin, and blood plasma all contain the ability to do these things in living things.

Let us briefly review the multiple actions by which melatonin reduces the damaging effects of daylight's ability to make free radicals and reactive oxygen and nitrogen species. It is well documented that melatonin protects macromolecules from oxidative damage in all subcellular compartments. This is consistent with the protection by melatonin of lipids and proteins, as well as both nuclear and mitochondrial DNA. Melatonin achieves this widespread protection by means of its ubiquitous actions as a direct free radical scavenger and an indirect antioxidant. This is how it lowers heteroplasmy in mitochondria. What is heteroplasmy?

A large number of pathogenic mtDNA mutations have been identified and the more severe mutations are frequently mixed with normal mtDNAs within the cell, a state known as heteroplasmy. Heteroplasmic alleles can shift in percentage during both mitotic and meiotic cell division, leading to a potentially continuous array of bioenergetic defects, a process known as replicative segregation. As the percentage of mutant mtDNAs increases, the resulting bioenergetic defect becomes increasingly severe. Because different tissues have different bioenergetic thresholds, as a patient's bioenergetic capacity declines it eventually falls below the minimum threshold for that tissue and symptoms ensue. Because the tissues and organs with the highest bioenergetic requirements are also those that are primarily affected in the common metabolic and degenerative diseases, it follows that mitochondrial dysfunction may be a major contributor to complex diseases.

Women that harbor deleterious heteroplasmic mutations have a high probability of having affected children, the nature and severity of the phenotype depending on the mtDNA mutation and

the percentage of heteroplasmy. Cells and individuals can accumulate an array of different mtDNA mutations over time, the aggregate of which degrade the energetic capacity of the cell. Such mutations are important in aging and cancer. Given the enormous potential explanatory power of heteroplasmic mtDNA mutations, it is striking that very little is known about the origin, genetics, and phenotypic effects of heteroplasmic mtDNA mutations.

Intermittent fasting works because it raises melatonin levels. But intermittent fasting is only optimized when the time we are spent fasting is when we are connected to the solar cycles and the Earth's magnetic field. This can be extended if we add meditation to the mix. Thus, melatonin directly scavenges a variety of free radicals and reactive species including the hydroxyl radical, hydrogen peroxide, singlet oxygen, nitric oxide, peroxynitrite anion, and peroxynitrous acid. Furthermore, melatonin stimulates a number of antioxidative enzymes including superoxide dismutase, glutathione peroxidase, glutathione reductase, and catalase. Additionally, melatonin experimentally enhances intracellular glutathione levels by stimulating the rate-limiting enzyme in its synthesis, γ-glutamylcysteine synthase. ***Melatonin also directly links to neuropsin function in the eye.*** *Melatonin inhibits the pr0-oxidative enzymes nitric oxide synthase and lipoxygenase on our surfaces in the presence and absence of sunlight in novel ways. These two enzymes are key photo-chemicals in the skin.* ***Melatonin and DHA are destroyed by artificial blue light after sunset.***

HUMAN BRAIN, MELATONIN, NEUROPSIN

In mammals the lipoxygenases isozymes are involved in the metabolism of eicosanoids. The eicosanoids are local hormones that are photoactive that work with and because of melatonin. These chemicals are prostaglandins, leukotrienes and non classic eicosanoids. Lipoxygenase are a family of *iron-containing* enzymes that catalyze the dioxygenation of polyunsaturated fatty acids like DHA in lipids containing a cis,cis-1,4- pentadiene structure. It catalyses the following reaction:

Fatty acid + O_2 = fatty acid hydroperoxide

This reaction is critical to all life because they make eicosanoids. The eicosanoids are considered "local hormones." Within the human brain they are critical to regenerative sleep. this is why melatonin is so critical in tissues that adapted from all neuro-ectoderm structures in humans. The skin and brain are key tissues derived from neuroectoderm. How does light link the tissues derived from the neuroectoderm? **They use a photochemical protein called neuropsin to make melatonin to control this process locally in WBC's, skin, and all epithelial derived cells.** This is why low melatonin levels are associated with cancers in these tissues. Neuropsin (Opsin-5 or OPN5) is a photoreceptor protein

sensitive to ultraviolet (UVA) light. **It is found in the skin and retina and allows the retina to maintain a separate biological clock from the peripheral clocks.** *This means the bio-hacker can take advantage of their environment when they know how it works*. What helps neuropsin signal properly normally? Sunlight, full spectrum AM and afternoon sunlight.

Time *re-creation* begins and ends with this neuropsin light meter in the cornea of your eye, fueled by UVA light because it forms the basis of the regeneration program that melatonin uses on cells. Melatonin and neuropsin recycle time by recycling mitochondria by lowering the amount of heteroplasmy a cell has. So if you have implanted cataract lenses that block UV light all hope is not lost of you. If you have implanted lenses, be earnest and not hip, and make sure you avoid eye wear fashions. *Make sure you skip sunglasses once you understand the power of this blog, and get your eye to see the full solar spectrum*. Get rid of your glasses and contacts, to regain you regenerative powers of neuropsin and melatonin. This sounds hard to fathom, doesn't it?

Your skin, cornea, and blood cell's have an ability to sense light without using eyes to make you young by raising local melatonin levels to lower the % of heteroplasmy in these cells mitochondria. This directly affects telomerase function in these cells to lengthening chromosomes to increase health to increase longevity. The synthesis and timing of melatonin production begins in the eye because it requires an afferent signal from light in the cornea and the SCN which projects to the hypothalamus and the paraventricular nucleus to the pineal gland and the *superior cervical ganglion*.

Neuropsin is one of seven related "opsin" proteins in mammals. Four enable the rod and cone cells of the retina to absorb light of different wavelengths and transmit that information to the brain so that the eye can see images. Another opsin, melanopsin (UBI 24), also absorbs light but uses it to guide processes like pupil constriction and circadian rhythms. It is

found in nerve cells that connect the retina to the body's master clock, the suprachiasmatic nuclei of the brain. On its own, this master SCN clock tends to run slower than 24 hours in humans (day dwellers), and faster than 24 hours in mice (nocturnal), *so it needs to be constantly reset to the light/dark environment by signals from the retina.* Nearly every tissue in the body also has a local molecular "clock" for regulating patterns of activity, but most of them cannot be reset by incident light on their own as the retinal clocks can. This makes the eye very special. Instead, all but one of these molecular clocks are synchronized by the master clock within the brain (SCN)—the exception being the retina, which maintains its own rhythms while sending the master clock the signals it needs to set the light-dark activity synch for the rest of the body.

