CPC #8: Brain Cancer

READERS SUMMARY:

1. How does water, non native EMF, and calcium efflux ruin mitochondrial signaling?

2. Is cancer an epigenetic or genetic disease or both?

3. How does Volkow, Achermann, and Bissell work all explain cancer?

4. What is the main mechanism of oncogenesis in our modern world?

5. How and why does cancer metastasize?

Everyone in science reads the same data. But few people see what I see. This blog is about that perspective.

Today we are going to follow up the last blog's idea's with a CPC designed to show you how the quantum biology of water and non native EMF sets the table for a disease like brain cancer. I will use the three research papers mentioned in Energy and Epigenetics 13 to open your eyes to a new reality. I am going to warn you this CPC is for the GEEKS and doctors who read my blogs, so expect your head to hurt a bit. There is no way to make this science easy to understand for the lay public. If you have any cancer or a low redox potential you are going to want to slowly dissect this one carefully. It is loaded with gems.

The science underpinning this blog can be found here.

Cancer is a disease where energy is being lost chronically over time. It is a disease state where a low redox state is

the rule not the exception. It is a state where the delta psi in mitochondria are off. A low redox potential = a low delta psi. It is a state where water chemistry is very altered. Water is lacking inside and around cells, and cells are dehydrated. The water that is left behind has very little oxygen tension in it. It has altered levels of protons and/or electrons in it too. This metabolic situation leads to karyotype contraction. This means the number, size and shape of chromosomes becomes altered. The exclusion zone of water is smaller than it should be in cancer and it has an altered ability to allow protons to flow through ATP synthetase Fo in mitochondria. This does not allow water to bind to proteins and cell membranes properly and it alters their ability to sense the magnetic fields in their environment. Sunlight and a magnetic field is capable of ionizing water in the entire universe, including inside your cells. This very basic fact is not yet a biologic law, however, it is a natural physical law found everywhere we look. Biology and oncology just have not changed their gaze from mutated genes yet. Moreover, oncology has not yet seen this significance for brain cancer. I am hoping to change that by showing you how this is tied together epigenetically to water and non native EMF exposure.

Researchers continue to believe these non native EMF frequencies have no capacity to "mutate genes" directly because their energies are too low. They are not like X rays or gamma rays (ionizing radiation). This perspective is a dangerous assumption. Following this line of reasoning, there is no simple and direct means of initiating cancer is possible in their eyes. This belief stops cancer researchers from even looking at this possibility from another viewpoint. Mina Bissell is not a "boxed in" thinker. She gives me hope that my profession will connect the dots soon for cancer generation.

What stops them is that since Richard Nixon declared war on cancer in 1971, cancer research has become a big business that

is now quite profitable to hospitals. When big profit is present, dogma soon follows in any field. **When dogma is entrenched in a field, ignorance soon follows.** That ignorance blocks us from asking the correct questions to solve the problem. I think solving the brain cancer equation is going to happen outside the mainstream oncologic world. I have heard my friends and colleagues say that do not believe this is possible today. Impossible is just somebody else's opinion. No tactic will help mankind out until someone does the work. I believe doing things correctly never goes out of style.

Why am I so optimistic? My failures in medicine have shown me the path I must travel to solve things no one else has with billions of dollars and lots of smart people supporting them. We need to look at the problem from a new vantage point to solve it. Cancer was as rare in 1900 in humans, as the truth is in Congress or the White House today. My failures taught me to "rethink the truth" about brain cancer. I began to question everything I was taught to believe about brain cancer. We have class one data linking some tumors of the head and neck to ionizing radiation, but no class one data to other parts of the electromagnetic spectrum. Today, I constantly seek to "unknow" many things. I now test everything that can be tested. I assume nothing. I have found, as soon as you think you know something, that's when you stop questioning it. If you do not continue to ask questions you keep traveling down the same road you're on, whether its right or wrong. I have learned, rather counterintuitively, that "understanding things" kills curiosity in most cases. Understanding kills your progress to a goal. We actually remain curious by asking better questions, once you think you have wisdom in "your pocket". That is how I see things now. It implies I can never stop asking questions no matter what I might find along the way. Today brain cancer is exploding in incidence and prevalence, yet we have no answers why?

LET US EXAMINE WHY?

Today, most oncologists think brain cancer is a genetic disease. They do because of bad research. See this link here. I don't, and never have. In fact, I know it is not based upon the questions I have asked myself over the last decade. Cancer incidence and prevalence is at an all time high across the world. We are not winning the war on cancer; in the 20th century it went from a nuisance disease, to an epidemic, and today, it is a pandemic. **Modern epidemics are not caused by genetics, but by epigenetics**! Any epidemiologist will tell you that this statement is fact. This concept is also a clinical fact that gets lost in the modern scientific literature but you would never get that from reading the literature on cancer. In fact, the totality of the cancer literature would have you believe the exact opposite. This is why oncologist believe what they do today. This is why they are on the same road looking for a "genetic cure". That "war on mutated genes" has been on going since 1971 at a cost of over 1 trillion dollars to society. With that kind of money spent, we have gone to the moon, and the outer reaches of the solar system, yet, we have found other galaxies and black holes but we have not put any dent in cancer incidence and prevalence overall. Puzzling no? Since 1900 cancer is a bigger problem than at any other time in human history. Einstein said that when you do the same thing over and over again and again and expect a different result that is the definition of insanity. The American Cancer Society, the NIH, oncology researchers apparently all think Einstein's insight must be foolish because that is precisely what they have been advocating since 1971. Because of this, I don't believe cancer's cure will be found on '***that road***', in my opinion. It is a neolithic thought that has hurt all modern humans afflicted with cancer and is at the seat of why modern medicine has failed to find a cure. The cure will come when we realize it is tied to circadian mismatches.

Today, oncologists have new drugs to treat certain leukemia's and this gives some people hope. Then they hear about the details of this new hope, and the details should alter their beliefs that Big Pharma has their best interests at heart. Ponatinib carries an annual cost of 138,000 dollars. Bostutinib's annual cost is 118, 000 dollars. Omacetaxine costs 105, 000 dollars a year. None offer a cure. Sounds like a bargain huh? Gleevac was the first drug developed to treat these diseases and it carried a 30,000 price tag when it was approved in 2001. Today, when the drug companies realized that governments and insurance carries would pay over a 100,000 a year for a drug that offers no cure in 2012, what did they do to Gleevac? Today it costs 92,000 dollars a year! It is a relative bargain, no? This story is being told to you for one reason.

You, the patient, must realize that drug companies care less about your health. They do not create cures, they create customers. Realize that most of the RCT's on cancers these drugs treat are paid for by them. Very few dollars for these drugs come from the NIH or private dollars. The results often suits their needs, not yours. You doctor has to "follow" this "evidence based game" they have created for us, with the federal governments help, of course. Otherwise, doctor's won't get paid for their care for you. CMS, the federal government dictate what and how doctors get paid today. This is why asking better questions must be done by YOU and no one else! You have an obligation to yourself only. You can only change yourself. You can't let other people tell you who you are. You have to decide that for yourself. Health improvements always begin with I, not we.

Who really can afford to pay 100,000 for any drug? This is society's cross to bear. Last I checked, every eyeball that reads this blog is part of greater society. That means you have a vested interest in this blog whether you know it or not. It may not come out of "your money" today, but it will

come out of your kids and grandkids salaries and their taxes.

