

Quantum Biology 4: Metabolic Syndrome

Readers Summary:

1. How are all scientific disciplines tied together?
2. What balances scientific observation vs. evidence-based medicine?
3. How does entrance into the electron chain transport help/hurt us?
4. How can Niacin help metabolic syndrome?
5. What are the downstream effects?

We have shared a lot of information in the EMF and Quantum Biology series. Today, we are going to tie some of these concepts together to give you a picture of what the causes of metabolic syndrome might really be. This disease is now a runaway neolithic disease over the last 20 years. The fact that close to

26{a7b724a0454d92c70890dedf5ec22a026af4df067c7b55aa6009b4d34d5da3c6} of the population is obese and T2D is now no longer just a disease of obese people should begin to get scientist asking a better question of the real etiology of this condition. It really has not. They remain prisoners to old beliefs about the disease. What is clear today, when a person has the up to date current perspective of published biology, chemistry, and biology, is that all disciplines are connected deeply. Biology is the science of life, but it becomes one with physics, and the science of matter and its motion. We only seem to become aware of this situation while living our lives and becoming very observant of this synergy. Our living as sentient beings makes us the real experts on the laws of real nature. Science is based on observation of nature at its core. Evidence-based medicine is far off this target today. This may help you understand why Metabolic Syndrome is a runaway disease today. In 2012, the US government spent 270 billion dollars on treatment for this one condition. 20 years ago it was around a billion dollars. Something has radically changed environmentally and it is not the 9 billion inhabitants genes on this planet that have adapted this quickly. Their epigenetic pressures however have, and this is the source of the medical bill we get. You need to begin to question much of what you believe about neolithic disease today. Today's blog delves into deep connections of those disciplines to help you understand what confuses your doctors today. Metabolic Syndrome is being treated via a cookbook called evidence-based medicine. Much of what we believe about this disease is based upon a house of cards.

Today's conventional wisdom advice is that people are told to eat a high carb and low-fat diet to combat metabolic syndrome by the system. They are treated with drugs to lower glucose levels and drugs to raise endogenous insulin levels, or insulin itself while living mostly inside under blue light. The numbers of diabetics just grow exponentially when we do this. This should be a sign to change course to a new approach. We might consider a ketogenic diet to tackle this disease while cutting artificial light, EMF, pesticides, and the use of omega six industrial oils. So far this has fallen on deaf ears. All mitochondrial diseases and inefficiencies are tied to alterations in calcium metabolism in mitochondria. This is precisely how EMF

acts in a biologic construct. So now that we live in an altered field [100{a7b724a0454d92c70890dedf5ec22a026af4df067c7b55aa6009b4d34d5da3c6}](#) of the time because of the modern tech boom you might see why I think the smoking gun is obvious. Biology and physics just need to get their researchers together to see the picture I am painting.

When you eat a ketogenic diet most clinicians seem to have forgotten what really happens in a human's biochemistry in their liver. We begin to force electrons to bypass complex one at the inner mitochondrial membrane during electron transport and enter complex two more consistently. FADH₂ is an integral component of complex two. You originally heard about this in the [quantum electron post](#), but what did not I tell you? Why does a ketogenic diet work in T1D, T2D, and to lose weight? When you lose water conduction ([protonicity](#)) to make energy, you begin to use ATP as a primary fuel source. The real problem arises when you no longer can make adequate ATP because of a protein conformational change which happens at cytochrome 1 called NADH. What allows this change? Might it have something to do with the hydrogen in NADH?

When this happens at the inner mitochondrial membrane you lose the ability of proper nanoscopic protein folding of cytochrome 1 and the result is constant chronic inflammation leak and a change of the "H" in NADH that depletes us of energy/information that leads to a loss of Vitamin D3 and causes a steady decline in the ability to make endogenous hormones from the PPP. The reason is the PPP needs NADPH to operate. The "H" in NADPH is critical to the operation of the process.