Russell van Gelder, M.D., Ph.D., a professor of ophthalmology at the University of Washington, studied the circadian rhythms of genetically tweaked mice *that were missing rod and cone cells and melanopsin*. As expected, the circadian rhythms of these mice continued cycling but could no longer adapt to changes in light exposure. Surprisingly, though, the activity patterns of their retina's were still responsive to light changes, suggesting that there was another pigment in play in the eye and skin. **That pigment was neuropsin.**

The retina is the only tissue known to ignore the master clock based in the SCN, but it does keep itself on a schedule, based upon all the data we have. That made me ask the question, how does this occur?

I looked at articles where opsin were removed from animals (see above). It turned out the retinas of the mice ***without neuropsin lost their ability to adapt to new patterns of light and darkness. I found out that neuropsin and melatonin function must be coupled because melatonin is the hormone of darkness that is regenerated by daytime later morning UV light (9-11AM). Was neuropsin key in this recycling of melatonin?***

Does neuropsin also work with UV light in some way during the day?

The scientists working with these mice began by repeating their experiments with different wavelengths of light, the they found that neuropsin responds to UV-A and violet light up to 400 nm. This implied that the retina uses separate light signals based upon frequency to set its own clock and that of the body's master clock in the SCN. The central retinal pathways controlled by melanopsin are exquisitely sensitive to light in 435nm-465nm light which is based in the blue/green part of the spectrum. 400-430 nm light is present in early AM sunlight, before UVA shows up, and it becomes very prominent at dusk when the sun slowly sets. These frequencies of light are ideally absorbed by melanopsin over several hours at dusk. Melanopsin can be thought of as an opsin that pays attention to light as it dims. Regarding neuropsin, we still don't know precisely what kind of signals, either photonic, chemical or electrical, that neuropsin uses to set the retina's clock. My current bet it will be found to be photoelectric because of another clue the eye has given us. *Neuropsin is found in the cornea and the skin and this makes it very unique.* Why would we have a UV-A light sensor for bright mid-day light frequencies in a transparent cornea? Since neuropsin works via UV-A light this is a "tell" that our modern beliefs that the cornea and lens block UV light is a pure fallacy. It also tells us that non UV-B sunlight is important to melatonin recycling.

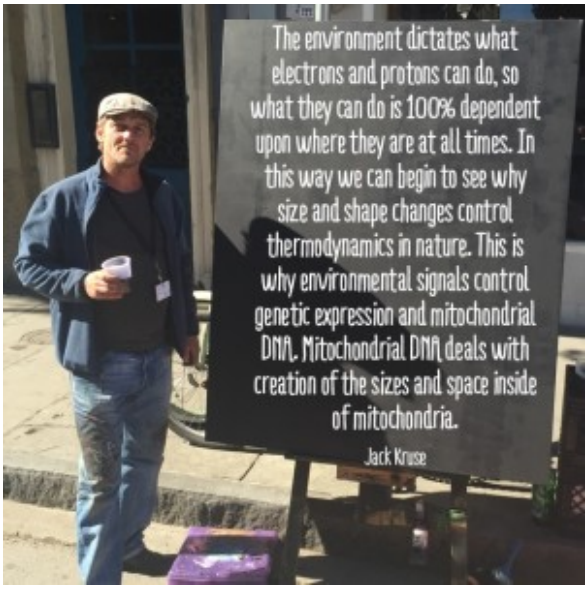
How did we find out neuropsin was in the cornea? Researchers used a specialized "locator gene," called a reporter gene, to figure out where neuropsin does its work. They found that, like melanopsin, it is located in neural cells that connect the retina to the brain via the central retinal pathways. From there though, we do not know precisely yet today where these particular cells go within the human brain. Using this

technique they did mysteriously confirm it is present it in the cornea. This shocked many, including the eye doctors. The cornea was not thought to contain any UV pigments by ophthalmologists, since its job is to let light through to the rest of the eye. We now know this data needs updating.

MY SPECULATION ABOUT NEUROPSIN AND IT LINK TO UV-A LIGHT

I currently believe neuropsin was selected for by the KT event in eutherian mammals that made it through the last extinction event. During this time it is believed that photosynthesis was disrupted for several decades to 1000 years. *This would have lowered the quantum yield of sunlight on the entire planet.* A lowered quantum yield of sun light would have sharply lowered UVB light more than UVA light. Mammals that survived would have needed some way to get environmental signals to their pituitary gland to drive the thyroid hormone cycle needed to drive seasonal changes and to control reproduction. Without these two factors, no mammals would have survived very long, in a low quantum yield environment. In today's world, we now see the same similarities in our environment but for different reasons. **As a result, in humans, thyroid diseases and fertility changes are now at pandemic levels.** I believe that UV-A light is critical in driving thyroid cycling in all modern mammals including humans. When we see massive spikes in hypothyroidism, it is a sign that there is a global lowering of the quantum yield of sunlight for some reason. 65 million years ago it was debris from an asteroid that cause blockade of specific frequencies of light. Today, it is non native EMF in the ionosphere that are destructively interfering with sunlight that falls to the Earth surface. This explains why we have a global Vitamin D3 pandemic and why we see massive spikes in hypothyroidism and infertility. Free T3 is needed for fertility (*leptin*) and for nerve function. Low free T3 levels also are associated with chronic pain and severe alterations in cardiac function. Free T3 levels have been studied in post heart surgery patients and

been found to be an very good reliable outcome measure. Free T3 tends to trend with melatonin levels because both need UV light exposure to recycle themselves in mammals. Low free T3 levels is a sign of low UV light exposure.



Somatic cells are kamikaze's for support of the germ line and melatonin is their General. Leptin protects that cell line by making sure it has light and electrons to maintain its existence. Somatic cell death requires the creation of a biologic timing mechanism locally, hence why every somatic cell has a clock gene in front of it.

Since eutherian mammals made it past this event horizon it stands to reason that this gene should be highly conserved in all mammals that survived. So what does the data say about my idea?

Within placental mammals, neuropsin is extraordinarily conserved, with percent identity relative to human protein 96%

averaged over 31 species; exceeding the 95% percentile of all coding genes proteome wide! That conservation drops considerably at marsupials and monotremes (86%), is less striking at tetrapods (78%), and not especially remarkable at teleost fish (68%). This pattern suggests neuropsin acquired significant new adaptive functionality on the placental mammal stem, leading to marked resilience to fixation of any further variation for the last 65 million years. That is why I believe as I do today. My beliefs are always subject to new data.

Does this explain why the modern world faces a pandemic of hypothyroid disease?