Where many see impossible odds to change a paradigm, I see the optimistic possibilities. If your dreams comes in a package with labels like “impossible”, “larger than life”, “scary”, in my opinion, then you have got the right dreams. Don’t let small minded people convince you that your dreams are too big. Turn your dreams into actionable goals that can be executed everyday by everybody. Turn your dreams into a process that can be followed everyday with discipline. The data and knowledge and wisdom in this blog are designed with all this in mind.

When dreamers wake up and become doers, then destiny will wake up and become reality for them. This information doesn’t need to go viral; I just need to set hearts on fire today, and every day, from here on in. In the depth of ‘your’ winter, just smile within and slow burn with these facts on cancer today; eventually you will find that when you let this information sit in your mind, you will find within us all is an invincible summer. To be your best, you must begin to strive to be. **Begin to rethink your current truths**.

Today’s blog is about what I see about brain cancer, water, glucose and non native EMF. No disease frustrates neurosurgeons more than brain cancer. Why? Because we have not many any impact on the natural history of the disease in 120 years.

CANCER’S PLAN FOR THE BRAIN:

Oncology believes “bad genes” cause brain cancer: **Implicit in this belief is the assumption that to get cancer or illness you must have a mutated gene.** This is what we call genetic determinism. What they miss is that **genetic expression is**

altered by the electromagnetic signals coming from RNA and DNA and the non-ionizing frequency EMF bands do change this information processing that occurs in the hydration shell around our nucleic acids. They are not studying this aspect of the cellular communication system because they do not understand the physics of biology. Nor are they studying how hydrated electrified proteins work normally, while dehydrated proteins have abnormal function because calcium levels and oxygen levels are altered and set the stage for brain cancer to occur.

In Energy and Epigenetics 13, I told you about 3 researchers work, Volkow, Achermann, and Bissell. Volkow and Achermann both showed definitively that brain physiology is changed by up regulating glucose metabolism with the respect to non native EMF. Both of their studies are the best we have in reference to this specific issue in the published literature. Brain tumors have risen 500% over the last 50 years and no one seems to know why. Our third researcher, Bissell, a breast cancer expert, has pointed the cancer causation gun squarely at an altered glucose metabolism. Bissell went back to the work of Otto Warburg to show us all just how deadly glucose can be in cancer. She has famously said, ***glucose metabolism in the presence of oxygen, is not the consequence of the cancerous activity of malignant cells but is itself a cancerous event.*** Her position has pissed off a lot of folks in oncology. I think she is perceptive. I think she is the brightest star burning in oncology today. She is telling them all they are traveling on the wrong road, and they do not want to even contemplate if she is right, because they all stand to lose a lot of money and face. They also realize they have spent careers studying the wrong effects in this disease. Pride and lots of money is at stake for many industries. So what might the "right road" look like for brain cancer?

Does this road tie to water chemistry and non native EMF?

THE MECHANISM:

Other researchers in neuroscience have pointed the smoking gun at the mitochondria in neuro-degeneration. These are diseases modern medicine has no answer for either. Could they be linked? Mitochondria are parts of our cell where food gets turned to electrons and water to protons. Mitochondria are basically small factories that pump out protons at a massive rates normally. These things do not happen in unison in brain tumor states. Within these protons is stored potential energy, and this potential energy is eventually transferred to water hydration shells around proteins and mitochondria throughout the entire cell. This potential energy is stored in the hydronium ion of the exclusion zone of water (EZ). THE EZ is critical to cellular signaling in all life forms. When mitochondria make protons, water must be available to accept this energy package. If it is not, and mitochondria make too many protons, a positive charge builds up in and around the mitochondria because protons have a positive charge. A positive charge is acidic. This causes the local and surrounding pH to fall. This signal is read by the cell as rising inflammation and the mitochondria response is to increase synthesis in ROS and/or RNS. These are the two signaling molecules that mitochondrial mainly use to 'spread the word' to other parts of the cell of how much 'gasoline' is in the gas tank to do cell work. When that message gets muddled for any reason, we see problems develop at **complex I of electron train transport chain (ECT).** When failures occur here, an awful lot goes wrong in a cell. Most of this signaling chaos surrounding the basic mitochondrial information comes from literature on Parkinson's disease or Alzheimer's disease. These are diseases one gets before cancer usually develops but shows the underlying problem at the mitochondrial and quantum level for all these diseases.

THE MECHANISM ORIGIN

So let's look at what happens at cytochrome 1 carefully, in this scenario. Cytochrome 1 uses NADH and NAD+ to work optimally. Cancer develops when NADH is too high and NADPH is low chronically. Carbohydrate electrons derived from foods increase NADH levels at cytochrome one. Remember, Volkow and Achermann said that non native EMF up regulates glucose metabolism. This means non native EMF increase NADH at cytochrome one naturally. NADPH comes from NAD+. Cytochrome 1 uses NAD+ and NADH to create a small pulse of superoxide normally. When you fuel your diet with glucose, you deplete cytochrome 1 of NAD+ normally. When mitochondrial NAD+ levels are low, this information about a "low energy state" is sent to the cytoplasm where mitochondrial DNA is and to the nucleus where the somatic genes are housed. Calcium is the cation that signals this event. Ca2+ signaling triggers a larger than expected elevation of reactive oxygen species in mitochondria, leading to the translocation of NF-κB and STAT-3 into the nucleus. These two signaling molecules were talked about in depth in the original leptin series on my blog. Calcium has this direct effect on water because it is ***hydrophobic*** and ***decreases the size of the EZ*** in intracellular water. This lowers the potential energy of the hydronium ions in the EZ. As calcium rises, magnesium falls dramatically, because it needs water to work because it is hydrophilic chemically. I told you in the Gnoll's blog post a loss of magnesium is one of the *first things* that goes awry in diabetes. Here you can see why diabetes and cancer are linked now. They share a common pathway.

When this happens simultaneously, water loses its ability to charge separate and the redox potential in the local area declines fast. When nuclear levels of NAD+ are low chronically, a sequence of events occurs in the cell nucleus that ultimately results **in a lack of adequate expression of mitochondrial DNA genes that are required for the construction of Complex I, III, and IV in mitochondrial electron transport.** In this scenario, the nuclear-encoded proteins

required for mitochondrial electron transport chain are still expressed normally, but the proteins whose genes are encoded only by mitochondrial DNA are not expressed. This is the critical point in cancer generation that occurs long before you have brain cancer diagnosed. **This is why the work of Volkow and Achermann are critical pieces of the brain cancer puzzle.** See, folks, your genes and mitochondrial genes are handled 100% differently in a quantum cell.

KEY POINT FOR THE NON GEEKS: Oncologist are looking at human somatic genes in the nucleus, when the real problem is the mitochondrial genes that allow us handle electrons and protons from food and water properly to signal how the cell sees the outside world within.

Modern technology uses non native EMF frequencies that dehydrates cells and up regulates glucose metabolism and causes calcium efflux in cells. Now let us look at what that means with respect to brain cancer.

GEEKS: Thus, when this happens, normal mitochondrial electron transport (published in any biochemistry textbook) cannot occur normally. This leads these altered cells to develop a metabolic picture which is “typical” in cancer, which is called the Warburg effect. Bissell is credited with rediscovering this type of metabolism in breast cancer research. With a Warburg-type metabolism, cells do not generate ATP from the mitochondria normally, and instead, become dependent on cytoplasmic generation of ATP via aerobic glycolysis. This is a very different and very inefficient way of transferring energy around a cell, but it is fast. Rapidly growing cells need energy delivered quickly. This is why this pathway is chosen. The same effect was shown to you in EMF 4 blog post.

