ORGANIZATIONAL STRUCTURAL FAILURE # 5 PRIONS ARE COMPLIANT DESIGN FLAWS

READERS SUMMARY

1. How do prions cause disease?

2. How does light in the brain work?

3. Why red light is critical in the brain?

4. How does Fronto-temporal dysplasia and Gliobastoma multiforman link together from a prion point of view?

5. How do all these neurologic and mental diseases link directly back to the gut?

Today in part two of our prion blog, you are going to learn the single most important function of life's design using quantum biology is shape adaptation in both proteins and in DNA. Shape change dictates tissue optics and tissue optics are all tied to the proper thermodynamic view of life. The reason now should be intuitive. Any changes in geometry alters energy profiles and physiologic function. The innate ability to alter protein geometry in real time enables nature's machines to operate with the utmost efficiency. Cells and tissues allow us to morph proteins to allow life to happen properly. When we do it incorrectly, disease and death emerges. It is not hard to understand……it is just hard to believe.

We do it by altering atoms using electrons and photons to move their shapes. Electrons and protons are made from leptons and

quarks. Nature builds enormous complexity via 6 leptons & quarks by evolving into a chemical evolution with the construction of atoms. This allows life to apply compliant design to all its systems of action. A compliant mechanism is a single-piece flexible structure that delivers the desired motion by undergoing elastic deformation as opposed to the rigid body mutations in a conventional evolutionary mechanism. Compliant evolutionary design is elastic because it utilizes the power in electrons and protons to alter mass and energy equivalence to morph its own structural characteristics and meet demand. Elasticity is another way of saying cellular metastability. Metastability is a function of the addition or subtraction of electrons and photons in proteins. DNA only codes for proteins. Proteins are "composite boson-like" modules that electrons and photons can change by rearranging their atoms. The less moving parts life needs to assemble, the more reliable control we have on a weight to power ratio. This allows us to perform work on the smallest stages with as little as energy as possible. Weight to power ratios scale to how cells solve life's thermodynamic challenge.

WHAT ELSE DID PRUSINER FIND?

Prusiner found experimentally that **beta amyloid plaques** in Creutzfeldt-Jakob Disease (CJD) and Gertsmann-Straussler-Scheinker Syndrome (GSS) both contained the same PrP as scrapies and Alzheimer's disease (AD). It turned out that PrP antibodies he made to interact with PrP in scrapie did not interact with Alzheimer's beta amyloid even though they had the **exact same** molecular structure. This made it clear that PrP was not a non specific response to brain injury as the critics had said. In fact, in Alzheimer's, it is the Alpha /beta prion peptides that join together to form AD amyloid. Immunostaining studies showed that amyloid CJD and AD plaques *stained differently,* but were also the **same** chemically. Congo red, along with other histologic stains, also showed that the

plaques in AD and CJD were, in fact, very different. *This implied that there was a specific change in each protein that was* ***independent of molecular structure.***

Prusiner still has not figured out **why** they are different even today. His work showed the disease **was not genetic** and was, in fact, *"emergent".* This should have been a clue that the addition of small quanta of energies might have led to the massive physiologic changes in these proteins. This was hard for science to swallow for 30 years, but today it is well accepted. PrP^c is a 27-30 kDaltons length protein that was the critical normal protein template common to both diseases.

The ability to change the power or energies of small proteins is the critical force that turned us from ape to human. This very same protein can morph into a new protein and lead us to extinction. Today, it is the loss of power that is robbing us of our humanity. A power loss causes the prions to shift from an alpha helix to beta sheet due to electron/photon loss. The result is autoimmunity and neuro-degeneration of the proteins related to the MHC 1 gene.

In physics, energy is power, and power comes in many different forms. The quantum mechanism provides proteins with small quanta of energy that can lead to massive alterations in function. This is where evolution gets its power to change and adapt proteins for good or bad. Proteins are the basic unit of what the matter is, and what we are fundamentally made of. This is where evolution starts and ends within each one of us. In order to see the true effects of evolution, we need to stop looking at "morphologic changes" in things we study and start looking at proteins in 3-D molecular simulations as we add and subtract quanta of energy. This is where quantum evolution begins to control biological trajectory in all species.

3 million years ago, PrP^c was working for our benefit to construct a new brain and immune system. Today, it is *the*

cause of our demise. Its alteration in confirmation is a physical process based upon the addition or subtraction of a quanta of energy delivered to or from the protein by the modern environment's electromagnetic spectrum. This exchange of energies is what causes the beta amyloid sheet confirmation to emerge, and that small change leads to emergent disease generation. Since the mechanism is physical, it requires no input from DNA or RNA. This offends modern beliefs in biology. I find that favorable because most new ideas are radically different.