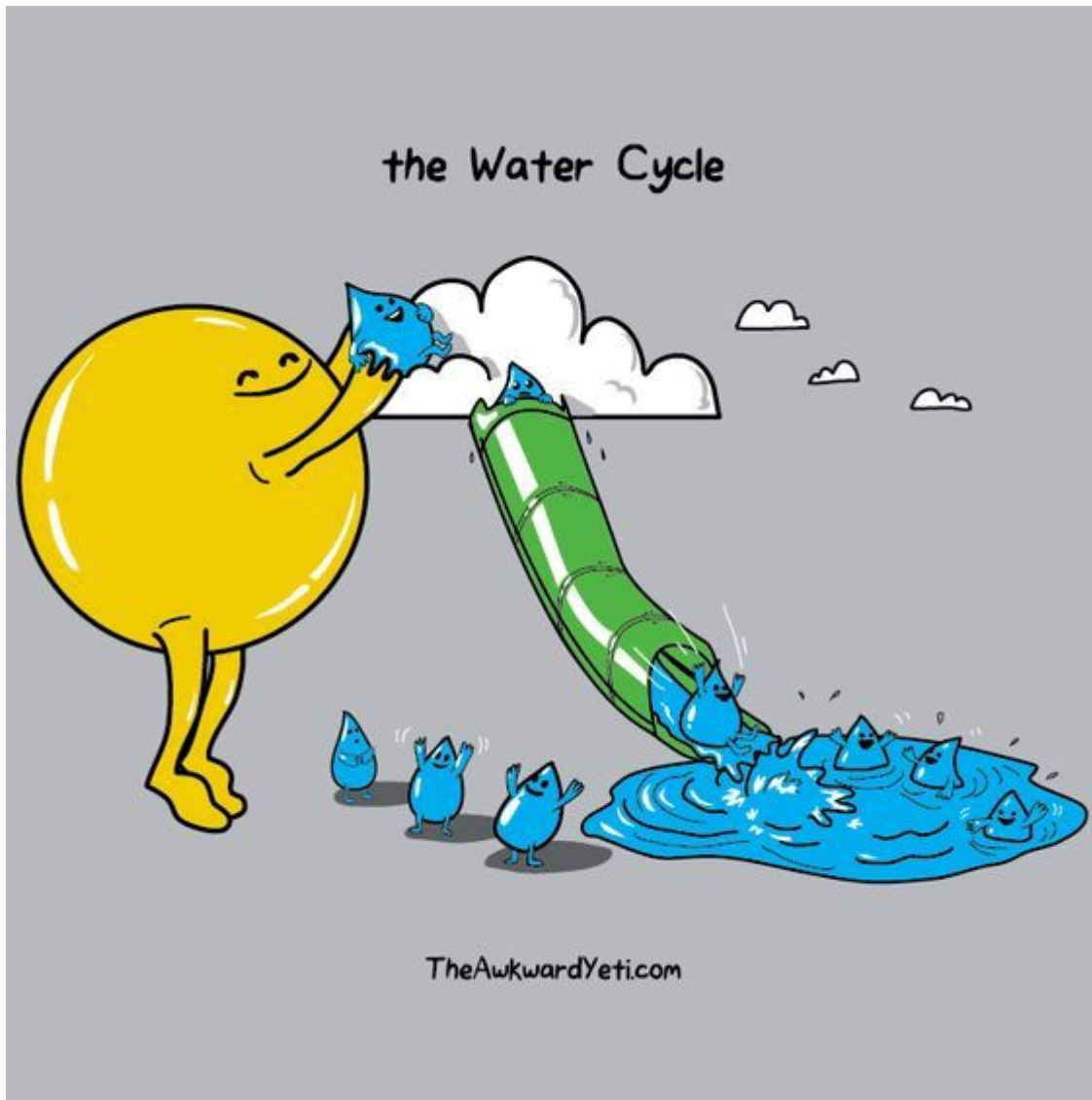
Geeks: the ketogenic diet with glucose depletion— When this is the dietary template over a period of a few weeks it induces nuclear gene transcription via mitochondrial signaling. When mitochondrial signaling is altered we get something called mitochondrial heteroplasmy. Mitochondrial heteroplasmy refers to the cellular phenotype where there exists a population of normal mitochondria called wild-type in many papers and books, and mutated mitochondria called mtDNA mutations. This scenario occurs in several inherited genetic disorders but also occurs with accelerated aging due to poor epigenetics. Accelerating aging is seen in T2D, Metabolic Syndrome, Obesity, and PCOS. This is why these diseases all share some degree of mitochondrial damage. In in vitro studies of artificially created cybrid cells with mitochondrial heteroplasmy, a [glucose-depleted \(not glucose free\), bathed in a ketogenic cell media, promoted an increase in wild-type mitochondria and decreased the proportion of mutant mitochondria.](#) **This means a ketogenic diet can potentially reverse a disease process at the mitochondrial level if mitochondrial signaling is altered in the pathophysiology of the disease. That signaling need the sun to operate. The sun restores autophagy and apoptosis in the colony of mitochondria. In Metabolic Syndrome it is the key factor.**

