

ORGANIZATIONAL STRUCTURAL FAILURE #6: THE MITOCHONDRIAL Rx

READERS SUMMARY:

HOW DO MITOCHONDRIA REALLY WORK?

The funny thing about mitochondria is that everyone seems to know they are not working well in most modern diseases, but only a few know how mitochondria really work. When most allopathic and alternative health practitioners talk about food, they talk about calories, carbohydrates, proteins, or lipids. You know what is ironic about these discussions?

Open up any biochemistry book and see if there is carbohydrate, protein, or lipid transporter on the inner mitochondrial membrane. There is not. But there is this thing called the "*electron transport chain*". Why is it that nobody in allopathic medicine or alternative medicine talks about electrons? All foods are broken down into electrons. This means that the input to our mitochondria are electrons.

Gary Taubes quoted Hans Krebs in *Good Calories Bad Calories* by saying, 'All three major constituents of food supply carbon atoms'. Most biochemists, clinicians, and scientists believe that metabolism is all about the carbon cycle. I think most of you realize by now that I do not agree with this. It is all about electrons. This is why input to the mitochondrion is called ETC. The second reason is that Gilbert Ling's work showed us it is not about the amount of ATP made or about carbon recycling, but about the charges added or subtracted to the backbone of the proteins. Ling showed us 60 years ago

that electrons and electrostatic charges change the superstructure around carbon atoms. The superstructure is what we call proteins. These charges can elastically deform and compliantly redesign carbon back bones to lead to innovative changes. We call these changes evolution.

When you have this fundamental insight of how electrons can alter proteins and mitochondria, your perspective has to change. It is not what happens to you that matters most, rather it is how you respond to the stressor. Adding electrons to your proteins makes them more elastic to life's stressors. Protons stiffen your response to environmental pressures. This leads to a less compliant design in our proteins' conformations. We become less adaptable to our environment which leads to cellular chaos. Medical schools and PhD programs are full of students who are good at adopting the given "wisdom" of their teacher, but not necessarily good at free thinking and questioning. This is because they have learned to follow precepts handed to them from presumed authorities. This is why so few really understand how a mitochondria works at a fundamental level. There are many misconceptions about mitochondria and ETC. What many allopathic and alternative practitioners often call detox/herxheimer reactions are actually the movements to a more healthy template from an illness platform. The alternative health movement often causes those misconceptions.

Recent findings indicate that apoptosis *does not even require* mitochondrial ATP synthesis, but it does need a functional electron transport chain (Jia et al., 1997a). ATP is not a main driver for mitophagy based upon the public literature, but few seem to realize it. Here is a bigger shocker. The release of cytochrome c from the mitochondrial intermembrane space, which is a key step of apoptosis, is not accompanied by $m\Delta\psi$ (delta psi) changes (Kluck et al., 1997). This opens us up to a new understanding of how mitochondria might really function. If it is not electrons that alter mitochondrial function, could it be light that does? After all, electrons

do carry the energy in photons to our mitochondrion via ETC.

Let us consider the key working membrane in a mitochondrion.

The inner mitochondrial membrane is where the action of life happens. It is covered by some unusual proteins. [Mitofilin proteins](#) are crucial organizers of mitochondrial architecture. They are located in the inner mitochondrial membrane and interact with several protein complexes of the outer membrane, thereby generating contact sites between the two membrane systems of mitochondria. Here you can see how proteins undergo elastic changes as electrons are added and subtracted to its structure. When electrons are abundant, the mitofilin works to maintain ETC. When it is not, the result is autophagy or apoptosis. Within the inner membrane, mitofilins are part of hetero-oligomeric protein complexes that have been termed as the mitochondrial inner membrane organizing system (MINOS). MINOS integrity is required for the maintenance of the characteristic morphology of the inner mitochondrial membrane where ETC occurs. The inner boundary region is closely apposed to the outer membrane and cristae membranes, which form large tubular invaginations that protrude into the mitochondrial matrix and harbor the enzyme complexes of the oxidative phosphorylation machinery. MINOS deficiency comes along with a loss of crista junction structures and the detachment of cristae from the inner boundary membrane. MINOS has been conserved in evolution from unicellular eukaryotes to humans.

Alterations of MINOS subunits are associated with multiple pathological conditions.

Humans are designed to eat an electron dense diet because they have a shortened gut and an expanded brain that steepens their energy needs while restricting their sleep needs to 7.5- 8.5 hours. All of these features are tied to how well we can access autophagy. This implies that humans must have evolved around an environment providing a constant source of electron density from their environment. [Humans who eat like Gazelle's do](#), or lose electrons to their environment faster than they

collect them, are now known as people with diabetics, fibromyalgia, MS, sleep apnea, and chronic pain. Let us see why.