It might. Why? How does this link to thyroid function? Most people in medicine today know about thyroid function but few can explain why hypothyroidism is now at pandemic levels. I think a global lowered quantum yield of sunlight on Earth maybe the reason. This sounds hard to fathom, because few really understand how the thyroid is controlled by light. In 2010, I came across a study that gave me some insight to build upon this idea that I wrote about in Cold Thermogenesis 4 and Cold Thermogenesis 6. Thyroid function, cold, and UV-A light exposure had to be linked in some way that biochemistry and medicine have not yet fully appreciated. The linkage seems to go all the way back to the last time the Earth faced a lowered quantum yield. The KT event was the last time Earth faced a "*serious brown out from the sun".* Neuropsin evolution was likely how life back then dealt with the last brown out to survive the event. I believe neuropsin isoforms were re engineered in the early eutherian mammals and therapy dinosaurs to absorb in the blue range to overcome the loss of sun light. This helped them live in a low quantum yield world and also allowed them to uncouple their inner mitochondrial membranes to make more heat. This radically changed mitochondrial haplotypes in eutherian mammals. The UV-A neuropsin has re-evolved in mammals as the sun's power was

restored. The link of blue light neuropsin's in bird and nocturnal mammals links to melatonin regeneration explains why life could survive in a world without full spectrum sunlight. After the asteroid impact UVB light would have been non existent on Earth because of the particulate matter in the atmosphere. UV-A light would have been present but rare. This is why I knew to look at the skin and eye surface for a possible link for a light sensor for blue light. I looked at the other animals that made it through the KT event beside eutherian mammals. They were the therapod dinosaurs. Their modern correlates are birds. Quails happen to be birds.

The 2010 study I mentioned earlier was about the quail brain. It mapped the bird version of neuropsin (NEUR1) to the paraventricular organ, a region of the diencephalon of nonmammalian vertebrates containing *aminergic neuronal cell bodies* (biogenic amine correlates in mammals) beneath the epithelial membrane lining the third ventricle. The Paraventricular region is where the PVN is located and controls the sympathetic nervous system and it very sensitive to the UV spectrum of light from the retina. The PVN is critical in the stress response and ROS signal from mitochondria from many different environmental signals. This peaked my interest because of what I imagined occurred globally 65 million years ago, to the quantum yield of sunlight, off the coast of Mexico. The modern vertebrate forebrain's diencephalon also contains the thalamus, hypothalamus and posterior pituitary. This linked many areas of the mammal brain to vital functions.
The specific idea laid out in this paper was that incident UV-A and violet light would be sparsely present, but would be clearly able to be detected by the bird version of NEUR1 opsin in paraventricular neurons. This would allow signaling via cerebrospinal fluid to the pars tuberalis of the pituitary gland, inducing there thyroid-stimulating hormone was present. This, in turn, would induce the thyroid deiodinase DIO_2 in tanycytes lining the third ventricle to affect fecundity and

seasonal adaptations. The 3rd ventricle is filled with CSF, which is 99.8% water. Light and water are critical in forming life's battery over the 4.5 billion year history on Earth. This interaction would lead to a photo-electric signal that produced a daylight long signal. This signal induced T3 in the mediobasal hypothalamus, and ultimately could have induce gonadotropin-releasing hormone to the testis and ovaries to control seasonal reproduction. Light and metabolism have always been linked to fecundity in birds and mammals. These are critically important factors, as I laid out in the last chapter of my book, The Epi-paleo Rx. In this paper, they mentioned something rather startling to me about light frequencies and the bird opsin. The peak adsorption was observed to be at 420 nm (blue) in this opsin and it was not readily transferred to orthologs in other species. They found this out by studying genomic alignment and tuning residue formulas. This meant birds faced something rather unusual in their evolutionary past, and it changed their genome in a specific way tied to light. Their opsin responded to blue and not UV light. Why would birds have an opsin that responded to blue and not UV light? Moreover, why would nocturnal mammals also have blue light neuropsin's and why would humans have an opsin that responded to UV-A light? The KT event fits this scenario quite well because of the brown out the two surviving animals were birds and small nocturnal mammals. I do not believe it is a coincidence that 420 nm is right below melanopsin's ideal optical range in today's modern versions of mammals. This made a lot of quantum sense to me considering what an asteroid would have done to the sun's power and how it would have shuttered certain frequencies between the UV and blue band during daytime hours.

A commentary piece assigned to this article linked the the paraventricular organ to the hypothalamus in modern animals. I immediately thought about the PVN in humans. I went digging and found that even though no exact mammalian anatomical counterpart exists completely, the Allen Brain

Atlas shows mouse expression of NEUR1 there (blue range). Things began to become more clear. This had to be how light and water helped birds and mammals survive the KT event. They survived because they has strong regeneration programs despite the fact UV light was nearly absent. These animals were able to navigate a low quantum yield event, by innovating an opsin in the skin and eye that responded best to a world that had buried the sun because of a bollide. This version of opsin allowed them sustain higher heteroplasmy percentages because they used the relative darkness of their environment to control regeneration. Melatonin was more important to these animals 65 million years ago than was the loss of daytime UV light. Plants are also capable of using blue green light in their photosynthetic centers too to sustain the food webs to allow for a recovery.

The hormone that controls dark = melatonin. When quantum yields are low from sunlight for any reason, sunlight would be like AM sunlight is today, darkness would be more plentiful. There would have been equal parts of blue, green, and red in this light after such an event. The particulate matter would have lowered the amount of UV light considerably. *This is how I believe neuropsin and melatonin made their alliance from an evolutionary perspective 65 million years ago in birds and eutherian mammals.* This all of course, assumes that the photoperiodic reproduction hormomal control had to be directed by the light and dark mechanism under melatonin's control. **It should be no surprise that even today, melatonin controls mitochondrial DNA heteroplasmy percentage.** This was a big clue for me why we are seeing the diseases we do today. This gives melatonin the master control switch that deals with the percentage of heteroplasmy and mito-nuclear co-aptation that Doug Wallace has laid out for modern biology. This melatonin/neuropsin mechanism is retained from the ancestral situation in all animals that survived the KT event. Even today, the paraventricular organ is known from teleost fish and frog as well as birds. Photoentrainment of daily

circadian activity rhythms should be expected to be quite distinct in terms of opsin use from photoreceptor control of seasonal reproduction. This idea fits precisely with what we now know about the human NEUR1 transcripts most commonly recovered from testis and ovary. Want some proof for this speculation? Google neuropsin transcript DB097202. It has a massive phylogenetic range back to amphioxus and sea urchins, past which, unfortunately nothing is known today. **END OF MY NEUROPSIN/MELATONIN SPECULATION**