A ketogenic diet is one that features ketone bodies as energy sources rather than glucose and involves metabolism in the **cytoplasm** instead of in mitochondria. Why is this important? The cytoplasm is where coherent water is bound to the positive semiconductors in carbon nanotubes. This is precisely where the quantum effects of energy transduction would occur. Any

decrease in cytosolic energy levels is then directly transmitted to the mitochondria as a signal. This signal is used to drive molecular pathways in mitochondria that tell the mitochondria what is the best choice to make for the cell, and then it stimulates that pathway to completion. This is how mitochondria sculpt intracellular evolution. Some of these processes are called [mitophagy](#) and mitochondrial biogenesis. Mitophagy is autophagy of the mitochondria itself. It has its own biologic pathway distinct from cellular autophagy with its own signaling. [Mitophagy or mitochondrial biogenesis](#) are closely coupled to avoid mitochondrial depletion (apoptosis) in tissues and cause cell death. **This coupling is maintained by melatonin levels.** If it did not operate this way organs would fail early because mitochondria would undergo chronic apoptosis. When this does fail in pancreatic beta cells, we see the development of T2D. Metabolic syndrome is also associated with high blood pressure. The reason this occurs is that metabolic syndrome patients rarely get any UVA sunlight to make nitric oxide in their skin to release it to lower their blood pressure. This is why diabetics have thick skin and so many dermatologic changes. Excessive autophagy without mitochondrial biogenesis overstresses the remaining "good" mitochondria, triggering cell suicide or apoptosis because of a lack of melatonin. Clearing our bad mitochondrial engines is a healthy thing for the cell and the organ involved. When this process slows or fails we get chronic organ failure. This is how heart failure and kidney failure develop in Metabolic Syndrome. Glucose inhibitors such as 2-deoxyglucose which prevent glycolysis and are essential "**calorie restriction mimetics**" have also been shown to select for healthy mitochondria and to get rid of damaged mitochondria. What else is a calorie restriction signal? The cold environment is very helpful for diabetics. Since most diabetics do not go out enough and spend most of their time inside close to the power grid this rarely happens. This is how the diet and the environment were effectively used to transduce the outside signals of the world to the internal signals in the cell in the mitochondria to drive intracellular decision making. The ketogenic diet is a dietary induction of selective mutant mitophagy. Ketogenic diets are designed to naturally occur in cold climates for a deep evolutionary purpose. They help reverse the damage the mitochondria face in the spring and summer months when carbohydrates are supposed to fueling electron chain transport via the diet. These foods are designed to cause more leakiness at cytochrome one over 4-6 months. In winter, life is looking to repair the damage with ketosis. When you never face a true winter in life, well you get Metabolic Syndrome.

The paleosphere calls people with this defect metabolic deranged. I do not. I call it a mitochondrial inefficiency because of uncoupled autophagy/apoptosis cycles. Circadian biology controls this process. It means diabetics have to rely on ATP from food electrons/protons to maintain ECT flow because they have lost their main ability to generate piezoelectric currents in collagen and water in their bodies. Normally humans can maintain ATP production without food using UVA and IRA light from the sun via the skin and eyes to lower food intake to keep motions on the ECT moving. This throws off the nanoscopic precision required for quantum tunneling that occurs in the inner mitochondrial membrane and this causes increased ROS and metabolic syndrome. Vitamin B3 can really help for those with minor protein folding issues at cytochrome 1 early on in this disease process.