It turns out that how water interacts with MINOS is hugely important and not well known. Water is an energy converter, or a liquid nano-machine that takes all radiant energy and changes it to other forms of energy that a mitochondria can use and distribute over a cell. Water surrounds MINOS wherever it is found in us. [Photosynthesis](#) and oxidative phosphorylation in mitochondrion have some major things in common. Water is one of them. To your detriment, modern science still fails to realize the importance of water in photosynthesis and in mitochondria. Plants are capable of taking sunlight and using non living pigments and chemicals to make simple chemical compounds and elements needed to power life. Here we have an example of how "an EMF" brings life to life. Mitochondrion use the Vitamin A and D cycle in your brain to do the same thing using light.

EZ charge separation is the generic first step of both pathways. In fact, the goal of both energy generation pathways is to split water. In photosynthesis, this happens due to light absorbing chromophores that lie adjacent to water. To synthesize one molecule of glucose by photosynthesis, 24 electrons must be removed from water molecules. These electrons are held by the redox potential of oxygen (+0.82V). They are pumped uphill to carbon atoms that are partially reduced to a carbohydrate with a redox potential of -0.42V. The potential energy difference is 1.24 Volts. This change in free energy is in the positive direction. The result of this energy transfer creates 2870.2 kJoules of energy. This is an astounding amount of energy capture when you understand the quantum dance of the sun on water.

A mitochondria's outer membrane is hydrophilic and lies next to a water hydration shell. In both cases, light induces water-molecule splitting. You learned about chromophores in

the brain in [OSF 4](#) and [OSF 5](#). You should also recall that the brain has more mitochondria in it per unit mass than any other tissue in your body. This tells you *light is ultimately* tied to mitochondrial function at a fundamental level. This is a radical idea today. This blog will show you why it should be obvious to anyone who understands the physics of the water and the photoelectric effect. Chromophores are all hydrophilic in both animals and plants. This should invoke you to think about Gerald Pollack's work. Water's EZ has the classic 270 nm absorption peak for light. Plant chromophores also have a large 280nm absorption peak. These are both in the UV range. Infra red radiation is the power used to charge separate water into a negative and positive zone. The EZ is the negative part. The hydronium layers (protons) are the positive part. The negative EZ acts very much like a n type semiconductor. Recall that the EZ is how water is split naturally into its charges when water abuts a hydrophilic substance. Proteins are made hydrophilic *by the addition* of electrons.

The input to mitochondria are through electrons via ETC. There is no other way to get into the mitochondrial matrix. The output or the signaling parts of mitochondria are all protons. When we are missing one electron from the process of ox/phos, we get production of the superoxide ROS ion. This is a signaling molecule that increases the mass of mitochondria by making it more leaky at its cell membrane. Here is where the MINOS system is elastically deformed to begin mitochondrial recycling or [suicide](#) (autophagy or [apoptosis](#), respectively). This increase in mass stimulates the two change programs in mitochondria called autophagy and [apoptosis](#). Mitochondria swell when calcium is effluxed from a cell and phosphate builds up outside the mitochondria. In the swollen mitochondria, shrinkage may be induced by adding Mg^{2+} ions in the absence of exogenous ATP. Mg^{2+} does not work well in the absence of water. Dehydration is critical to mitochondrial function. I covered this in the [EMF Rx blog](#).

I cannot emphasize this enough. Metabolic rates decline tremendously when people are dehydrated for any reason. In our modern world, the two most common reasons for dehydration are non native EMF and fluoridation/bromination of our water.

This shrinkage due to water and Mg^{2+} has been determined by spectrophotometric, as well as by water, content and packed volume measurements. [Insulin resistance](#) eventually lowers the number of mitochondria we have as a protective function and this [drops NAD+ at cytochrome 1](#). When this occurs it means mitochondria no longer have a good handle on normal circadian cycles.

The output of mitochondria is a solid stream of protons that carry a positive charge. You should recall that all inflammation is also positively charged in life. The redox levels in mitochondrion are like a scales of justice. When protons are more prominent, the redox potential is poor. When we have more electrons in the system, the redox potential is excellent. The more electrons we have, the more potential and kinetic energy is stored in our proteins and water hydration shell to allow us to deal with the stressors of the environment. The human species is designed to collect electrons in excess. Think of the redox potential as a giant sink of electrons and protons. We need more negatively charged electrons to run our mitochondria and proteins well. This is our redox sink or bank account.

When mitochondria lose electrons to the environment for any reason at all, mitochondrial signaling to the nucleus is altered. You may not know this, but mitochondrial density is greatest in the subcellular region within 8 – 10 Angstroms of the nucleus. Moreover, not all mitochondrion are created equally. Mitochondrion closer to the nucleus have [far different functions](#) than ones located closer to the cell membrane. This means that these mitochondria act as a main sensory organ for the nucleus and cell membrane. It translates signals from the environment in this way via

integrins and two key proteins called YAP/TAZ. When they swell, they change the epigenetic expression of the cell. When they do not swell, they also change the epigenetic expression within the cell. This is the first “knob” in how mitochondria affect nuclear expression of RNA and DNA. There is a gradient system in mitochondria to tell the nucleus how many or how few electrons are present.