THE BRAIN'S PUFA's AND MELATONIN

The brain contains two main polyunsaturated fatty acids (PUFA), arachidonic acid (AA) and docosahexaenoic acid (DHA). These PUFA are located almost exclusively in the SN2-position of phosphoglycerides which are found in the neural cell membranes. Liberation of these PUFA from the phosphoglycerides occurs via the action of specific phospholipases (PLA2). Free AA can be metabolised by cyclooxygenases to prostaglandins and thromboxane, while both AA and DHA can be metabolized by lipoxygenases to form hydroxy derivatives and leukotrienes. AA is also metabolized to lipoxins via the 5-lipoxygenase pathway. The eicosanoids formed play important roles in neural function including sleep induction (PGD2), long term potentiation, spatial learning and synaptic plasticity (PGE2), resolution of inflammation (lipoxins) and anti-inflammatory and neuro-protective bioactivity (dihydroxy-docosatriene, neuroprotectin D1, formed from DHA). COX-inhibitors have been shown to reduce oxidative stress and cognitive impairment. Additionally, drugs which are used to treat depression have been shown to reduce the turnover of AA to PGE2 in the brain. **Diets deficient in omega 3 PUFA lead to reduced DHA in the brain and increased turnover of AA to eicosanoids with lowered melatonin levels locally and globally in the human body;** this is an effect which is overcome by restoring the omega 3 PUFA to the diet. In neural trauma and neurodegenerative diseases, there is a dramatic rise in the levels of AA-derived

eicosanoids. In contrast, DHA-derived compounds can prevent neuroinflammation. Clearly, the eicosanoids are very important for the normal functioning of the brain, while the PUFA themselves are important in membrane structure and function. Both need to be present together. If the neuropsin melatonin cycle is broken eicosanoids cannot protect DHA and this ruins the SCN clock and increases inflammation.

Finally, there is evidence that melatonin stabilizes cellular membranes, especially around mitochondria DNA, thereby probably helping them resist oxidative damage. Most recently, melatonin has been shown to increase the efficiency of the electron transport chain and, as a consequence, to reduce election leakage and the generation of free radicals. *These multiple actions make endogenous melatonin a potentially useful agent in the treatment of neurological disorders that have oxidative damage as part of their etiological basis. Most neurodegeneration is associated with very altered melatonin cycles. This is why sleep is disrupted in these patients early on in their diseases.*

The precursor to melatonin is serotonin, a neurotransmitter that itself is derived from the aromatic amino acid tryptophan. Aromatic amino acids are relatively nonpolar. To different degrees, all aromatic amino acids absorb ultraviolet light. Tyrosine and tryptophan absorb more UV than do phenylalanine; **tryptophan is responsible for most of the absorbance of ultraviolet light at 280 nm by proteins in humans**. Tyrosine is the only one of the aromatic amino acids with an ionizable side chain. Tyrosine is one of three hydroxyl containing amino acids. 5-hydroxytryptophan is the base molecule for serotonin that eventually forms melatonin in tissues. UV light is critical in this dance, just as it was for tyrosine and UV light and dopamine creation. Melatonin is a UV tryptophan story that links to serotonin.

UV light at 337 nm is capable itself of photoactivation of the 5-hydroxytryptophan decarboxylation reaction producing

serotonin. This has been observed in many experiments. The photoactivation effect was investigated as a function of fluence rate and fluence, and pH. pH links the photochemical effects to protons because it affects the exclusion zone in water. The exclusion zone affects the charge separation in water. CSF carries a lower pH at dusk after being oxidized all day by light's effects on our tissues. Photoactivation of decarboxylase activity was found to occur at nearly neutral pH values (low activity of the enzyme in the dark). The findings indicate that the effect of light is similar to a pH shift toward the acid region (low pH), which causes the ***enzyme conversion from the inactive to active form.*** Here you see why I believe a low acid pH is needed to make melatonin from serotonin properly at night when light is missing from striking our surfaces from the environment we allow. The 337 nm light is likely the critical frequency in the small bowel that the controls quorum sensing in the microbiome. I am sure this frequency is also important in the photochemical actions of the superior cervical ganglion, and within the CNS. Pyridoxal phosphate, is the decarboxylase cofactor, in the form of an adduct absorbing protein it is optimized at 330-340 nm. This is why it has been suggested as a candidate for the role of the photoactive chromophore of decarboxylase reaction critical in melatonin synthesis. **90% of all serotonin is produced by the microbiome in our gut. It does not come from food as most believe. Remember bacteria release massive amounts of light. That light and the aromatic amino acids in serotonin are what make the gut our melatonin factory for the brain. So how does the gut link to the brain? An environment with a low quantum yield is a huge problem for the regeneration pathways. The natural fix is in the eye:**

NEUROPSIN

Melatonin appears to the guardian of the Mitochondrial genome of 13 genes. All of these genes code for the proteins that tunnel electrons and spit protons out of the mouth of cytochrome proteins. The mitochondrial genome undergoes 3 times as many genomic mutations as the nuclear genome by design. Melatonin levels are critical in monitoring this behavior. The major function that melatonin help smooth in mitochondria are

1. Energy metabolism and flux via control of electron and proton tunneling.
2. Redox balance within the mitochondria
3. Ion homeostasis
4. The signaling of cell death and mitophagy = % heteroplasmy in a mitochondria

ARE DREAMS A CLINICAL SIGN OF MITOCHONDRIAL HETEROPLASMY?

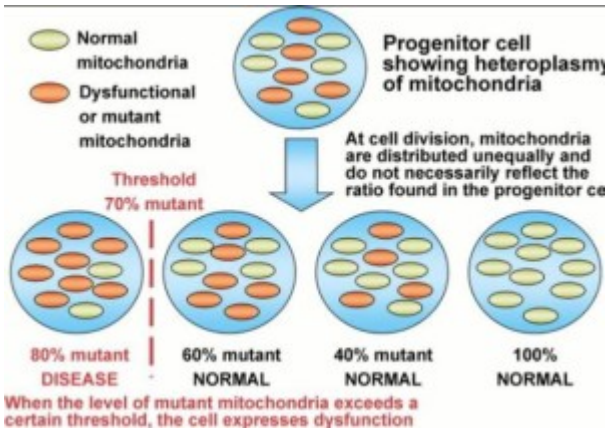
Might dreaming be tied to the harvesting of serotonin stores being converted to melatonin? I think so. Might dreams be a photoelectric spark tied to the 337 nm ELF -UV light from cells after the absence of light for 2-4 hours? Yep. Might the lack of dreaming be a sign of a poor conversion because of some type of light pollution from the eye and gut to slow energy metabolism in our mitochondria? I think so. This is

why I told you in the Leptin Rx to not eat after dinner and not to eat after 7 PM. Not only is poor sleep a sign of stunted longevity, but it causes an altered regeneration in all cell lines. This is associated with a lowered DC electric current during daylight and an absence of dreaming at night. Might lack of dreaming be due to a lack of energy loss from light from mitochondria in some way? Might too much UV light release from cells chronically, also lead to symptoms as poor dream creation? I think so. Why do I think this?