Niacin: This is also why niacin works in these diseases because it is a **ketone mimic drug**. Why? Niacin also is known as vitamin B3, nicotinic acid and vitamin PP is an organic compound with the formula $C_6H_5NO_2$ and one of the 40 to 80 essential human nutrients. In fact, when niacin deficiency is present it is one of 5 vitamins that causes a pandemic disease condition called pellagra. But it can be used to bypass a “broken” cytochrome 1 when misfolding (due to a chronic ATP deficiency, [think EMF 7](#)). Why is understanding this hardcore biochemistry important? It underpins why understanding precision that QED requires is paramount in semiconduction. Without the proper protein folding, you lose quantum tunneling of electrons and you lose the ability slowly to remain an omnivore. This is where QED takes us. Vitamin B₃ can help activate PPP when they are broken for some reason when we are ketotic from a dietary standpoint using vast amounts of coherent water as I laid out in the [quantum electron blog post](#). This occurs because it helps restore anions to the TCA and urea cycle. The PPP is responsible for making the chemical rings found in all human hormones using NADPH. The hydrogen in NADPH must come from the anions in the TCA cycle. They become unstable as well when *intracellular water* is lost because cholesterol is a polar molecule as well and does well in a lipid and water environment to act. Its action at receptor sites is critically important in how the hormones work in the nucleus to turn on and off our epigenome.



People continually forget sun exposure makes water in the matrix and cytosol of cells because of the order and information processing in mitochondria

Yes, people, niacin is a precursor to NAD^+/NADH (cytochrome 1) and $\text{NADP}^+/\text{NADPH}$ (the magic of the PPP), which play essential metabolic roles in all living cells as I showed in [EMF 4](#). The key is where the hydrogen comes from and what isoform it is in the matrix. Niacin cannot be directly converted to nicotinamide, but both compounds could be converted to NAD and NADP in vivo using matrix derived hydrogen and *this information* is very valuable when you have liver leptin resistance, metabolic syndrome, or epilepsy. It also is important for those with liver viral diseases like hepatitis B, C, D, and E. These patients also tend to well on niacin and a higher-saturated fat diet for these reasons. This type of fat helps make matrix water.

When cytochrome 1 has misfolded electron transport proteins (quinolone issue of CoEnQ10) you need to bypass it
100{a7b724a0454d92c70890dedf5ec22a026af4df067c7b55aa6009b4d34d5da3c6} of the time by eating fats, predominately, because if you don't, your mitochondria begin to make massive amounts of ROS that overwhelm the cell, thereby

lowering your vitamin D levels as well as most other hormones. This is due to the pregnenolone steal syndrome we spoke about in the [Hormone 101 blog post](#), and as time elapses, eventually shortens its telomeres to cause cellular signaling problems that lead to either senescence or cancer. The reason why this occurs is more interesting.

NAD⁺ builds up in relation to NADH levels on a per unit basis biochemically. Increasing the NAD⁺ in comparison to NADH changes a most of the mechanistic reductive biochemistry that occurs downstream. This occurs by signaling in the protein kinase B pathways of cells. Why is that a big deal you ask? The AKT pathway is also known as protein kinase B pathway (PKB). It is a serine/threonine-specific protein kinase that plays a key role in multiple cellular processes such as glucose metabolism, apoptosis, cell proliferation, transcription and cell migration. It is a major player in all things tied to metabolic syndrome via melatonin controls