The primary driver of this “metabolic reprogramming” is the transcription factor called, Hypoxia Inducing Factor 1 alpha (HIF-1α). HIF-1α normally only activates Warburg-type metabolism **when oxygen levels are low**. Hypoxia-inducible factor (HIF)-1, has an α-subunit and a β-subunit. It is a sequence-specific DNA-binding transcriptional complex protein that regulates genes involved in angiogenesis, glucose metabolism, cell proliferation and invasion/metastasis. This protein links oxygen tensions to cancer generation in cells.

Oxygen levels in tissues are low when we are dehydrated naturally. Non native EMF dehydrates cells of both intracellular and extracellular water. It also releases calcium from the cells endoplasmic reticulum and lowers magnesium levels dramatically. When oxygen levels are lowered in intra or extracellular water, it acts differently chemically than it does under normal conditions. The chemistry of water, in this case, carries a lower oxygen tension. **Most oncology research never studies the effect of intracellular water in cancer, but every MRI scan done on brain cancer shows an altered water response in their images. This is why in the redox Rx I told you MRI’s are extremely valuable to the astute clinician.** The brain and spinal cord are enveloped by CSF made from this water. CSF is 99% water by volume. This water comes from the blood plasma. Within the CNS, CSF is made in the ventricular system. CSF is designed to turn over 4 times a day in humans. CSF water must be recycled for brain functioning to remain optimal. In today’s modern world, these factors in cell’s are under 24/7 assault. Water chemistry is very altered in all brain diseases. This is why neuro-degeneration and brain cancer are exploding in incidence and prevalence today.

<u>Consider the following brain facts:</u>

Total volume of cerebrospinal fluid (adult) = 125-150 ml
Total volume of cerebrospinal fluid (infant) = 50 ml
Turnover of entire volume of cerebrospinal fluid = 3 to 4

times per day (from Kandel et al., 2000, p. 1296)
Rate of production of CSF = 0.35 ml/min (500 ml/day) (from Kandel et al., 2000, p. 1296)
pH of cerebrospinal fluid = 7.33 (notice it is slightly acidic due to the proton force of mitochondria from Kandel et al., 2000, p. 1296)
Specific gravity of cerebrospinal fluid = 1.007

Now let us look at what calcium in a neuron should look like under normal brain conditions.

Ion Concentration (mM) – MAMMALIAN NEURON		
	Intracellular	Extracellular
Potassium	140	5
Sodium	5-15	145
Chloride	4-30	110
Calcium	**0.0001**	**1-2**

When we have an altered field due to excessive EMF that blocks the normal ELF EMFs from the Earth's magnetic field. We also get non-thermal effects directly in our brain and across our brainstem. This was shown by Allan Frey's research in the 1960's. It was reconfirmed by the work done by Robert O. Becker and Carl F. Blackman a decade later. Their data are critical in understanding how a field effect can lead to brain cancer. Blackman found that non-thermal EMF exhibited both frequency and intensity windows on biologic cells. Blackman was a research scientist at the Environmental Carcinogenesis Division of the EPA. He also conducted work with Dr. Abraham R. Libroff of Florida Atlantic University. Blackman and Libroff combined a static (DC) and an alternating (AC) magnetic field on human cells. They found doing so caused an increase of the concentration of **free calcium ions in nervous tissues** in a *very narrow resonance window* of the AC magnetic field. The maximum calcium efflux from neurons corresponded

to the cyclotron frequency of the calcium ions. (Smoking gun alert)

The reason why this is important to you and your biology in the brain is that when calcium rises inside your cell membrane it is pumped out immediately because a rise intracellular calcium mediates apoptosis and autophagy and causes massive problems in intracellular signaling. Look at the above table. Calcium is tightly regulated in neurons and glial cells. This is why apoptosis and autophagy are rare in the human brain. Brain cells do not divide a lot compared to places like the gut which turns over every 48 hours. This occurs because calmodulin signaling is critical in keeping calcium levels low in neurons and glia.

But under the modern world's non native EMF assault it becomes radically increased and altered from baseline. Calmodulin controls calcium and magnesium ion movements in cells. When these two processes are usurped, brain cancer is a possible result if the redox potential remains quite low chronically. Remember, abnormal Ca2+ signaling triggers the elevation of reactive oxygen species in mitochondria, leading to the translocation of NF-κB and STAT-3 into the nucleus. You might want to click on those hyper links you just read and re read those two blogs. NF-κB and STAT-3 are the '911 signals' when they are found in higher concentrations in the nucleus where somatic DNA lives.

Rising intracellular calcium goes by another name you might have heard; ***it is called excitotoxicity***. Excitotoxicity is tied to heart disease, neuro-degeneration, atherosclerosis, sleep apnea, alterations of circadian cycles and autoimmune conditions. Calcium efflux occurs due to direct alteration of voltage gated channels when an altered electromagnetic field is present. When the field strength in the environment is above 30 Hz, we now know that we have altered calcium and calmodulin signaling issues. To date, 23 studies have been done and repeated this finding since 1980.

Non native EMF causes calcium efflux everywhere in all cell lines. Those cells with tighter calcium control (neurons and mitochondria) will be most affected by these changes. This is why brain cancer and heart disease is rising and rising fast in our modern world. Higher intracellular calcium levels in biology all lead to more inflammatory cytokine production in all cell lines in the CNS. In fact, this is why the war on cancer is being lost.

These events also occur and are additive to the cell's "redox sink" when a person is leptin resistant. Overall, when energy is being used up faster than it can be made, this lowers someone's redox potential quickly. **This is why metastatic disease occurs in cancer.** Brain cancers rarely metastasize. Do you want to know why this is the case? The brain has the highest density of mitochondria than any organ in the body so these cancers rarely run out of mitochondria that are employing Warburg metabolism to support their energy needs. The most malignant brain tumor, called gliobalstoma multiformans (GBM) just continues to invade the hemisphere to usurp the adjacent brain's mitochondria to fuel its growth using cytosolic glucose metabolism.

Cancers that spread easily to other organs all have the lowest amounts of mitochondrial density within them. Therefore when they have exceeded their mitochondrial efficiency to make ATP and oxygen, cancer cells have to travel to other tissues to find suitable mitochondria because cancerous mitochondria have altered complex one, 3, and 4. These altered complexes are limited in their ability to make ATP and O2 production in aging and neolithic disease states because they can no longer tunnel electrons well. Remember those electrons come from food. Breast cancer and prostate being the ideal example. Breast cancer loves to metastasize to bone and brain tissues. Prostate cancer loves to metastasize to bone as well. Why does cancer pick these two tissues over other organs? Because both tissues have superior densities of mitochondria to

replace cytochrome 1, 3 and 4 to make ATP and oxygen to fuel cancer's growth.

Breast tissues only has mitochondrial densities increased and expressed when it is making milk for a pregnancy. When a women is non pregnant the mitochondrial density is low because her hormone levels are not calling for her mammary glands to express more mitochondria to make milk. Maybe you are beginning to understand why now, many breast cancers have up-regulated hormone receptors on them? This up-regulation of the hormone receptors on their cell membrane allows the breast tissue to make more mitochondria, via mitochondrial biogenesis, with the lower levels of hormones present in the non pregnant state. It does this so the cancerous cells do not have to metastasize to go elsewhere to find new mitochondria.