The major store in the body of serotonin is found within the first 20% of the small intestine. Peripheral serotonin is produced in the digestive tract by the interaction of the microbiome and enterochromaffin (EC) cells. It is modified by particular types of immune cells and neurons. After four hours of darkness the gut should quiet its motor function and begin releasing ELF-UV to command this conversion of serotonin to melatonin provided there is no photonic interruption from extraneously light via the eye or gut surfaces. I would remind you light is released from the microbiome when there is movement in the small bowel or we eat. This is why in sleep phases we have gut small gut paralysis. I believe the serotonin is harvested and decarboxylated to melatonin that will fill up our portal circulation to help reload our circulatory system and WBC's with melatonin. The melatonin made in the brain needs serotonin from the gut to travel via our vagus nerve and autonomic plexus to reach the pineal by way of the superior cervical ganglion. This only occurs when peristalsis is absent and blue light is absent from the retina in the 400nm to 480 nm range. When it is not absent you don't dream well. If it persists you won't sleep well either. Within the pineal gland, serotonin is acetylated when blue light is absent from the central retinal pathways; simultaneously the gut is quiescent, cells in the CNS are actively releasing ELF-UV to signal the conversion of serotonin to melatonin for sleep induction. This light UV frequency is likely to be critical in the methylation of

serotonin substrates to yield melatonin.

Sleep always begins in stage one. Might this be why during this initial stage of sleep we experience strange and extremely vivid sensations or a feeling of falling followed by sudden muscle contractions? Might those contraction be due to a change of frequency occurring in mitochondrial oscillations? My June 2016 webinar has some interesting thoughts on this. Could this be serotonin substrates altering our consciousness by altering ELF-UV light release? Could these muscle changes be phenotypical responses of how we move from the photochemical stage of sleep dominated by CSF pH changes to photoelectric control in the absence of light to alkalize the pH and stimulate the release or ELF-UV light from cells to make melatonin? I think so. During this time cell are ketotic; this raises NAD^+, while improving electron chain transport speeds which increases tunneling speeds of electrons from our fat stores. This is why melatonin is associated with higher tunneling speeds in mitochondria. As tunneling speeds increase more oxygen is drawn to the terminal aspects of mitochondria demolishing pseudohypoxia. This increases their magnetic flux. oxygen is drawn to mitochondria at this time because oxygen is paramagnetic and all things paramagnetic are drawn to a magnet. During these speed up phases during the initiation of sleep, mitochondria are known to create a superoxide burst at cytochrome 1 to lower the **heteroplasmy percentage** in mitochondria. *This regenerates tissues with mitochondria and pushes cells further from illness.* These reactions in stage one sleep are known as hypnogogic hallucinations. This is how life regenerates in quantum fashion in my opinion.



MELATONIN CONTROLS LOCAL HORMONES THAT CONTROL THE LOCAL EFFECTS OF LIGHT

How? Melatonin is known as a neurohormone because it is produced in the blood plasma by white blood cells (leukocytes) and within the pineal gland that regulates sleep and circadian functions. Melatonin also regulates inflammatory and immune processes acting as both an activator and inhibitor of these responses. Melatonin demonstrates endocrine, but also paracrine and autocrine effects in the leukocyte compartment of blood: on one side, leukocytes respond to melatonin in a circadian fashion; on the other side, ***leukocytes are able to synthesize melatonin by all by themselves!!!*** With its endocrine and paracrine effects, melatonin differentially modulates pro-inflammatory enzymes, ***it affects leptin sensitivity***, it controls production of inflammatory mediators such as cytokines and leukotrienes and regulates the lifespan of leukocytes by interfering with apoptotic processes in cells and mitophagy in mitochondria. Moreover, its potent antioxidant ability allows scavenging of oxidative stress in the inflamed tissues that interest me most. Melatonin is an amazing biogenic amine. The interesting timing of pro- and anti-inflammatory effects, such as those affecting lipoxygenase activity, suggests that melatonin might promote early phases of inflammation on one hand and contribute to its attenuation on the other hand, in order to avoid complications

of chronic inflammation. Human lipoxygnease are known by their short hand name called LOX's enzymes. Why am I spending so much time explaining these linkages?

MELATONIN MEETS DHA

LOX enzymes are how we recycle DHA into things that are highly anti-inflammatory secondary messengers called eicanosoids. Eicosanoids are the local secondary signal transducers that have a major impact on human homeostasis and energy balance. Melatonin re-cycling controls their creation. **In this way, melatonin is the "gateway" neurohormone between regeneration and inflammation.** For this reason they are involved in many disease processes such as inflammatory responses, cancers, cardiovascular and kidney diseases, neurodegenerative disorders and metabolic syndrome. This is how melatonin modulates things in our CNS and especially in our retina. It does the same thing on all our surfaces where sunlight impacts cellular components. This is why I have been aiming for quite sometime at giving you a comprehensive overview of how biogenic amines (think dopamine) control various inflammatory pathways. Melatonin regulates all our biogenic amines making it a "*pleiotropic hormone*". Melatonin has many different functions in light and dark environments. ***It used to be the "boss of cells" until big brains were invented by evolution.*** Melatonin is the protector of DHA stores in cell membranes and the protector of our mitochondria DNA. It is where the rubber meets the road in human biology. Before leptin evolved to specifically control energy balance and fecundity, melatonin was the gatekeeper hormone of the central nervous system of eukaryotes. ***Melatonin still is boss over mtDNA but not over nuclear DNA.*** In higher eukaryotes, leptin now modulates nuclear control factors we call epigenetics. Melatonin is more like a former head of state; it wields incredible control over growth and metabolism programs because of its linkages to light and darkness. This makes melatonin a chameleon, like hydrogen, like water, like sunlight

frequencies. It is a jack of all trades and master of none. *In science, we call this behavior pleiotrophy.*