Akt1 is involved in cellular survival pathways, by inhibiting apoptotic processes. Akt1 is also able to induce protein synthesis pathways and is, therefore, a key signaling protein in the cellular pathways that lead to skeletal muscle hypertrophy, and general tissue growth. Since it can block apoptosis, and thereby promote cell survival, Akt1 has been implicated as a major factor in many types of cancer. Akt (now also called Akt1) was originally identified as the oncogene in the transforming retrovirus, AKT8. In a mouse which is null for Akt1 but normal for Akt2, glucose homeostasis is unperturbed, but the animals are smaller, consistent with a role for Akt1 in growth. In contrast, mice which do not have Akt2, but have normal Akt1, have mild growth deficiency and display a diabetic phenotype (insulin resistance/T2D), again consistent with the idea that Akt2 is more specific for the insulin receptor signaling pathway. The role of Akt3 is less clear, though it appears to be predominantly expressed in the brain. It has been reported that mice lacking Akt3 have small brains. This is what many believe causes brain shrinkage in all the neurodegenerative disorders that are linked to cytochrome 1 issues. This also happens to be why every neurodegenerative disease describes to date has an element of protein folding mishaps tied to its pathophysiology. PCOS is tied to this early on in its evolution before the more chronic diseases like metabolic syndrome develop fully. It is a gateway disease for what is coming in future years.

Akt2 is an important signaling molecule in the insulin signaling pathway. It is required to induce glucose transport in cells. Here is where metabolic syndrome eventually leads to severe T2D.

The follow through for the biochemistry geeks? AKT1 is involved in the PI3K/AKT/mTOR pathway and other signaling pathways. These all cause massive changes in cell cycle signaling and in cellular communication. mTOR is a huge factor in longevity and in cancer progression via the dysregulation of the p 53 gene. Inflammation or ROS must be present simultaneously for this to happen. If inflammation is not present mTOR activation increases protein synthesis and promotes survival. Why? Collagen is the P semiconductor of life. It is made from protein. High dietary protein won't harm or kill you unless excessive inflammation, RNS, or ROS from cytochrome 1 misfolding is also present. This changes electron flow in mitochondria. That ruins

autophagy programming. This ruins information processing in the mitochondria. Context and details matter in quantum biology and too many scientists, researchers, and doctors remain unaware of these pitfalls. I spoke about this very issue in Cold Thermogenesis 6 blog post 18 months ago.

Let's talk about chronic fatigue patients. Do they suffer from the same condition but on a more subacute level? Yes, they do. Patients with ME/CFS patients usually have dramatic increases than normal levels of *ventricular lactate* in the CSF, and

36{a7b724a0454d92c70890dedf5ec22a026af4df067c7b55aa6009b4d34d5da3c6} less GSH in the cortical regions of the brain. The cortex is bathed in surface CSF that usually is not able to "proton superconduct" for many reasons. Fluoride is a dielectric blocker in CSF. The industrial omega 6 PUFA fats in the surface of their cell membranes in their cortical neurons are the most common reasons for avalanche collapse of proton superconduction in "CSF water" by displacing iodine and causing an upregulation of lactate production to use as a fuel when ketone is not present for energy production to make maximum ATP.

When we have an energy inefficiency in the brain we get neurodegenerative disorders soon following. This is why Alzheimer's disease and metabolic syndrome are bedfellows. Today we call diabetes type 3 diabetes. It is also why so many fibromyalgias and chronic fatigue patients also have similar phenotypes.

Neurons in the CNS and PNS love lactate, especially when they are suboptimal in ATP production or suffering from a neurodegenerative disease. Neurons become inefficient because of poor mitochondrial function due to lowered ATP production. This poor ATP recycling is the signal to the neurons and the brain mitochondria that they are trying to increase proton superconduction in CSF (density of CSF water) to improve $CMRO_2$ and the current in the brain's other semiconductor, water. The higher $CMRO_2$ is the less the brain relies on ATP and the more it uses water superconduction. It is like a high octane fuel for neurons. Patients with ME/CFS are very energy inefficient and subsequently, they have high lactate levels in their CSF because they ARE energy inefficient and poor ATP recyclers. The lactate supply in the brain is made by up-regulation of MCT1 which is called monocarboxylic acid transporter 1. People with ME/CFS generally generate a lot of superoxide in the brain as the cause of their disease and not being able to make a lot of ATP and this is why they eat way too many carbs too fast recycle ATP. This never allows them to open their proteins fully to open the maximum amounts of water binding sites to foster water/proton superconduction.