HORMONES AND CANCER:

When you understand this issue it helps clear "another paradox" in breast and prostate cancer with respect to hormone levels. This implies that we should be giving people with hormone sensitive cancers these *hormones exogenously* to save them metastatic disease spread to other tissues. This strategy may increase the "local effect" of cancer growth but it likely will confine it. Metastatic spread is what kills humans. Today, oncologic beliefs are 180 degrees opposite this idea. Ask any doctor if this is not true. Modern medicine and oncology still have not explained why a good progesterone/estradiol ratio in women and high testosterone levels in men are GOOD prognostic signs and not bad ones. This has been shown recently in the literature. This is why the most malignant prostate cancers are seen with the lowest testosterone levels. It is also why women with a poor hormone panels are at the highest risk for the worse type of breast cancers. No one is connecting these dots today. If you carry

these risks you must pay attention to it. Sex hormone bonding globulin (SHBG) is the wild card action in these hormone sensitive cancers and is tied to its molecular acrobatics. It explains why it walks the hermophrodite pathways between estrogen and testosterone levels in the face of a low redox potential.

BIOCHEM GEEKS: Biochemistry in a text book will never show you the magic of SHBG in hormone sensitive cancers. When one understands the quantized mechanisms at play in melanopsin, actin, myosin, and rhodopsin you can begin to understand SHBG interaction with estrogen and testosterone. It all comes down to a transition metal at a key binding site. The primary and second structure of proteins are all controlled by nucleic acid codes. The tertiary and quaternary structure of proteins is the domain of epigenetic modifications of non coding DNA. This is where the action happens for SHBG. The primary structure of SHBG comprises tandem laminin G-like (LG) domains. The amino-terminal LG-domain includes the steroid-binding site and dimerization interface, and its tertiary structure, resolved in complex with natural and synthetic sex steroids, has revealed unanticipated mechanisms of steroid binding at the atomic level. This LG-domain interacts with fibulin-1D and fibulin- 2 in a ligand-specific manner, and this is attributed to the unique way estrogens reside within the steroid-binding site, and the ordering of an otherwise flexible loop structure covering the entrance of the steroid-binding pocket.

Estrogen is designed to work in with SHBG because it is associated with a lower level of iodine in women. This changes the delocalization of electrons of zinc’s D shell electrons. There are however two other transition metals binding sites (cation) The cation changes make SHBG resonant to different electromagnetic frequencies when different sex steroids bond to the loop ring. This mechanism enables estradiol to enhance the sequestration of plasma SHBG by the stroma of some tissues

through binding to these extra-cellular matrix-associated proteins. Just look at this picture! This is how breast cancer up regulates its own biogenesis of mitochondria to get new energy sources in its own tissue. The human SHBG amino-terminal LG-domain (important hyperlink alert) also contains several cation-binding sites, and occupancy of a zinc-binding site influences its affinity for estradiol. To prove to you I might be correct about this quantum SHBG dance, biochemistry still does not have the complete quaternary structure of SHBG even today. The reason? They do not realize it changes with the electromagnetic force of the environment that the tissues are in. It also varies with the redox potential of the tissues where it is made.

When you understand protein structural predictions of tertiary and quaternary structures suggest that the carboxy-terminal LG-domains extend laterally from the dimerized amino-terminal LG-domains of SHBG.

Men and women have different "Maxwell demons" to account for the different results of the same quantum actions. When you understand the molecular formula printing in a textbook does not need to change to get a different end point results your view point changes, and you begin to see why oncologic researchers are barking up the wrong tree of cancer causation. You don't believe it can happen? Look at the mechanism of action of rhodopsin, melanopsin, actin and myosin. The only thing that changes in their structures is rotational bond angles and their atomic positions but the molecular structures printed are exactly the same. Estrogen, testosterone and SHBG do the exact same thing. When you get that it can and does happen in real biochemistry a lot more often than anyone realizes……then I can explain to you how the sex steroid hormones do their magic in health and in cancer states. They are not the cause of cancer, they act to help it when you understand the quantum molecular actions of how they change. You got to run from biochemistry's current beliefs. Everything

is based on the atomic molecular actions of QED. **When your target is wrong……..what good is your literature.** This is the core message of Mina Bissell. I am just putting an exclamation point on her work.

BACK TO THE BRAIN

Brain cancer does not have to travel because the brain is loaded with mitochondria everywhere one looks. All of this is tied to the ideas in Energy and Epigenetics 13, and the work of Bissell, Volkow, and Achermann. Today, I am laying it all out to show you how cancers really begin and act using the 3 legged stool buried in Energy and Epigenetics 4. It is a water, non native EMF, and photoelectric story and how mitochondrial function is usurped in cancer and low redox states.

The brain has a massive mitochondrial density normally. Many studies have frequently focused on mitochondrial density, which, although a major determinant in energy generation, does not in itself present a full picture of the bioenergetic profile of the tissue in question. Mitochondrial populations from different tissues or species are known to be functionally distinct, having different respiration rates for a given unit of mitochondrial mass, and this is by no means a new concept. It has been known for over 35 years that even within a single cardiac muscle fibre there are two biochemically distinct mitochondrial subpopulations situated in the subsarcolemmal and intermyofibrillar regions. (Palmer et al. 1977). What is not so well known is that cancer seeks out that mitochondrial density and the excellent respiratory burst when it can no longer use its own mitochondria.

Places where mitochondria are most active and have a high respiratory rate are where cancer will seek to go in metastatic disease. They will leave their hypoxic beginnings to find oxygen and mitochondria to survive. Dr. Bissell and Warburg ideas were spot on. Why they were spot, has never

been uttered in any literature I have read as far as I know. As a neurosurgeon, I see most metastatic disease I have treated in the brain occur at the grey/white neocortical junction of the cerebral hemispheres. At this junction one can find the highest density of mitochondria with brisk respiratory bursts that make massive amounts of oxygen for the neocortex. Form meets function in all of evolution , even in cancer states.

TABLE OF BRAIN FACTS: Grey mater is where the oxygen is found not the white matter.

Ratio of the volume of grey matter to white matter in the cerebral hemipheres (20 yrs. old) = 1.3 (Miller et al., 1980)

Ratio of the volume of grey matter to white matter in the cerebral hemipheres (50 yrs. old) = 1.1 (Miller et al., 1980)

Ratio of the volume of grey matter to white matter in the cerebral hemipheres (100 yrs. old) = **1.5** (Miller et al., 1980) ***As humans age the ratio of grey to white matter increases and this is why cancer seeks out the brain's mitochondrial oxygen sink.***
% of cerebral oxygen consumption by white matter = 6%
% of cerebral oxygen consumption by gray matter = 94%

Cerebral blood flow (gray matter) = 75 ml/100 g brain tissue/min
Cerebral blood flow (white matter) = 45 ml/100 g

Oxygen consumption whole brain = 46 cm^3/min
Oxygen consumption whole brain = 3.3 ml/100 g/min
Blood flow rate through each carotid artery = 350 ml/min
Blood flow rate through basilar artery = 100-200 ml/min

This is why most metastatic lesions are supratentorial and not infratentorial. There is more grey matter in this location in the brain. This means there are more mitochondria there. When infratentorial metastatic disease is present the neurosurgeon should realize that this is likely because there is a metabolic problem with the mitochondria in the hemispheres above. This implies that in those with pre existing cerebral disease we should see more "mets" in their cerebellum. This is precisely what we do see today. This is also why in kids most of their malignant brain tumors are found below the tentorium and not above it because in a developing brain there is less grey matter than in an adult. In children, blood flow through whole brain = 105 ml/100 g/min. Hind brain development is the most active site of grey matter development between 6 months old and 6 years old. This is why Autism begins in the hind brain too. Children also have lower levels of myelination than adults and this is what make them so susceptible to non native EMF of all frequencies. Volumetric MRI studies, using various methods, have reliably found that gray-matter volume increases and peaks in late childhood in the cerebral hemispheres, followed by a slow but continued loss, whereas white matter increases rapidly until age 10 years with continued development well beyond adolescence.