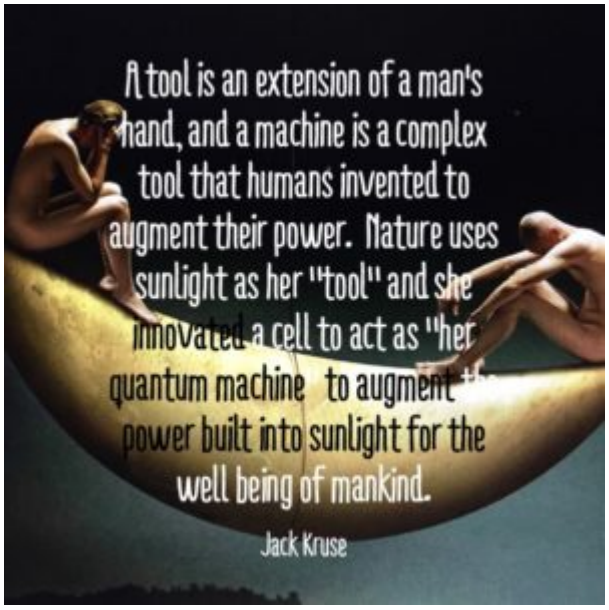
PLEIOTROPHIC = a chemical or gene capable of producing more than one effect; especially : having multiple phenotypic expressions. This chemical can open the door to heaven or hell if it it is not controlled properly. Melatonin is the most pleiotropic hormone life has. Outside of leptin, no neurohormone is more important to life remaining metastable.

How does the retina transmit information about light-dark exposure to the pineal gland to affect melatonin coupling? Light exposure to the retina is first relayed to the suprachiasmatic nucleus of the hypothalamus, an area of the brain well known to coordinate biological clock signals. Is there another way to transmit light from outside in that melatonin links to in some way we are not aware? Yes. *Neuropsin pathways have now been found*. I first publicly mentioned neuropsin in September of 2015. **Neuropsin light sensors send light info from our skin and eye to our hypothalamus.** Fibers from the hypothalamus descend to the spinal cord and ultimately project to the superior cervical ganglia, from which post-ganglionic neurons ascend back to the pineal gland where the mother load of melatonin awaits to clean up the mess a day creates in our brain. Thus, the pineal gland is similar to the adrenal medulla, in the sense that it transduces signals from the sympathetic nervous system into a hormonal signal.

KEY MELATONIN EFFECT

Melatonin is the key control light hormone that protects the mitochondrial DNA. You won't hear that from a food guru. I found this from the work of mitochondria pioneer Dr. Doug Wallace about ten years ago. I was stunned. This means melatonin is the photochemical gate keeper of the % of heteroplasmy in mitochondria in a cell. It controls inflammation but it also controls turnover of DHA in our

cells. This complex dance begins in the eye, at the RPE, where ocular melatonin begins its magic by using AM light to regenerate the melanopsin receptors in broad daylight using UV and IR light. They are only present together in daytime when we do not need to use the melanopsin receptors. So how does all this couple to circadian signaling?



Humans created fashion, civilization, and vanity, and this is why just being human as we where when we fell out of a vagina has become so unpopular. Can just covering our skin and our eyes have tremendous power of the diseases we get? Can this be exacerbated by modern technology and blue light at night? I think so. Oh so you still think sunglasses, clothing, and sunscreen are good modern humans? I am going to show you how these modern ideas directly destroy everything that controls the circadian coupling in every cell in your body. Alteration of light and dark cycles on our surfaces directly affects the SCN in our eye because of how light interacts on the RPE of the retina. This clock then controls and orders the peripheral clocks in our cells. Melatonin and Vitamin D3 cycles must yoke properly together to work our circadian clocks in the brain and peripheral clocks correctly. Today this situation rarely happens naturally as it should because the quantum yield of the sun has been altered by our modern civilization’s creations. Those creations, no longer allow

our surfaces to accurately sense or match the sun's frequencies or the angle of inclination, based upon our location in our latitude or altitude. This has happened because of the ubiquitous effects of nn EMF from technology and the fake blue light. They form constructive and destructive wave forms that interfere with incoming signals from our star. It also interferes with the low frequency pulsations from the Earth's magnetic field.

Consider nature's lessons carefully here. What does full spectrum sunlight in the later morning naturally destroy in our skin and circulatory system naturally in us? UVA light shows up in late AM and earlier than UVB light which shows up closer to midday. All frequencies of UV light from the sun naturally lowers adrenalin. Adrenalin is the stress hormone of the sympathetic nervous system. It is a catecholamine. Adrenaline is the main hormone secreted by the adrenal medulla in humans. The morning sun also has another major effect that is lost in many diseases. Initial sun light at sunrise unzips collagen in cells to allow water to flow between glial cells and neurons to stretch the space to wake us up. This water flow has to be controlled. In seizure disorders and MS cases this control is altered because melatonin cycles no longer exert control of the human LOX enzymes that control eicanosoids that control the aquaporin 4 water gates in the brain and retina. Control of water allows us to slow light down to create a biologic timepiece.

The morning light contains photons that are designed to re-zip collagen that was initially un-zip by the energy in light. That light release controls cortisol release that occurred several hours earlier in the dark at 4AM. This is from ELF-UV light release that is controlled by the melatonin cycles, I mentioned above. That cortisol that wakes us up by allowing water flows to occur between glial cells and neurons via the aquaporin 4 gates in the brain and eye. This is why light

stimulate seizures in many epileptics and why the most common presenting symtpom in MS is an ocular abnormality. This link directly back to altered melatonin control in the eye and the brain. The sunrise brings AM light, but this light initially has no UVA or UVB present. i It has blue, green, and red present and is quite balanced. It has more blue than any other part of the spectrum, and this is highly stimulatory to water flows and helps in alerting us and waking us up. This light also is the initial stimulus to anterior pituitary hormone release. Naturally, UVA light shows up later in the morning. This light begins to limit hormone release from the anterior pituitary gland. The time this occurs depends upon your location in latitude and in latitude if you live over 5000 ft up where UV light will show up at earlier times. This is why the world best sprinters, running backs and ski racers all come from high UV environments. They usually have the most brisk hormone release followed by a massive surge in dopamine creation to build muscle for fast twitch action. 19 of the top 20 NFL all time running backs come from subtropical environments. The worlds fastest man comes from Jamaica and the world's best marathoner runners today come from Kenya where they run close to naked and barefoot in equatorial sun. They have optimized hormone function but extremely optimized melatonin cycles in their eyes and tissues. Every AM these UV-A frequencies of light act to begin to re-zip the collagen in our skin and eyes and regenerate the melanopsin receptors in the retina.