A loss of one electron is very normal for the mitochondria and nucleus. It results in superoxide production. If we lose two electrons in our mitochondria, we develop more swelling and the signaling molecule is hydrogen peroxide (H_2O_2). In order to control these two positively charged signaling molecules, we evolved an enzyme to counter act and control their effect.

This enzyme is called superoxide dismutase 1. This enzyme uses manganese as its transition metal to control the positively charged molecules. The problem for this system arises with the technology of our modern world. We have created 2.5 billion times the amount of electromagnetic radiation in our atmosphere than we had at the genesis of our species. This causes us to lose a lot of electrons to the environment, producing a lot more positively charged protons.

Our modern world is making a ton of non native EMF in our ionosphere. The analogy I would give you is to think of a big flower pot with a flower in it. Today, most healthcare practitioners push supplements to offset the loss of nutrient density in modern foods. But when you consider that the modern ionosphere has so much EMF energy, it is akin to placing 19 holes in the bottom your flower pot and allowing the water to drain freely. If the water cannot be contained around your roots, it becomes clear that, no matter what you put into your flower pot, you are losing way more to your environment. When this happens, your flower won't and can't grow. You need light and water to make carbohydrates if you are plant. We have the same problem in our mitochondrion. This is exactly what we are facing today.

When electrons are being stolen at higher rates, a new type of ROS is formed. When we lose 3 or more electrons from our mitochondria, we make the hydroxyl free radical. This is called a Fenton reaction. Why is this a problem for us? We have no enzyme that can contain this reaction. The hydroxyl free radical irreversibly damages membranes, organelles, mitochondria, and DNA in a cell. This means we have to constantly recycle these cellular parts to maintain our cells' integrity or limit its entropy. This means we have to use more energy to remain metastable. This also steals electrons from the brain and immune system when it goes on chronically because both systems are loaded with electron starving mitochondrion. The hydroxyl free radical causes a dramatic rise in entropy and increases the thermodynamic cost of life.

It has been known that oxidative DNA damage also compromises telomere integrity. Remember that telomeres are on the caps of chromosomes in our nucleus. Here you can see why epigenetic expression is altered. All DNA and RNA is also surrounded by water hydration shells. When protons rise, positive charges predominate and temperatures in water rise. Warmer water has a higher mass which means it can carry fewer electrons and less energy as a result. This has massive implications for our cells and our nucleic acids. Thermal tolerances are costly to life. The reason for this is that thermal tolerance costs us electrons in our mitochondria. It also causes protons to build up rapidly, increasing the thermodynamic costs of life because we are losing electrons to our environment. The thermodynamic cost is in a loss of complexity because we lose electrons. **Mitochondria are designed to make massive EZ's from the charge separation of water. That is really how they generate energy of life.**

In [OSF 1](#), I showed you these results in the loss of diversity in the human gut microbiome. When this happens, the human gut loses its diversity and number of species. The gut microbiome is designed to **consume protons** and *reserve electrons* to our

eukaryotic system that uses mitochondria. The gut microbiome is designed to work in an environment where no oxygen is present. This is why they use protons for energy generation, not electrons. Oxygen and electrons are coupled in nature and build complexity because of mass equivalence. Today, this relationship in the human gut is rarely maintained because of the formation of massive amounts of hydroxyl ion free radicals that ionize zonulin to make it more leaky, exposing the GALT to foods in the gut. People forget that these cells turn over every 2-3 days, making them exquisitely sensitive to daily electromagnetic signals. Every cell in our body has circadian clock genes in their nucleic acids. When your mitochondrion are lacking electrons, you can no longer tell proper circadian time in your cells which causes your mitochondrion to act differently, changing its epigenetic expression. Circadian Clock Gene regulators (CCGs) respond very quickly to electron steal syndrome. This is what alters our genome and our epigenetic expression. This happens in the gut fastest because these cells turnover the fastest. This implies that the [most electron dense, chemically reduced pathway for life](#) would allow for maximal mitochondrion function.

What increases electron flow?

1. Excellent water supply without halogen additions.
2. Avoid the use of products with bromide, like pool or hot tub cleaners. Don't use any grains in your diet
3. Rebuild the gut flora to be diverse in species and numbers. This increases electron delivery to the leptin receptor.
4. Good pine forest surroundings and being away from the city because they make massive amounts of O₂
5. Mitigate your local environment to limit non native EMF as much as possible
6. Cold Thermogenesis on a consistent basis: Spot CT or surface CT would be good, but it is not as effective as the cold water on the body. This is how you control mass, improve electron flow, and increase energy efficiency, all of which

activate beta 3 SNS to turn on irisin to convert WAT to BAT to empty it; you basically turn WAT into proton heat using CT and irisin to do so. While this happens, leptin levels fall and adiponectin levels rise, tightening the tensegrity of the adipose layer..... as this happens you get even more energy efficient because of the mass equivalence equation relationships.