This is why I am very concerned about the explosion of use of technology devices around a young developing brain. My prediction based upon the science in this blog is the current generation risk of brain cancer and neuro-degeneration will be explosive to compared to any other cohort we know of today. This will take 25-50 years to see but I think this is why we are seeing neurodegenerative disorders and spectrum disorders explode already in the below 50 year cohort even today compared to 50 years ago. This is already happening in AD, PD, and in brain cancer. It is also why many of these tumors are associated with drop metastasis in kids. The tumor is following the CSF/water energy gradients, in my opinion. No

one in neurosurgery has published a good idea why drop metastasis happen in kids and rarely in adult brain cancers. I think energy and mitochondrial power dynamics is why it might happen.

Metastatic spread is the cancer is looking for a highly oxygenated tissue that signals " here is a tissue with better mitochondrial respiratory burst than the parent tumor bed" to generate energy for itself. It is looking to survive a chronic low redox state. Ironically, today we think of metastatic disease with a negative connotation, when we should realize clinically that it is the perfect sign that the main tumor's mitochondria are about 'shot' because they can not generate an electric charge (delta psi) and therefore can no longer make new mitochondria via biogenesis.

A lowered redox potential is due to a loss of charge for some reason on the inner mitochondrial membrane. Cancer cells use glucose for their fuel source because this allows for a lowered electric charge than ketones or fat would. Glucose only generates 36 ATP while fats generate 4.3 times as much ATP (147). ATP is what opens proteins water bonding sites. When you have cancer you do not utilize all your water binding cites around proteins for two reasons. One, you are dehydrated from the non native EMF inside the cell, and two, your cancer cells predominately only use glucose which lowers the charge on membranes in a cell and in mitochondria. This is why all conditions tied to leptin resistance can all be associated with cancer generation at some level. Leptin resistance (high NF-kB nuclear translocation) is also a low redox state, a low delta psi state, a state where **water is absent or can not charge separate for any reason**. This is built into the scale of bio-energics of a cell. It is built by evolutionary fractal design. Oncology still does not see any of this self similarity.

RETURNING TO THE MECHANISM OF ONCOGENESIS: GEEK FEST

Hypoxia has major effects on mitochondrial efficiency and biogenesis. Mitochondrial signaling is a big deal in the case of cancer because superoxide generation from cytochrome one (NAD+) is altered forever. They must use other means of energy generation. When nuclear NAD+ levels are low, HIF-1α is stabilized chemically and is not degraded by von Hippel–Lindau protein (pVHL). In non cancerous states, VHL degrades HIF-1α very rapidly. Why is this point a big deal in cancer? pVHL ties directly to the ubiquination pathways in cells. Ubiquination tags cellular proteins, glycoproteins, and cell membrane lipids for replacement. They must be working well on a atomic or quantum level to signal environmental cues from the environment to the mitochondria. They are all yoked to circadian biology by SIRT 1. They cannot in cancer. SIRT 1 and 6 are massively important. I have also told you that ubiquination or cellular catabolism and resultant anabolic processes are the most costly things a cell can do on an energy basis. In cancer, energy is already low because you can not make ATP well because cytochrome 1, 3, and 4 proteins are not being expressed by mitochondrial DNA.

When all these things happen in simultanity within a cell, as occurs in our modern world, cells are “metabolically shifted and reprogrammed” to use aerobic glycolysis in the presence of normal oxygen. HIF-1α inhibits a chain of events that ultimately results in a reduced expression of the mitochondrial transcription factor, TFAM, which normally migrates from the cell nucleus to the mitochondria to stimulate mitochondrial DNA replication. This is 100% an epigenetic shift to the environment the cells are in and not from the DNA they inherited from their parents.

As a result of this epigenetic shift, mitochondrial encoded genes are altered and their expressed function changes. This implies that we can pass on this environmental information to future generations via our germ cell lines. This is why today we are seeing massive neolithic disease generation in younger

people today. Since 1970, these children are being born with lowered redox potentials because of the environments their parents were forced to exist in by societies choices.

When HIF-1α inhibits **mitochondrial transcription factor A this can't occur. Mitochondrial transcription factor A is** abbreviated as *TFAM* or *mtTFAM.* This is a "**big assed**" deal in cancer. These circumstances allow nuclear encoded mitochondrial proteins to still to be created via the transcription factor Nrf1. But where the rubber meets the road in cancer is with TFAM. When TFAM is suppressed by HIF-1α, Nitric Oxide mitochondrial-encoded protein components of the electron transport chain are expressed even though their mitochondrial DNA mutations do not allow them to properly tunnel electrons from foods. When they are placed into the ECT they become non functional in passing electrons to oxygen. This means no oxygen is reduced. This results in mitochondria that can no longer make ATP at all; because they still have a defective ECT that has to "run in reverse" to get around this situation. The significance of this is, that unfettered "reverse electron transport" results in the production of **uncontrolled amounts of charged free radicals from the mitochondria.** These are both ROS and RNS moieties I have been telling you about.

Mitochondria normally pump out huge amounts of positively charged protons. This action is what drives the earth magnetic field and it is what drives your life force (Bronson/Ali alert). This altered electrical charge on the inner mitochondrial membrane immediately destroys the pH gradient of the proton motive force in mitochondria. This directly destroys proper redox signaling in mitochondria. The charge on the inner mitochondrial membrane drops quickly. This then creates the "primordial molecular signal of aging and disease" we see in all experiments. The signal is mitochondrial dysfunction with a higher free radical production. **All of these events can be traced back to**

inadequate NAD+ in the nucleus not the cytoplasm. Without adequate NAD+ in the nucleus, SIRT 1 and SIRT6 cannot function. Sirtuin 1 and Sirtuin 6 is directly tied to excessive nuclear aging. Why is this a big deal?

SIRT 1 plays the pivotal role as a "deacetylase silent mating-type information regulation 2 homolog". What does this mean in english? SIRT1 is the major molecular effector of the environment in shaping the circadian epigenetic landscape present within the quantum cell. The circadian clock is a key element in homeostatic regulation by controlling a large array of genes implicated in cellular metabolism. Importantly, SIRT 1 links epigenetic regulation to the circadian clocks in our brain and in tissues to help control and master the direction of the plasticity of the response to the environment. (Think metastability)

In the nucleus is where our somatic DNA resides. This is the DNA that the oncologist and their researchers have been wasting billions of dollars studying. The problem is upstream of somatic DNA. It is an epigenetic mitochondrial shift that causes cancer. This is where the epigenetic changes from altered mitochondria hijack our DNA mechanisms in the nucleus and cause all the changes oncologists are studying. None of these changes are the initial cause. This is why cancer remains vexing to them. The key change is NAD+ levels in the nucleus. **Guess what replenishes NAD+ fastest? A specific ketogenic diet does. Do you see why I wrote the Epi-paleo Rx yet? It is the cheapest easiest way to avoid all neolithic disease. This blog is the basis of the entire book.**

SIRT6 regulates genomic stability in the nucleus where your DNA lives. SIRT6 promotes genome stability by regulating DNA single-strand and double strand break repair pathways and by facilitating telomere maintenance. When SIRT 6 is altered the caps on our telomeres shorten via an enzyme called telomerase.

As telomeres shorten with each cell division, the heterochromatin marks are lost when SIRT6 is not working due to NAD+ deficiency or due to a lack of SIRT1. It turns out SIRT1 binds to the promoter for SIRT6 to allow it to function. Here you see the direct linkage of environmental circadian signaling and NAD+ levels in the mitochondria. This is massively important concept to master.