When we are missing UV and/or IR light frequencies for any reason, the biogenic amines that are built photoelectrically and photochemically are not made properly nor are they coupled to the cycles of light. When full spectrum sunlight is absent from any surface in someone, sleep cannot be induced properly because melatonin is not being regenerated. Adenosine is a chemical signal that is generated during daylight as a by product from energy metabolism. This helps the ocular melatonin production in the eye which directly affects the

hypocretin neurons in the hypothalamus when darkness begins. Adenosine levels increase with the incident light signals; but as dusk comes incident light falls and this lowers adenosine and melatonin secretion begins. Any blue light at night blocks melatonin production by acutely raising adenosine levels. What else blocks cellular regeneration by circadian de-coupling? Lack of full spectrum solar exposure during the day is the most common reason and most overlooked issue in all of medicine these days. This is how adenosine rises and it is when melanopsin receptors are being recycled. Proper ocular melatonin cycling requires that these two frequencies (UV/IR) be present to stimulate the regeneration processes in the eye during daytime. It also requires ABSENCE of blue 400-465nm at sunset!!!! When these things are off the result always = INFLAMMATION = too many protons and/or not enough electrons at the mitochondrial cellular level = lowered melatonin levels in the eye, brain, blood plasma.

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 this situation is common should be simple to understand now; sunlight can induces sleep because the regeneration pathways that use melanopsin need daylight to regenerate while acutely raising adenosine levels to very high levels. Those levels can signal sleep cycles in humans not normally used to full spectrum light on a large portion of the body or in their eyes. When you do fall asleep, the redness of your skin really does not come from the the UV light or the thermal burn as most believe. Instead, this comes from the increased blood flow to the surface of the skin because sunlight also releases massive stores of nitric oxide (NO) from our skin. Guess what can turn off NO release? Melatonin can do this if it is present because of its effect on nitric oxide synthetase. If it is not present in sufficient amounts, too much NO is released and we absorb massive amounts of UV and IR light in our blood plasm and in our RBC’s. This can overwhelm the system and

this is what usually causes the severe burns when people fall asleep at the beach. Falling asleep at the beach is also a pretty good sign that this person live sin a light stressed environment at night or during the day. The most common causes is blue light from indoor living. Normally NO release is well controlled by melatonin in our WBC's. NO acts to bring the arterioles of the dermis layers to the surface to absorb UV light. UV light cannot penetrate skin deep at all. The same is not true for blue or red light which is deeply penetrating to human tissue. This release of NO is a natural photochemical change induced by sunlight to allow the skin to absorb the UVA and UVB light at the surface. In diseases like MS, NO is lacking in the skin and melatonin is also lacking in our WBC's and in our eyes to drive the ocular melatonin cycles. UVA light penetrates deeper than UVB light can so this is why Vitamin D levels are disrupted in people with altered melatonin cycles in the eye and brain. Poor sleep and dreaming is a sign of a light stressed human. To absorb the UV light to replace the ELF-UV all cells release to signal properly, we need the circulatory system to come from the dermis because UV light does not penetrate skin more than a millimeter. The NO engorges and vasodilates the arterioles containing the RBC's. The RBC's are filled with hemoglobin and porphyrins that absorb both UV and IR frequencies. Form meets function photoelectrically in this process. The sunburn is really an absorption of too much thermal IR energy and not the UV energy in sunlight. UV toxicity is usually related to time exposure in the sun on an acute basis in a person not normally used to being in the sun. Often they live indoors in an altered spectrum and this is the real problem. When you are out of the sun you develop anemia and lowered melanin levels and altered skin thickness. You become ill equipped to deal with sunlight. Doctors and patients often blame the problem on the sun, when in reality it is caused from chronic exposure to an alien spectrum and it is not a UV phenomena at all. It is based upon your sun callus, your skin type, your RBC status, and the amount of melatonin in our WBC's.

Did you know that WBC's make melatonin using neuropsin? Might this be why Becker found leukemia develops in areas where nnEMF is? It is well known that nnEMF lowers melatonin levels. It is also well known that blue light penetrates deep into the dermis of the skin and destroys melatonin levels locally. Would surface level blue light uncouple melatonin from neuropsin? Neuropsin responds to UVA and violet blue light. Check the links below. Might surface blue light somehow select for more immature WBC's to photochemically block how WBC's obtain their neuropsin light detector? Might neuropsin not be expressed until late into WBC's development to cause this problem? Isn't gene expression controlled by circadian biology? Does blue light somehow block peripheral circadian clocks? Yep. WBC's form deep inside of our bone marrow where other blood cells develop. Human neuropsin is closely related on a molecular basis to kallikreins. Protease inhibitor -6 (PI-6) is a potent inhibitor of the monocyte/granulocyte protease cathepsin G in WBC's, which is stored in azurophilic granules and then released into phagolysosomes or secreted during inflammation. PI-6 is present in epithelial cells, endothelial cells, myeloid cells, and platelets all of which form in bone marrow. Interesting coincidence huh? Didn't Dr. Robert O. Becker also show that the periosteum of bone works photo-electrically in several of his key experiments? Yep. So how might these facts link to sunburns in the skin and blood plasma?

Deep sunburns usually result from several factors: excessive sun; low melatonin in WBC's, anemia, dehydration, thin skin lacking melanin, 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. Today, humans bury the sun chronically and get diseases caused by inflammation. The sun is not the cause of our problems, no matter what the paradigm's current beliefs are. All modern humans however are afflicted today by altered light spectrums since we began wearing clothes and using fire at night. We began wearing

clothes 700,000 years ago. Use of fire likely goes back even further. These likely were the first ways our species affected circadian signaling in our skin and eyes.

BIOLOGY GEEKS: In vertebrates like humans, melatonin secretion is regulated by norepinephrine another catecholamine called noradrenaline (synonymous with norepinephrine). It is the main neurotransmitter of the sympathetic nervous system. This system is driven by the PVN in the hypothalamus. It is responsible for tonic and reflexive changes in cardiovascular tone. Here again we see how a nucleus linked to the eye links directly to the circulatory system. This is important for collection of light frequencies from the surface skin where the circulatory system can reach the surface when incident light hits it. Here you can see how melatonin in the eye, WBC's, and skin are coupled to the circulatory system. Norepinephrine is a cold mediated catecholamine hormone. Cooling skin temperature stimulates plasma norepinephrine release. UV light also stimulates it. When noradrenaline is released calcium is released into the cytosol of a cell. This calcium release increases the amount of ELF-UV light from fluorophore proteins in cells under stress using the IP3/DAG signaling pathways. ELF-UV and cooling both increase catecholamines and this in turn helps increase melatonin secretion in WBC's to modulate inflammation in the skin and blood plasma. Melatonin is a free radical scavenger and it is acts to alkalinize the blood plasma in the circulatory system to affect the size of the exclusion zone in blood as well as the lipoprotein particle size and phenotype in the plasma. The lower melatonin is in WBC's, the lower the EZ is in blood, and the higher triglycerides are in the blood and the one would expect higher levels of inflammation in the blood markers. This is why HS CRP, ferritin, low Vitamin D3 levels, and lowered sulfated cholesterol are all linked in inflammatory states. All are associated to problems with light and water bio-physics.