7. You can increase electron flow to a body part using the Fournier effect as mentioned in the Webinar's Q & As.

8. Another one would be easy access to decent electron dense foods from the marine food as outlined in my book.

9. Strong local magnetic field under your body during sleep. Consider a Magneto sleep pad.

10. Encourage grounding as much as possible while outdoors.

11. Consider camping out on a regular basis. Go out into nature.

12. Use of red light phototherapy for recovery from exercise, even as a way to increase tissue optics.

13. Consider the use of HBOT, ozone, and TENS units.

14. The use of transcranial magnetic stimulation or neurofeedback

15. Acupuncture/Rolfing/massage therapy on a regular basis to tighten collagen network. Collagen is a N-type semiconductor. The EZ acts like an n type semiconductor as well. What does this mean? Through various techniques, like [doping](#), the semiconductor can be modified to have an excess of electrons.

When a transition metal is placed in collagen, it can increase the electron flow within it. This is called doping of the semiconductor. Copper is the most common atom used in collagen of all types to increase semiconductive flow. This is what a N-type semiconductor is. When you have a tissue made of collagen that is not working well, you have a problem in doping, gating, or dehydration and tearing within the tissue. When this occurs, you lose electrons and charge, and the collagen ionizes. When doped semiconductors are [joined to metals](#) (transition), [to different semiconductors](#), and [to the same semiconductor with different doping](#) mechanisms, the "resulting junction" often strips the electron excess or deficiency out from the semiconductor near the junction. This is why the use of modern gadgets made of silicon pulls electrons from your carbon based semiconductors.

16. Sexual activity. Face to face relations put you in an ideal situation to benefit from the magnetic field that comes from your partner's heart.

17. Meditation is one of the best ways to use the power of your CNS to direct flow to circuits that are electron poor.

18. Acro Yoga brings two humans close together to share electrons and tighten their collagen networks.

19. Use of compression shirts, socks, or gear to control mass equivalence.

20. Keeping the HbA1c below 5.0 to maximally (using ketosis with DHA) improve tissue photonics to be able to capture and use the photon's spin characteristics. [Optimal optokinetics](#) is highly dependent upon tissue glycation and sulfur levels.

Recent analysis has suggested that light exhibits extraordinary spin properties. Evanescent waves carry momentum and spin components of photons. Evanescent waves do, in fact, manifest in light-matter interactions, potentially leading to effects that are impossible to achieve and observe using normal light. For example, [evanescent waves](#) exert a transverse force and a transverse torque on small particles, where the force is dependent on the circular polarization of light in a material, but it is not dependent on the torque. Evanescent waves are a type of light wave that travel close to the surface of material objects. Their intensity decreases exponentially, rather than varying sinusoidally, from the interface where they were formed.

These waves maybe used in all collagen and water based semiconduction design. They propagate in tissues when its optical density is optimal in an [orthogonal manner to the direction of wave](#) propagation. When high levels of glycation occur, the optical density of tissues is altered and there is a loss of photons/electrons as a result. This is likely how peripheral neuropathy results.

You need to realize that DHA is the best material to catch electrons and photons from our environment. Collecting them has to be done in the perfect seasonal circadian manner because seasons yoke electrons with photon power. Highly powered photons are tied to electrons that make low levels of ATP. Catching electrons is a function of the proper topologic semiconductors in us. Assimilation of those electrons begins in the mitochondrial cytochromes. They, too, have a circadian

arrangement. This is why electrons from carbohydrates come from long, light, highly powered electrons that are funneled into cytochrome one. It is an extra step that can unload the power these electrons have because of the photonic power they contain. They have more kinetic and potential energy in them. Modern blue light is a form of non native EMF, in case you were wondering. We are only designed to see blue light in sunlight, and its power varies with the seasons we are in. I told people in [Brain gut 11](#) that humans have 48 % of their neural circuits dedicated to light. Now you might begin to realize why. These circuits link the brain to the mitochondrial proteins by way of the leptin receptor. The leptin receptor accounts for the electrons and the mitochondrial harvest of light released to the cytochrome proteins. The water micelles surrounding the mitochondria accept the photon light and distribute that power to proteins all over the body because every protein is covered by a water hydration shell. Fundamentally, water is a chameleon. It is globally elastic and locally fragile because it can act in many different forms. Water can organize itself differently when it is adjacent to different materials, allowing it to be the perfect chemical for interactions with the electromagnetic force. Sun light is part of the electromagnetic spectrum. When you have **too much blue light** and/or *too little water* within the system, it can short out. You know, this might be interesting for biology researchers when they eventually realize the benefit of a high fat and low carb diet on a fundamental quantum basis. A high fat diet is protective to shorting out the system that is blue light toxic. When you are blue light toxic, your sulfated vitamin D falls in a big way, and you will find supplementing with D3 won't allow it to rise. The brain will protect itself for as long as it can. Carbohydrates speed up our circadian clocks, shortening our telomeres and depleting stem cells. The electrons of carbs have more blue light in their photons because they grow in more powered seasons by the sun's light. So how would you slow down a fast clock??? You would use something that lowers the charge on the input to mitochondria. Charge is called the *delta psi* on the inner mitochondrial membrane. Fats lower the delta psi best of all foods because they have lower powered photons in their electrons. They just provide more

electrons. This is why they enter at cytochrome 2 and not cytochrome 1. This is why a ketogenic diet helps any short circuited mitochondrion. Pretty simple concept, no?