Normally in the embryo and in the young person, as a cell ends up with a critically short telomere, the cell undergoes apoptosis. However, as one ages, the possibility of the cell undergoing cellular senescence rather than apoptosis (with telomere shortening) increases. The shorter a telomere gets the higher the chance that the genes in the genome are altered instead of the telomere cap when the cell undergoes chromosome separation. So what does a cell do that has a short telomere cap? The subtelomeric DNA gets silenced by the sirtuins and the probability of cellular senescence increases and cancer lessens. **When this does not happen, cancer is the result.** Normally, subtelomeric DNA is heavily methylated at CpG dinucleotides. Telomeric DNA is NOT normally methylated, however, because there are no cytosines are found in telomeres. Only cytosines can be methylated in DNA. It also turns out that only T, A, and G bases are found in the telomeric repeats at the end of chromosomes. The methylation of subtelomeric DNA is very important, however, because when DNA methyltransferases are inactivated in embryonic stem cells, the cells loose their telomere length a rapid rates.

SIRT 6 has two major functions in humans. It deacetylates histones and mono-ADP ribosylation of PARP1 proteins on Histone 3 subunits. SIRT6 also deacetylates the H3K9 and H3K56 lysines. This increases telomere stability and also promotes double stranded DNA break repair by both the homologous recombination (HR) and non-homologous end joining (NHEJ) routes via PARP1 independent routes. PARP1 is the metabolic route in a cell that needs a constant high NAD+ to

keep us well. This high level of NAD+ is also linked to how growth hormone acts in the brain. Many doctors believe today that high IGF-1 levels can cause cancer by itself. Nothing could be further from the truth. To cause cancer the redox potential must be low. Why can I say this?

Growth Hormone and high IGF-1 expression in the brain is tied to SIRT6 function in neurons. It also turns out SIRT6 activation decreases triglyceride synthesis. Sirt 6 also sensitizes the genome to the electromagnetic spectrum. Thus, SIRT6 deficiency or SIRT6 dysfunction due to nuclear NAD+ deficiency can possibly explain many of features of aging and why no one seems to understand the paradoxical data on growth hormone. It is simple: control for NAD+ levels and you will see IGF 1 can diminish aging. If NADH is high IGF-1 can kill you by activating the cancer mechanism in this blog. The same molecular key can open the door to heaven or hell depending upon the redox potential of the surrounding tissues. NAD+ is a required cofactor for histone deacetylation. NAD+ dependent histone deacetylation is an important part of DNA repair. NADH is the inhibitor of this reaction. Both SIRT1 and SIRT6 are recruited to sites of DNA damage. SIRT6 deacetylates H3K56ac and SIRT1 deacetylates H3K16ac. Both SIRT6 and SIRT1 can deacetylate H3K9ac. SIRT 1 and 6 mark DNA sites for repair from epigenetic stressors.

In addition, SIRT6 puts multiple mono-ADP-ribosyl groups on the DNA repair protein, PARP1, which also repairs DNA in response to oxidative stress. Unfortunately, all of this DNA repair by PARP1 uses up NAD+, which leaves the cell nuclear levels of NAD+ low. As a result, all of the SIRT1 proteins cannot work since they all need NAD+ as a **cofactor for enzyme action. (BOOM)** Thus, the dual role of SIRT6 in both telomeric silencing and DNA repair is the root issue associated with aging and cancer generation.

PARP1 = Poly(ADP-ribose) (PAR) and the PAR polymerases. (PARPs) that catalyze its synthesis from donor nicotinamide

adenine dinucleotide (NAD^+). Poly(ADP-ribosyl)ation (PARylation) plays diverse roles in many molecular and cellular processes, including DNA damage detection and repair, chromatin modification, transcription, cell death pathways, insulator function, and mitotic apparatus function. These processes are critical for many physiological and pathophysiological outcomes, including genome maintenance, carcinogenesis, aging, inflammation, and neuronal function. When you slow aging down you slow all neolithic disease down. This is how it works across the board.

I apologize for the head splitting biology but you do not need to know it all to get the gist of the blog. You don't have to speak Russian to like the town of Odessa.

SUMMARY FOR THE NON GEEKS:

It turns out that nuclear NAD+ is a mandatory co-factor for all of the proper functioning of sirtuin signaling in humans. Sirtuins control the mTOR (SIRTUIN) pathway in humans. Many diet docs talk about the mTOR pathways but few of them truly understand how it answers to the redox pathways in cells. What happens in a worm or yeast is not congruent to humans. Today you got that introduction why. Worms and yeast use SIRT 2, 3, 4, 5 and we do not. The sirtuins have there own "levee" in my Quilt document. So does apoptosis, autophagy, and oncogenesis. Researchers across the world are waking up that quantum tunneling of electrons is directly linked deficiency of NAD+ in the nucleus of cells. This produces a state of "pseudohypoxia", where there is adequate oxygen levels but also high levels of HIF-1α. 24/7 carbohydrate exposure cause this effect. I agree with Mina Bissell. The number and quality of mitochondria are directly proportional to the effects of electromagnetic force brought to bear on the mitochondrial cytochromes. 24/7 carbohydrates deliver fewer electrons with higher energies simulating a summer energy footprint to cytochrome one. This generates excessive ROS and

RNS and the epigenetic changes in mitochondrial DNA begin.

Carbohydrate foods are designed to grow and be eaten only in long light cycles and seasons and they enter at cytochromes and give us a specific “redox footprint” to the mitochondrial cytochromes. Delta psi is also known as the inner mitochondrial membrane electric potential. We can also call it the redox potential of the membrane. Saturated fats (FFA’s) drive reverse electron flow through complex I when delta psi is high (redox high) by adding electrons directly to the CoQ pool via FADH2 at cytochrome 2. When delta psi is lower (low redox potential) or depressed (think too much glucose), oxygen consumption is also depressed, and NADH accumulates at the expense of NAD+ levels, which drop. A reduced CoQ couple is required for reverse electron flow through cytochrome one. Therefore you can see excessive glucose blocks reverse electron flow and can shut down energy production and superoxide production. The brain hates superoxide but it loves ketones. You might want to click on that hyperlink you just passed. I hope today’s blog is showing you why my book is “lady evolution’s” answer to our neolithic problems now.

If you want more fuel for the fire have a look here at this feasibility study on a ketogenic diet from 2005. a. Hyperlink

b. hyperlink from 2006 about a diet for Parkinson’s disease.

The data is there is you bother to look for it. Parkinson’s disease is a disease humans tend to get before they develop cancers. So we need to realize this small feasibility study is a big deal. Why? The trial had only 7 people in it, yet 5 of them got better!!! This happened in a disease that my current profession believes is incurable and uses deep brain stimulation to treat. The study only lasted 4 weeks. So let us review this. In an incurable disease, you show 5/7 people getting better; this should have been a signal to researchers and clinicians that this was massively significant

clinical result. Why? If you have a therapy which produces a large difference in a bad disease it shows up in a very small trial, it indicates the TREATMENT IS very powerful. Conversely, if your trial needs 100,000 people to show a minor clinical benefit it tells you the treatment is likely not powerful. This is lost on neuroscience researchers and neurosurgeons today. It was never followed up as far as I know. Today's blog and my book are showing you why ketosis is a powerful treatment for all diseases linked to poor mitochondria.