Neurons in the brain try to avoid superoxide generation at all costs and neurons usually attempt to increase lactate production as a consequence to offset superoxide generation to use lactate as a secondary fuel source to ketones. Most people with ME/CFS do not use ketones for electrons along their inner mitochondrial membrane for ETC; instead, they use glucose for fuels because they are no longer metabolically flexible due to the defects of protein folding at cytochrome 1 (NADH), because of what I wrote about in [EMF 4](#). I believe this effect is due to anion depletion of the TCA by protons isoforms. They use too much complex one because of glucose, glutamate fuels are needed to quickly make ATP and they try to run their mitochondria on NADH

complex one. It can lead to misfolding if they can not make enough ATP to complete the task. Oxygen and ROS are kept low in this clinical scenario. If oxygen was raised oncogenesis would be more likely. When this fails you activate apoptosis pathway and some of the things I spoke about in the recent [webinar](#) I did.

When you have metabolic syndrome you really want to access ECT at FADH₂, or complex two in electron chain transport on the inner mitochondrial membrane instead using FFA or the ketogenic diet to bypass this issue in these diseases. When one fuels their diet with glucose/carbs you are increasing ROS and superoxide as a consequence. The more you use these fuels the more you increase ROS and superoxide and limit your ability to make ATP. I covered this in big detail in [EMF 4](#).

Electron-dense foodstuffs supply the best possible neuronal FADH₂: NADH (F/N) ratio of 0.2 to minimize ROS, ELF-UV light release, and superoxide generation. Moreover, this is why this pathway of electrons gives one FADH₂ for 5 NADHs when you do this to minimize superoxide maximally. I talked about this very issue in the [quantum electron blog post](#) over two years ago. **The more NADH you make the higher lactate will be made to offset the glucose in the brain.** Lactate actually allows the mitochondria to make more matrix water to recycle hydrogen atoms to NADH and NADPH. This is why we need to use lactated Ringer's solution for people with mitochondrial diseases. This shows you why the brain really **does not like nor need a lot of glucose** as a first line fuel contrary to what most believe or regurgitate. Ketones and lactate support fat metabolism optimally in neuronal use from foods/fuels to protect the brain.

QUANTUM GEEKS: When you think back to Quantum biology one I told you a sphere within a sphere creates an electrical tension or a difference..... a mitochondrion within a cell is also a sphere within a sphere. The Earth and the ionosphere are also a sphere within a sphere. On Earth, this relationship creates the Schumann resonance frequency. This frequency of the Earth matches alpha wave frequency in the brain. This is no coincidence. In cells, the proper yoking of the alpha waves to circadian rhythms allows for the proper oscillation frequency that quantum tunneling requires on the inner mitochondrial membrane. This implies the ELF of the environment sets the tone for how biochemistry operates. The field the cell finds itself in is how the genome will be expressed. Darwin said in 1859, that the conditions of existence are far more important than natural selection. Today, conditions of existence have been renamed epigenetics. The environment's ELF controls epigenetics. This insight maybe your surprise corollary how the macrocosm of the environment meets the microcosm of the cellular milieu. Our mitochondria are designed to resonate with the Schumann frequency and the brains alpha's rhythms. In fact, every spherical organelle suspended in the cell cytosol's water and collagen cyto-architecture filled with water. The ELF field a cell finds itself in can structure the water in the cytosol by changing bonding strength and bonding angles in water's hydrogen bonding network. This improves or ruins energy and information transfers inside the cytosol. If it is ruined the cell has to rely more on ATP and less on semiconduction. This creates more ROS. These factors are all determined by your photoperiod and

circadian rhythms. These frequencies are in the ELF band of the electromagnetic spectrum of energy to control the frequency and energy oscillations along the ECT that allow for proper tunneling in your ECT on your inner mitochondrial membrane. They yoke quantum time with neurologic or biologic time in the brain or cells in order for proper cellular metabolism and order and limit entropy or stress. This is precisely what a zero entropy systems is.

PORPHYRINS Hb ARE ARTERIAL SENSORS FOR UV-A, UV-B and IR-A SUNLIGHT

DAY = WAVES NIGHT = NONE = 5 PHOTONS

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HOW IMPORTANT IS CURRENT OF FLOW TO CELLULAR ENERGY/INFO PRODUCTION?