Carbs are a direct product of the power of the sun's light and, therefore, are the most SEASONAL OF FOODS. Carbs have a lot more blue light photons in them which is why they enter cytochrome one and not cytochrome two. We need more proteins to harvest them of their photonic power. This is why high powered UVB light makes Vitamin D. More cytochromes are used to move the electron down to the ground state while taking the photon's energy into water of the hydration shell/micelle around the mitochondria. Remember that water absorbs best at 270 nm according to Pollack's work on water chemistry. It is also why all fruit has a naturally high water content, too. This is why we make 147 ATP from beta oxidation of fats and only 36 ATP from carbohydrate metabolism. This difference is not just a calculation of ATP stoichiometry, it is a function of the electromagnetic photons during the seasons present on Earth as they change. Therefore, the electromagnetic spectrum of the sun carries information and energies of these seasonal factors directly to mitochondria.

Mitochondria and photosynthetic cells have a lot in common. Photosynthetic cells contain special pigments that absorb light energy. Different pigments respond to different wavelengths of visible light. Chlorophyll, the primary pigment used in photosynthesis, *reflects green light and **absorbs red and blue light most strongly***. We do the exact same thing using different quantum mechanisms in our mitochondrial cytochromes. In plants, photosynthesis takes place in chloroplasts, which contain the chlorophyll. Chloroplasts are surrounded by a double membrane and contain a third inner membrane, called the thylakoid membrane, that forms long folds within the organelle. In electron micrographs, thylakoid membranes look like stacks of coins, although the compartments they form are connected like a maze of chambers. Our mitochondria have the same type of architecture.

Chlorophyll A is the major pigment used in photosynthesis, but there are several types of chlorophyll and numerous other pigments that respond to light, including red, brown, and blue pigments. These other pigments may help channel light energy to chlorophyll A, or **protect the cell from photo-damage**.

Mitochondria do the same thing using physiologic insulin resistance to alter the charge on the inner mitochondrial membranes when it senses too much blue light toxicity. For example, the photosynthetic protists called dinoflagellates, which are responsible for the "red tides" that often prompt warnings against eating shellfish, contain a variety of light-sensitive pigments, including both chlorophyll and the red pigments responsible for their dramatic coloration. We don't want to reflect red light.....we want life to keep it within their optical design as I showed you in OSF 5. When we lose it, it is a bad optical sign that thermodynamics are altered unfavorably. I showed this to you in OSF 4.

RADICAL IDEA ALERT: Mitochondria make life work by solar power, just like plants do.

Humans are designed to catch electrons in their central nervous system and then deliver their energy, information, and spin through the DC current in the interfascial water below myelin and outside the axon to all parts of the body.

Eventually this winds up in the mitochondria. This is a critical point between the two major arms of metabolism. Mitochondrial cytochrome proteins are like *small optical magnetic nanomachines* for electrons and protons. They are designed to tell the difference in the energies, information, and spin of electrons and protons. Higher energy photons from the sun in summer go to cytochrome 1. Here water is turned into an EZ to power life. Lower powered photons of fall and winter go to cytochrome two. The cytochrome proteins actually have the ability to decipher the season based upon the characteristics of the electrons delivered to it. [Circadian locomotor output cycles kaput \(CLOCK\) is a nuclear transcription factor](#) that is a component of the central autoregulatory feedback loop that governs the generation of biological rhythms. This is how the cell membrane senses the seasonal electromagnetic changes. Electrons are sorted by our mitochondria to harvest their energy, information, and spin characteristics for our body and brain to use, setting our metabolism to meet environmental demands. This is why data now shows that when circadian cycles are off, exercise loses its benefit. Autophagy decreases when circadian biology is off. **This is why the Leptin Rx tells you not to exercise when you're energy inefficient. I explained all this detail in**

EMF4. Have a re-read.

Like the mice in Prusiner's lab, we emit bio-photons back to the environment when we lose energy! Fritz Popp has measured this and found it to be true. Clinicians do not seem to know this. They also do not realize how this fundamental process is linked to health and illness.

When autophagy is poor in the CNS, we expect to see low levels of DHEA = higher IL 6 levels in the brain = poor sleep = electron steal syndrome. This is why DHEA is a big clue to a clinician. When it is poor, you lose electrons in a big way from your thermodynamic system. When autophagy is poorly functioning, mitochondria begin to swell and increase their mass. This makes them less able to generate energy. This tells the cell that, since autophagy recycling is off, only mitochondrial or cell suicide programs remain. If this goes on chronically, you deplete all your stem cells and your telomeres shorten. All are tied to a chronic loss of electrons. Autophagy is how mitochondria reorganize and renew themselves to improve cellular thermodynamics.