SEASONAL INFORMATION QUANTUM STYLE

It turns out seasons on Earth also have their own special electromagnetic footprint that the cytochrome proteins are exquisitely designed to also sense. This is not just a "food electron carbohydrate story". It is also an electromagnetic force story because of the how mitochondria are built by evolutionary design to sense the daily and seasonal changes of the light cycle and the electromagnetic spectrum and its associated forces. Water is the molecule that is the "quantum communicator" or the chemical molecule fully capable of deciphering the environmental changes and delivering that energy message to the proper cytochrome place in mitochondrial based upon the energies and information included in electrons from food and the sheer number of protons delivered to mitochondria. Cytochrome 1 is where electrons from carbohydrates enter the ETC and cytochrome 2 is where electrons from fats enter the ETC. Electrons impart a direct quantum effect on quantum electron tunneling based upon where they go and the proteins they face. These proteins are designed to harvest the energy and the spin information of these particles as they pass on down to oxygen.

If you do not think electrons have a direct quantum effect consider this link: Red blood cells are glycolysis-dependent cells which lack mitochondria naturally. NADH is recycled out towards the plasma membrane transport system. RBC's

naturally export 2 electrons to give rise to intercellular ROS which acts a signaling mechanism. This oxidizes ascorbic acid (vitamin C) and directly affect glutathione levels inside the cell as well as raised iron levels. Ascorbic acid is an antioxidant that maintains hemoglobin in a reduced state and minimizes RBC oxidative injury. It directly effects collagen and fibrin actions in RBC's. When electrons are missing the RBC's are oxidized RBCs are elongated and not biconcave and easily deformable. The oxidized ascorbate (AA) then transitions to its deoxy form called dehydroascorbic acid (DHA). When DHA enters the erythrocyte its reduction to AA regenerates another NAD+ and this is able to reduce glutathione to make the RBC a deformable biconcave cell. RBC's normally exhibit a Warburg metabolism their entire life because they have no mitochondria because their cell is loaded with hemoglobin. Two electrons cause this effect!!!

BACK TO US

When electrons tunneling is altered, charge is lost on the inner mitochondrial membrane, and the redox potential falls and the pH gradient vanishes. ***When the redox potential falls this means you are less reduced and more oxidized***. Oxidative stress makes calcium go from endoplasmic reticulum to the mitochondria, where it also inhibits complex 1. This is a bigger effect in our modern world because non native EMF causes massive calcium efflux from all cell lines. This extra calcium really shuts down cytochrome 1. Calcium is hydrophobic in water. It destroys the exclsuion zone in water and cause avalanche collapse of electrical signaling. As a result of this release of calcium, this means more NADH and less NAD+ is made at cytochrome 1. Calcium activates TCA cycle dehydrogenases, which also means more NADH and less NAD+. The cell is making boatloads of NADH which leads to boatloads of ROS. The ROS made sits right next to mitochondrial DNA stores in cells. That ROS destroys the mitochondrial DNA that make your cytochrome proteins. Here

you can see why cancer and low NAD+ levels walk hand and hand all the time.

When quantum tunneling is affected on the ETC, so are the ways protons are handled in mitochondria. This ultimately results in the inhibition of the mitochondrial transcription factor (TFAM) which inhibits the expression of mitochondrial DNA which is located right next to cytochrome one. As a result of this "shift", mitochondria can not, and do not produce the proteins encoded in mitochondrial DNA that are required for "forward electron transport" and normal ATP production. When this happens proteins can not be unfolded maximally to allow water to behind these proteins for proper energy transfers. All proteins must have proper hydration cells to work well or they get marked to be taken out (apoptosis) earlier than they should and this further zaps energy from a cell with an already lowered redox potential. The same thing is true of mitochondria within these cells. They must have water surrounding them to transfer the potential energy stored from protons to the hydration shells surrounding mitochondria. That energized water is charged separated by the infra red heat generated by our cells (the reason why mammals are really warm blooded) to create a large EZ to power voltages surrounding the inner mitochondrial membrane to send electrons from foods to oxygen for cellular respiration. When they do not have the ability to go from cytochrome 1 to 4 normally, reverse electron transport occurs to make ATP and deliver electrons to reduce oxygen and deliver protons potential energy to intracellular water. As a result of this circuitous path for the subatomic particles, and high levels of free radicals are produced in ROS and RNS. These signal the 911 molecules in the cell and deliver a nasty package to the nucleus. This all leads to the exact picture we see in early aging and neolithic disease generation in humans. Now think Alzheimer's , Parkinson's disease, heart disease, atherosclerosis and eventually cancer. This is how cancer happens in human biology. It is due to a chronic seasonal

progression of a loss of redox power over decades in a cell that eventually catches up to us when we have lost enough energy. In this way, you begin to see each disease we have a plateau in the loss of the redox potential as time elapses.

WHY KETOSIS MAKES SENSE WITH A LOW REDOX/DELTA PSI

In this metabolic scenario, cells are dependent on aerobic glycolysis in the cytoplasm and cannot burn fat well in theor mitochondria to make ATP. Cytochrome 2 is their bail out when this occurs because they can no longer generate cytochrome proteins from their own mitochondrial DNA in those tissues or cells. Cytochrome 2 is not altered by this epigenetic metabolic shift in cells. Cytochrome 2 is where fats normally enter the ETC in a healthy situation. This is why fats can still be handled well in low redox states. It also shows you why ketosis is protective in cancer and neurodegenerative states. This is why ketosis is protective when this "shift" occurs. These cells develop the exact metabolic picture of cancer, well known as the "Warburg effect." The Sinclair paper shows that mitochondrial dysfunction and this Warburg-type metabolism are fully reversible with the supplementation of NAD+ precursors (niacin). Ketosis also happens to replenish NAD+ for the nucleus! Ketosis is protective because it allows passage of electrons and protons from fats (FFA) to make some ATP and salvage the bad mitochondria they possess and keep them from oncogenesis. Ketogenic diets have also been shown to aid deep sea divers and special force soldiers overcome situation where they face chronic hypoxia. Ketogenic diets help prevent brain cancer and they treat it best. Read this hyper link. There are many reports that a sick brain does not really use glucose but changes it to lactate which neurons love to use as a fuel with fats. The humans brain does like fats and lactate, but guess what also likes lactate and fats? All other tissues in your body do. So when you eat fat you will not get cancer that tries to metastasize to your brain to steal its mitochondria that love ketones and makes gobs of

oxygen. (Big wisdom point here) Lactate usage by the brain appears to be the best way of postponing apoptosis (cell suicide), short of abandoning your brain mitochondria altogether. Glucose use comes in as a second choice in the brain. This is why brain cancer seeks out new mitochondria in its local environment and does not have to travel to find it in far away tissues. This is why brain cancer almost always stays inside the cranium!!!

We need to think of food as hormone information, not as a metabolic fuel to begin to grasp the science of cancer. Food captures photons and electrons from the sun. Molecular Maxwell demons in foods capture the sun's potential energy first. Molecular Maxwell demons take advantage of the fact that the Second Law of thermodynamics is statistical and not absolute. In this way, the electromagnetic force of sunlight electrically induces the Molecular demons in our mitochondria to capture that potential energy in electrons and the protons of water around our mitochondria. Water is the molecular adapter of the potential energy transfer in this quantum dance. Water is 100% to the cells redox potential. In our mitochondria is where the potential energy of the sun is unlocked from our food and water, and it is turned back into photons and electrons that we can use within our quantum cells. This is a text book definition of how the First Law of thermodynamics operates. Potential energy can be transformed into something to do work in a cell. Energy can not be made or destroyed, but it can transformed. Water is our molecular quantum chameleon.

Molecular demons in cytochromes steal the energy in photons and electrons excited by the sun and turn then back to their lowest energy state in molecular oxygen in our mitochondria. Molecular demons captures this change in energy in an energy free manner using the cytochrome proteins as its "baseball glove", and couples it to water chemistry allows the transfer to happen from food to the cytochrome proteins. This quantum

dance happens in your inner mitochondrial membrane to reduce entropy system wide.