Norepinephrine elevates the intracellular cAMP concentration via beta-adrenergic receptors and activates the cAMP-dependent protein kinase A (PKA). PKA phosphorylates the penultimate enzyme, the arylalkylamine N-acetyltransferase (AANAT). On exposure to daylight, noradrenergic stimulation stops and the protein is immediately destroyed by proteasomal proteolysis. Production of melatonin is again started in the evening at the point called the dim-light melatonin onset (DLMO). This is how sun light controls this process in the stress hormones in the blood. Sunlight is capable of lowering the stress response.

It is principally blue light, around 460 to 480 nm, that suppresses melatonin, proportional to the light intensity and length of exposure. Until recent history, humans in temperate climates were exposed to few hours of (blue) daylight in the winter; their fires gave predominantly yellow light. The incandescent light bulb widely used in the twentieth century produced relatively little blue light. Kayumov et al. showed that light containing only wavelengths greater than 530 nm does not suppress melatonin in bright-light conditions. Wearing glasses that block blue light in the hours before bedtime may decrease melatonin loss. Use of blue-blocking goggles the last hours before bedtime has also been advised for people who need to adjust to an earlier bedtime, as melatonin promotes sleepiness.

SUMMARY

So you read this monster and you're thinking I am going out and taking melatonin. Not so fast. Taking melatonin orally chronically without blocking blue light can lead to eye damage. This eye damage will cause you to get all the diseases we see in the modern world. Yep……I said it and went there. This is where the foundation of modern disease is being generated, in my opinion. All exogenous doses produce the same response. If our cells make it…….you're not designed to take it. Hyperlink.

And here is more data to back up that bombshell. Hyperlink. No dose is safe with blue light. Although melatonin is present in food such as fruit, vegetables, and wheat, melatonin ingested with a normal diet does not significantly contribute to circulating levels. So why would anyone try to sell it in a pill? Money…….that is the only reason. Be smarter. Don't let marketing distinguish anything. The first casualty in marketing is always the truth. Do your own biohacks to get your own conclusions. If you use melatonin you might be destroying the only quantum computer ever made, your brain. The miracle of your brain's quantum physiology isn't that you can see the world as it is. It's that you can see the world as it isn't.

Light responses in bipolar cells are initiated by synapses with photoreceptors. The bipolar cells then transmit the signals from the photoreceptors or the horizontal cells, and pass it on to the ganglion cells directly or indirectly via amercing cells in the retina. Unlike most neurons, bipolar cells communicate via graded potentials and not action potentials. This means light frequency is on a slope. That slope is related in quantum fashion to the neurotransmitters in the brain. All NT are also quantized to light. This is why dopamine and UV light are linked. Like horizontal cells, amacrine cells work laterally also. However, horizontal cells are connected to the output of rod and cone cells. These are the main photoreceptor of the eye clock and camera. Amacrine cells affect the output from bipolar cells directly (they are inhibitory), and are therefore more specialized. The specialization comes in the form of light frequencies they respond too. Each type of amacrine cell releases one or several NT's where it connects with other cells in the brain to do what they do. This is why there are 33 types. If you understand factorial math, that means within our octave of the visible spectrum amacrine cells can handle 8,683,317,618,811,886,495,518,194,401,280,000,000 different frequencies. So when you realize that biochemistry only uses

100,000 reactions per second light frequencies can easily handle this task via your retina. A biochemist should recoil at the leverage of the power of light to control every aspect of biochemistry. Biochemists have no clue what controls enzymatic flux but now you do. “Let there light” was no bullshit story.

This is how sorting out the visible spectrum of light occurs. We operate in just one octave of 73 octaves of light total spectrum. The visible spectrum of the sun is one octave!!!! How nuts is that!!!! Within that one octave are hundreds of thousands of frequencies of light. Amacrine cells determine what frequencies we pay most attention to and link them to neurotransmitters like serotonin, dopamine, melanin, melatonin, acetylcholine, GABA, carotenoids that respond to these frequencies. They are respond in quantized fashion controlled by light alone. The rest of the frequencies we remain oblivious too in our retina because our visual sense is not attuned to them. Do you hear radio stations if the dial is not tuned to them? No……..but do those radio stations still exist? Yep. Same thing is true with your eye’s tuning frequencies. If humans created a type of light, we ‘ain’t’ supposed to see at night can you see how this ruins EVERYTHING?

Everything we put on our bodies distorts our sensory perception because it changes mitochondrial heteroplasmy rates. That change links to our haplotypes origin of our maternal mtDNA. This alters our physical reality. I look at diseases as alternative realities now because of this blog. The senses are specific to our morphologic development and present for us to use the specific spectrum of our local environment in the best ways ways possible. When you move away from your ideal adapted environment things change. Why? Blocking one part of the spectrum alters biochemistry because it often regulates another. This is akin to having sex with your clothes on. We can do this, but nature wants us naked for

a reason. Moreover, it's probably not a good thing to consider. We need the sun signals to make order from the chaos. Mother Nature perspective is the one makes the point we should adhere to. Our surfaces are designed to decipher wave forms from the space around us; they are invisible to us unless we have our senses intact. If you take melatonin pills you destroy the circadian sense and the gears in your eye clock.

We don't need to improve sleep with drugs……..we need to innovate darkness and sleep comes naturally as a result. Big difference. The inconceivable waves of light are everywhere but your eye is only attuned to a very specific octave of the spectrum. Focus in on what matters and not on what doesn't. Nothing improves sleep like sunlight prescribed at the correct time during the day when UVA works its magic with neuropsin. How many times do you fall asleep at the beach? Now you might begin to realize why. When you embrace the sun, now, becomes most powerful aspect of your being and then it's the dark's turn to be afraid. Try not to be afraid because it shortens our time on Earth. Without the sun, our life becomes a series of fears fed by a series of chronic illnesses. When you become afraid, you become a book that no one reads. You become music that no one listens to anymore. You'll become so afraid of fears that you'll be abandoned like a movie playing in an empty theater. That is what burying the sun does to you. Dopamine and Melatonin are key gears in the biology of time.

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