Insufficient or poor sleep can have a lasting consequence on gene expression. This is how epigenetic switches are altered.

It turns out that RNA methylation underlies the transcription and translation feedback control loops that manage circadian signaling. So when you lose electrons, you directly alter the methylation and histone acetylation patterns everywhere in the nucleus. RNA translation and transcription is one such key place. This is where proteins are made. Autophagy is when proteins are marked for replacement, so if this process is inefficient, elder proteins and misfolded proteins predominate in tissues, causing more energy loss.

In mitochondria, all food is turned into electrons. The input to mitochondria is called electron chain transport.

A. When you lose one electron in mitochondria, superoxide generation is the result. This is physiologically OK because it is a signal that the mitochondria normally produce signals about what they should do next. Autophagy.

B. When you lose two electrons in mitochondria, H₂O₂ is generated and is a more serious signal. Superoxide, hydrogen peroxide and hydroxyl free radicals are all made from the decreasing levels EZ of water around the MINOS. We make more

of them when water is missing due to intracellular dehydration for any reason. This can be good or bad within the cell depending on when it occurs. This is when apoptosis or autophagy can be signaled.

C. When you lose three or more electrons in mitochondria, the result is the generation of a Fenton reaction. This causes the build up of hydroxyl free radicals. This is usually always a bad sign, with the exception of our immune cells. Within immune cells, we kill pathogens using the hydroxyl free radical. When this occurs outside our immune cells, it causes massive tissue damage which results in a low DHEA level in the CSF. It also leads to an *altered adrenal stress index* for cortisol and markedly abnormal melatonin cycles in the brain. Re-read the Quantum Autism blog where I first discussed Fenton reactions. It has a detailed area on this where I discuss this issue and how devastating it is in the developing neocortex. Usually the only place a hydroxyl free radical is used in biology is in macrophages that kill bacteria. This occurs within the confines of these cells, not in our tissues. All autoimmune diseases have lost control of this mechanism.

When they constantly occur in tissues due to non native EMF exposure, collagen is destroyed and unzipped from its triple helix, losing its ability to be piezoelectric. This means electron loss makes collagen floppy. This means masses increase and energy is lost. In the atmosphere, hydroxyl radicals are also produced during UV-light dissociation of H_2O_2 and likely in [Fenton chemistry](#), where trace amounts of reduced transition metals catalyze peroxide-mediated oxidations of organic compounds. The same process now happens in us. When non native EMF interacts with collagen, "dislodged transition metals" become unzipped because the hydroxyl free radicals cannot be controlled by superoxide dismutase 1. This is why unzipping proteins is a "new big problem" for Lady Evolution in our modern world. It frees the transition metal to interact with the higher powered non native EMF. What happens to us when this happens on a chronic basis? The higher energies contained within the non native EMFs are transferred to the liberated electrons in the D shell of these transition metals to cause [ionization of the metal](#). *This destroys the proper protein bending from occurring and affects how certain anions interact with proteins.* This further unzips

protein and lipid fragments in the cell, by activating YAP/TAZ, destroying the ability of the cell to function. It also creates massive protein and lipid damage that overwhelm an already poor autophagy program in our tissues. These changes collectively and cumulatively destroy your physiology by altering their optimal physio-chemistry in you. Hydroxyl free radicals cannot be cleared by SOD 1. And taking SOD 1 in our current environment is not smart either.

SOD 1 takes care of rust or oxidation in the liberated transition metals, like I mentioned above in the FENTON RXN example, so people with a monolithic understanding of cell physiology might assume taking SOD1 as a supplement is a great idea.....NOT SO FAST.

LONGER YOUTH ALERT: Fruit flies bred with a dose of SOD1, an antioxidant enzyme that breaks down free radicals, lived 40 percent longer than normal fruit flies did in a University of Guelph laboratory. Notably, the phase of life extended was youth, not old age.

The hydroxyl radical can damage virtually all types of macromolecules by energizing their molecular structures using the quanta of energy to alter its structure and function.

This is true of proteins, carbohydrates, nucleic acids (mutations), lipids (lipid peroxidation), and amino acids coded for by DNA and RNA. The hydroxyl radical has a very short *in vivo* half-life of approximately 9-10 seconds, along with a high reactivity. This makes it a very dangerous compound to any living organism's mitochondrial membranes or cell membranes. It causes you to lose electrons in massive numbers everywhere very quickly.

Unlike superoxide, which can be detoxified by superoxide dismutase, the hydroxyl radical **cannot be eliminated by an enzymatic reaction.**

Mechanisms for scavenging peroxy radicals for the protection of cellular structures include endogenous antioxidants such as melatonin and glutathione. This is why cysteine is used up quickly and it is also why melatonin cycles are way off in people's brains. It is the best single test to assess the effect of non native EMF in the brain. When you marry it with DHEA, Vitamin D, and IL-6, you get a complete idea why your zip code is far more dangerous than your genetic code.