In QED, in fact, even in Newton's physics, as time goes on, entropy increases in any system. In Newton's physics time was considered absolute and non varying. Einstein's relativity made us all realize time was not absolute either. It was relative to who was measuring it. This implies just the act of observation can throw reality off. Quantum systems are supposed to be destroyed by the act of measurement, which brings them abruptly into the ordinary classical world. This is another reason why today's scientists and clinicians might have cause and effect all wrong for cancer. What we believe now in cancer, might not be that important as we all think. As hard as this is to fathom for some, it is a consequence of nature. This one observation has had a huge effect on subatomic physics, but it has not, yet, had any effect on oncology or biology, or its researchers. They seem to act to believe "their science" is exempt from this basic physical fact. I decided to rethink that truth long ago.

It should have caused them to consider the implications of this decision, because the business of biology happens at a molecular level in a cell, not just at a chromosomal level. Mina Bissell is now the lone researcher with the megaphone screaming this to them. Will they listen? I don't know, but I know it no longer matters to me what they believe. I see things a lot differently than most now.

Entropy in life, is not chaos, it is information stored in the quantum particle. In brain cancer, we lose the ability to reduce entropy and chaos is the result. Think of this analogy for brain cancer. Think of a vase in your hands, and then you drop it and it smashes into trillions of pieces. Each piece is made up of subatomic particles. Each subatomic particle is a fragment of the glass vase the "great architect" dropped in the Big Bang 14 billion years ago. Each piece forms a piece of what was the vase. Life is designed to capture photons and

electrons to gain this energy and information for free to reduce entropy in the entire system to animate life. **This is exactly what drives evolution, in my opinion, not natural selection.** We evolve better entropy dumps back to the environment to become more energy efficient. As we improve, hormone panels reflect this efficiency in our mitochondria. Today we do not truly understand what a hormone panel means in medicine. I showed you what I think it means, in the cancer process above.

As information is collected as a life unfolds, living tissues comes aware….and conscious within its cytoarchitecture and then its protoplasm. With more time and embryologic development it is then shared with its brain. Mitochondria is what links the body to the brain in all things tied to energy and information from our environment. Ever see a two ice dancers dance together? Body coherence/connection due to conscious entanglement. As brain develops on the tree of life it becomes entangled with body. The brain becomes the librarian who can sample the consciousness throughout the entire life form. Better brain, better cognition, to tap more information to collect more energy and data. Hormones = energy + information. Electrons, protons, and water is are life's vehicle. When this system is broken, cancer can result. This blog shows you precisely how it can happen.

PHYSICS GEEKS: When a photon interacts with a material particle on our globe (think food) it lifts one electron from an electron pair to a higher 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 in mitochondria allowing us to utilizing its excess energy for life processes. In this sense, you can begin to see how

living organism are organized. The physics of a cells organization dictates how biology will unfold. This is exactly what Mina Bissell has said about cancer, but today, I am using my words and not hers to explain how it works in brain cancer.

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. 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 likely evolved. It allowed animals to live disconnected from the Earth and sun for period of times. This was not afford 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. 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 thought of a system of 'excitable media', tissues, or organs called excitable cells poised to respond specifically and disproportionately to weak low electromagnetic signals from its environment. Cells are built to decipher these signals using the Calcium/calmodulin system in neurons that couple all processes to circadian biology. Electromagnetic signals are the strongest forces in our universe because they have this unusual ability to bind the smallest ***CHARGED*** particles in nature. Because large amounts of energy stored everywhere in cells and tissues, they automatically amplify these weak electromagnetic signals, to often cause macroscopic actions. In brain cancer, we lose all these abilities. This is the simplified version to give you the essence of what I wrote in Energy and Epigenetics 6. What loads the entire system with energy and information, if

you are following me? Sunlight does it 100% of the time. This is why I had a huge problem with the conversation I began the sunshine of your life blog with. Re read that one.

It is the basic electromagnetic force that binds all life forms. We eat electrons and photons from the sun and collect their energies and information in our cytochromes……y'all just do not know it yet, but you will by the time I am done teaching. Go back and read how I began this post in paragraph 2. Still think I might be off?

THE TAKE AWAY FOR THE NON GEEKS: Dehydration, calcium efflux, and glucose which all deplete NAD+ in the nucleus. These set of events all lead to cancer in the brain. Our modern world provides that perfect storm 24/7. This sets the table for brain cancer to begin and take hold. We can no longer settle for a science that is not going to harvest the answers we need for our modern health conditions. I firmly believe the answer won't be found in biochemistry but in QED. Paradigms change slowly……because when dogma is deep, it reveals our ignorance of reality and shows how it rules our beliefs. In my opinion, the essence of leadership in medicine is being able to see the iceberg before it hits the Titanic.

As Mina Bissell would say, what good is your literature if your target is wrong?

CITES:

1.https://www.cell.com/abstract/S0092-8674(13)01521-3

2.http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1299287/

3. Jacobs RA, Díaz V, Meinild A-K, Gassmann M & Lundby C (2013). The C57Bl/6 mouse serves as a suitable model of human skeletal muscle mitochondrial function. Exp Physiol 98, 000–000.

4. Kuznetsov AV, Veksler V, Gellerich FN, Saks V, Margreiter R & Kunz WS (2008). Analysis of mitochondrial function in situ in permeabilized muscle fibers, tissues and cells. Nat Protoc 3, 965–976.

5. Larsen S, Nielsen J, Hansen CN, Nielsen LB, Wibrand F, Stride N, Schroder HD, Boushel R, Helge JW, Dela F & Hey-Mogensen M (2012). Biomarkers of mitochondrial content in skeletal muscle of healthy young human subjects. J Physiol 590, 3349–3360.

6. Murray AJ (2009). Metabolic adaptation of skeletal muscle to high altitude hypoxia: how new technologies could resolve the controversies. Genome Med 1, 117.

7. Palmer JW, Tandler B & Hoppel CL (1977). Biochemical properties of subsarcolemmal and interfibrillar mitochondria isolated from rat cardiac muscle. J Biol Chem 252, 8731–8739.

8.Miller, A.K., Alston, R.L. and Corsellis, J.A., Variation with age in the volumes of grey and white matter in the cerebral hemispheres of man: measurements with an image analyser, *Neuropathol Appl Neurobiol.*, 6:119-132, 1980

9.Kandel et al., Principles of Neural Science, New York: McGraw Hill, 2000

10.Aghababian, R., *Essentials of Emergency Medicine*, 2006

11.http://caloriesproper.com/?p=3995

12.http://www.ncbi.nlm.nih.gov/pubmed/11569918

13. http://www.ncbi.nlm.nih.gov/pubmed/12228253

14.http://www.ajnr.org/content/23/8/1334.long

15.http://www.ncbi.nlm.nih.gov/pubmed/20600789

16. http://www.ncbi.nlm.nih.gov/pubmed/21915942

17. http://www.ncbi.nlm.nih.gov/pubmed/2120033

18. http://www.economist.com/news/science-and-technology/21598944-sloppy-researchers-beware-new-institute-has-you-its-sights-metaphysicians

19.http://usatoday30.usatoday.com/news/health/2006-05-14-diet-treatment_x.htm (Parkinson's disease diet)

20.http://www.amazon.com/Thomas-N.-Seyfried/e/B006M7MT86

21.http://www.sciencedirect.com/science/article/pii/S0306452214000645 (SIRT1 and circadian linkage)