The more energy inefficient we are the more reverse flow we get in ECT on the inner mitochondrial membrane and we get electron flow that results in backward flow and leakiness on cytochrome one. Quantum tunneling on the inner mitochondrial membrane requires the nanoscopic precision of the cytochromes and precision of the aromatic amino acids in the proteins that make them up.....why? **Increasing the distance between two coupled carriers by 1.7 Å slows the rate of electron transfer 10-fold.** The flow (current) causes a sharp change in the NAD⁺ and NADH ratio and results in superoxide formation. How do you ask? When we eat food it is broken down to electrons from its substrate foodstuffs. The electron density of these foods is determined by the power density of light in the seasons they normally grow in around the globe. For example, foods low in electron density, grow in the summertime because the power of the sun in our environment is offset by the lower density of electrons in food. This means in summer our matrix makes less water than it

does in winter. It works like this because we do not need as much water because more light is present to be buried in the exclusion zone of water in summer months to power life.

It is also why fruits have a high water content to offset the lower electron density and higher fructose and glucose levels. The higher the sugar level the more superoxide is made to cause ROS. This is called energy/information balance and why evolution selected for the formation of leptin. Leptin is a "photoelectric effect accountant" for the brain. It also allows the brain to know the information in the sunlight that programs food electrons and moves food protons. When foods with lots of fructose or glucose are eaten a lot of NADH is made which fuels generation of superoxide. NADH is an electron transfer protein that requires excellent movement of substrates in the TCA cycle to give the H to NAD^+ . When there is a lot of NADH is around and not enough solar stimulus in the eye or skin, we tend to eat more foods to make up the deficit, the electrons begin to access other points to the electron transport chain's CoEnQ10 coupling mechanism. These cytochromes are called electron transporting proteins that work via quantum tunneling mechanisms. Their names are flavoprotein dehydrogenase, mtG3Pdehydrogease, NADPH dehydrogenase for example.

By transferring electrons from the food to the cytochrome proteins it chemically reduces the proteins that CoEnQ10 couple too. Electrons delivered to the ECT from other cytochromes than cytochrome 1 promotes excessive reverse electron flow through complex I. This is what excessive fat or ketones can do. As a result superoxide generation begins and signals the cell to become insulin resistant because so many electrons are present. A person who employs a ketogenic diet does not get metabolic syndrome if they get decent sun exposure because they quickly become able to uncouple ECT and direct the excess electrons due to temperature changes. A person who eats a lot of fat but also eats a lot of foods that use cytochrome one, like glucose and fructose becomes leptin resistant. People who are Leptin resistant can not uncouple well. If a person can not uncouple, ECT metabolic syndrome is the result. To uncouple ECT requires UCP3, a high T3 level, a decent Vitamin A level, and leptin sensitivity all to be present in neurologic and quantum time. Vitamin A biology is linked to all opsin photoreception in humans. So if one is in any blue light it is physically impossible for this to happen.

Blue light also destroys melatonin levels and this uncouples autophagy from apoptosis as well. When you are leptin resistant, as I mentioned in the [Oprah blog post](#) if you can't uncouple and you develop metabolic syndrome and its associated conditions related to mitochondrial dysfunction.

In ME/CFS patients, they also constantly and chronically activate the PI3K/AKT/mTOR pathways like a diabetic does, but they do it for different metabolic reasons. This is why the disease phenotype presents differently. Under the hood, however, the quantum biochemistry is the same but the biophysical levers that control them are absent. It is caused by poor energy utilization at the mitochondrial level in neurons in the CNS and PNS to cause the symptoms of fibromyalgia. Now you can see why when this pathway is activated many bad things can happen that are common to T1D, T2D, most cases of leptin resistance with elevations in HS CRP.

These steps are just bus stops on the way to either final pathway; and we call them neolithic diseases or diseases of aging. This gives you an insight into the etiology of the “quantum causes” and effects seen in metabolic syndrome early at the mitochondrial level. All low energy state diseases alter quantum tunneling of electrons in some fashion to cause the disease they do. Life and death is a dance of energy utilization along our inner mitochondrial membrane and on our water molecules to transfer energy for life well.

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