Oxidation of any organic compound by Fenton's reagent is

rapid and **exothermic**, resulting in the oxidation of contaminants to primarily carbon dioxide and water. This is why IL-6 rises in CSF and why melatonin is destroyed in the brain. The exothermic reaction in CSF raises the amount of protons to electrons in CSF, limiting neo-cortical electron flow. What happens in a mitochondria when there is too few electrons and too many protons? **Cytochrome c** is a small **heme protein** found loosely associated with the **inner membrane** of the **mitochondrion**. Cytochrome c is a highly water soluble protein, unlike other **cytochromes**. Cytochrome c carries one electron. It is capable of undergoing **oxidation** and **reduction**, but **does not bind oxygen**. It does interact with nitrogen and sulfur. It transfers electrons between **Complexes III** (Coenzyme Q – Cyt C reductase) and **IV** (Cyt C oxidase). Cytochrome c is also involved in initiation of **apoptosis**. So when electrons are lost to the environment, apoptosis runs rampant. Cytochrome c is suspected to be the functional complex in **Low-level laser therapy**. In LLLT, red light and some near infra-red wavelengths penetrate tissue in order to increase cellular regeneration. Red light provides photons to help restore us by rescuing Cytochrome c. Cytochrome c binds to **cardiolipin** in the inner mitochondrial membrane, thus **anchoring** (tensegrity) its presence and keeping it from releasing out of the mitochondria and initiating apoptosis. The initial attraction between cardiolipin and cytochrome c is **electrostatic** due to the **extreme positive charge** on cytochrome c. The electromagnetic force is what controls charged particles. When electrons are lost, your ability to sense this force disappears. The final interaction is **hydrophobic**, where a hydrophobic tail from cardiolipin inserts itself into the **hydrophobic** portion of cytochrome c. When water and electrons are missing in the equation, **cardiolipin is protonated**. The sustained elevation in **calcium** levels precedes cyt c release from the mitochondria. In Energy and Epigenetics 4, I showed you that non native EMF effluxes calcium. The release of small amounts of cyt c leads to an interaction with the **IP3 receptor** on the **endoplasmic reticulum** (ER), causing ER calcium release. All of this is tied to the electromagnetic force. The overall increase in calcium triggers a massive release of cyt c, which

then acts in the positive feedback loop to maintain ER calcium release through the IP3Rs. This explains how the ER calcium release can reach cytotoxic levels. This release of cytochrome c, in turn, activates [caspase 9](#), a **cysteine protease**. Cysteine is where sulfur is locked up. Besides cytochrome c, extramitochondrial localization has also been observed for large numbers of other proteins, including those encoded by mitochondrial DNA.....interesting huh?

This raises the possibility about the existence of “yet *unidentified*” specific mechanisms for protein translocation from mitochondria to other cellular destinations because of a quantized mechanism. OSF 3, 4, and 5 showed you that mechanism. Once you alter their charges, the electromagnetic force can direct proteins to where they need to be by elastic deformation. Free radicals accumulate in mitochondria and in cells during aging and disease, but do not necessarily cause it. What effects do they have?

They simply alter the charge on proteins on the MINOS to alter the size of the EZ of water surrounding mitochondria. These changes are what stimulate autophagy. When you take exogenous antioxidants and your redox is low.....you can facilitate worse disease because you destroy normal signaling. In the evolutionary package we call food, the charges come balanced.....and then we use them as the quantum blueprint determined by the redox potential of the cell membrane and mitochondria. I'd also point out this gem based upon this reality: If genes matter so much, why is it that most, not all, circadian gene regulation is [post transcriptional](#)? This is a huge problem for neo Darwinians. Why is that? A post transcriptional protein is non functional for life. So how could life's stage be gene based? It is not. It is a modern belief that will cause you to subjugate your epigenome. It is epigenetically based and electronically induced. It turns out Lamarck was more right than we knew.

This is precisely how non native EMF destroys your redox potential everywhere a hydroxyl radical is made. They are made when electrons are lost.

The end game for any mitochondrial Rx is to collect as many electrons as you can to affect a disease reversal or a positive change in body composition. You also need electrons to build a large EZ in water surrounding mitochondria. Some

will never get this effect no matter what they do because of where they live.

ULTIMATE TRUTH BOMB: Electron steal syndrome is most significant from non native EMF = Calcium efflux from cells = water cannot stay bound to proteins with negative charges = why everyone appears dehydrated despite your positive actions to drink reverse osmosis water. BOOM.

Yes, technology is killing us all softly while we *can't sleep*. Humans are not designed to deliver energy to other tissues before the CNS. The CNS is designed to get the lion's share and then deliver them to the rest of the body. This is precisely why omega 3 levels correlate with healthy organs.

This is why body building, marathon running, and extreme exercise can be associated with a higher incidence of cardiac mortality. These people are driving electrons into tissue systems that cannot handle the load of current. The muscle skeletal system does not have the DHA stores that your brain/immune system does to offload the power of these electrons. Normally, a good brain, immune system, and heart have a lot of DHA to funnel these electrons off to mitochondrion that can handle this current of flow. When you have a chronic deficiency in DHA, the mitochondrion in those tissues do not funnel into them. This leads to a massive influx of energy to tissues that cannot handle it. The result is fibrosis. Normally, these tissues have a backup system to offset the load. The brain and immune system have massive ability to catch electrons when DHA is present. When you move away from a DHA laden diet, you create the ideal situation to cause short circuits in your body. The excess energy leads to short circuiting of mitochondrion and the development of cardiac fibrosis. This is what happens in marathoners who eat carbs and forsake dietary DHA.

All of the above examples increase electron flow through the neocortex down into the white matter tracts of your brain and out through the peripheral nervous system to drive regeneration of your tissues. Autophagy and apoptosis are the change programs of the mitochondria designed to keep us metastable, young, and full of stem cells. When this balance is upset, neolithic disease is the result.

All disease begins with a loss of electrons somewhere in the system, but it is eventually felt in the electron transport

chain at the input of our mitochondrial cytochromes.

When electrons are not present on your cell membranes, you lose the ability to retain intracellular water because calcium is being effluxed. This is often why so many people can drink water, but it never seems to address their BUN/creatinine ratio.

Water hydration shells surround mitochondria, but when it is not there, protons build up around the mitochondria to further destroy the intracellular charges present.

Life is all about the recipe that is the **Mitochondrion Rx**.

The Mitochondrial Rx is all about electronic induction and protonation. Therefore, it is a quantized process just like the 30 steps of photosynthesis.

Bread Crumb alert: Light from electrons causes colloidal particles to draw closer together to [condense](#). This [condensation](#) draws additional microspheres toward the light in brain networks. Suspended particles in neurons, their mitochondrion, or in the CSF should then be expected to draw to incident light naturally with no added energy needed.

Glutathione is made in the cytosol, and all mammals should have massive amounts of glutathione provided (sulfur cycles are working in unison with light) in them to coordinate the redox potential to make sure proteins fold correctly. It is not a story about carbohydrates, proteins, or fats as most believe. If it were, the input to mitochondria would not be called the electron transport chain.

Soon, optical scanning via spectrometers will be available to show us when our mitochondrial cytochrome proteins are not handling electrons well. If you don't think this is coming, you might want to read cite 16.

Seven of the most abundant elements in the universe (hydrogen, helium, oxygen, nitrogen, magnesium, silicon, sulphur, and iron) are represented in most organisms' mitochondrion. This is the ingredient list for the recipe of life. What binds them to create life is energy from the sun's photoelectric effect. Light is primordial to how a mitochondrion functions. It seems highly probable that life anywhere will require the same ingredient list. After all, nature's laws are how the cook has to prepare their dishes. The dishes will vary based upon the conditions of existence, but we know the background epigenetic environment for life is water, light, and magnetism.

Photosynthesis = Mitochondrion. Both pathways have many

things in common like light, water, and electrons. Both use [bright white-light emitting atoms of manganese](#) and copper to harvest light. The cytochromes in our mitochondrion appear to dope with electrons to harvest the photons buried in electron energies. Mitochondrion appear to be doped to ZnSe proteins to form quantum dots in the brain as you heard in the June 2014 webinar. The take home: the pathway in our mitochondrion are a bit more complicated than they are in plants, but the story follows a very similar path.

CITES:

1. <http://caloriesproper.com/?p=4559>
2. <http://www.ncbi.nlm.nih.gov/pubmed/18779586>
3. Mitochondrial Rx webinar on this site
4. Robbins and Cotran (2008). *Pathologic Basis of Disease – 7th edition*. Elsevier. p. 16.
5. Brömme HJ, Mörke W, Peschke E (November 2002). "Transformation of barbituric acid into alloxan by hydroxyl radicals: interaction with melatonin and with other hydroxyl radical scavengers". *J. Pineal Res.* **33** (4): 239–47
6. Goldstein Sara, Meyerstein Dan, and Czapski Gidon (1993). "The Fenton reagents". *Free Radical Biology and Medicine* **15** (4): 435–445.
7. <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1459516/>
8. <http://www.ncbi.nlm.nih.gov/pubmed/19413949>
9. <http://www.ncbi.nlm.nih.gov/pubmed/21352799>
10. <http://www.sciencedirect.com/science/article/pii/S0003986103006519>
11. <http://www.pnas.org/content/99/3/1259.full>
12. www.ncbi.nlm.nih.gov/pubmed/2080472
13. <http://www.nature.com/scitable/topicpage/photosynthetic-cells-14025371>
14. <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3561461/>
15. <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2821140/>
16. <http://www.newswise.com/articles/view/618106/>
17. <http://www.plantcell.org/content/23/11/3879.full>
18. <http://www.life.illinois.edu/govindjee/papers/milestones.html>
19. <http://www.sciencedaily.com/releases/2014/01/140114203123.htm> (new way to make energy)