This implies proteins coded or uncoded for by nucleic acids actually direct the arrow of time in biology. In physics, time is invariant. Time is created because of things created to be metastable or dissipative in nature. Such systems rely totally upon nonlinear protein/quanta interactions that can form their own mass, spatial, and temporal structures. These aspects of proteins are synonymous to the "dissipative structure" of a cell. These cells can exist as long as the system is continuously held far from equilibrium due to a continual flow of energy or matter through the system. Autophagy keeps us in this state. This means the organization of the cell is far more important than the things that *feed our metabolism*. Anything having these qualities has to be kept far from equilibrium to maintain a healthy life. Only at death, is equilibrium obtained. I view the arrow of time and the inability of reforming the shell of a cracked egg as playing a constructive role in nature. The arrow of time is essential to the existence of biological systems because they contain highly organized irreversible structures. In other words, the cell's organization is the critical part of life, not the fuel we put into it. Once you lose this organization time becomes your enemy in a quantized world. Nature builds enormous complexity via 6 leptons & quarks by evolving into a chemical evolution via the construction of atoms. It is the arrangement of these atoms that dictate the life we live.

This allows “emergent physics” to develop new proteins, which can improve upon the cell’s ability to handle chaos and generate more energy to drive new complexity. This quantum mechanism can move backward and forward because the laws of physics allow for it. This means that if it destroys cell structure, such that a cell loses mass and swells, it will lead to illness. Today, humans find that to be the case.

Some changes will drive evolution progressively, while others result in new diseases that show up out of thin air. Prion diseases are a classic examples of diseases that break all the rules and just show up out of no where. These proteins once helped us evolve from chimp to human as they helped sculpt our neocortex to become more complex. Today, they are working against us because the environment they are working within is changing the quanta that energizes their amino acids. In this way, many of yesterday’s diseases become solutions under the physical laws of nature. Again, evolution is a physically quantized process, not a biological one. This is why Prusiner’s work was, and is still, so offensive to people like Taubes.

LIGHT IN THE BRAIN

There was another critical difference in Prusiner’s experiments using colored stains that highlight the optical changes and quanta effects of light in the brain. Initially, Congo red dye studies and histo-pathologic staining were indistinguishable from the CJD and Alzheimer’s diseases Prusiner studied. **Immuno-staining with antibodies, however, told a different story.** The plaques had a very allosterically different optical specificity basis. This told the reader of Prusiner’s data that the difference between CJD and AD amyloid would not be found in its molecular formula, but in its quantum energy profiles of the bent proteins in the brain. This is what caused Prusiner himself to look more closely at

the optics of animal models he developed. He noticed early on that his transgenic mice *were emitting light* from their brains **before** prion disease manifested.

He found that the Congo red dye bound to purified prion rods and exhibited a green gold birefringence. This tells us there has been a dramatic change in the optics of light within the brain tissue. Today this science is called optogenetics. Congo red is used as a pH indicator in a lab. This means excessive Congo red staining of the brain tissue in prion diseases tells us this tissue has a massively altered water exclusion zone. Pollack's work has shown us that water loses its exclusion zone (EZ) at high salinity and/or low pH. Heavy Congo red staining in prion disease is a marker for a poor water EZ in the brain. ***It turns out all mis-folded prion proteins show this very basic physical effect with lowered voltages on EEG's***.

BOOM

<u>WHY RED LIGHT IN THE BRAIN IS OPTIMAL:</u>

I want you to remember what I told you in OSF 3 about the 'red giant star" and how a normal brain works in parallel fashion. In OSF 3, I showed you how red light optimizes mass equivalence for energy production in a star. Red stars that are colder emit less energy in the form of light because they are more thermodynamically organized. Since they have a better organized core, they emit red photons to their surroundings. Red light does not carry as much power as blue or white light. These stars are more energy efficient thermodynamically, and their lifespans are much longer. Red giants can remain energy efficient for trillions of years because they are very stable in how they balance their mass energy equivalence.

Now consider your brain's use of red light. Melatonin is destroyed by blue light and restored by red light. Blue eyes

are more common further from the equator because they have more melanin to absorb more light. A blue iris in an eye is due to Tyndall scattering in a turbid layer in the iris. Brown and black irises have the same layer except with more melanin in it. The melanin absorbs light better to improve vision. So blue eyes evolved where light photons do not have as much power. Color has a big impact on the physics of optics.

Did you know that the water in you brain's microtubules absorbs photons best in the lower powered red photon band?

Carefully consider the "red giant star" analogy above with respect to the prion laden brain emission of green gold light. Optically, they are polar opposites. A normal brain should be optically optimized to release red photons that are polarized down our microtubules precisely the same way a red giant does it in space to live long. This is how synapses are strengthened in the brain by long term potentiation. Blue light weakens synaptic connections.

Quantum electrodynamics is able to fully explain the effective weakening of the electromagnetic charge with distance. This become very important in the human brain. The electric charge carried by an object (think DHA) is a fixed and definite quantity. When a charge is surrounded by other freely moving charges (think water), however, its effects may be modified. If an electron enters a medium composed of molecules that have positively and negatively charged ends (water is the perfect dipole), it will polarize those molecules. Polarization is a property of waves that can oscillate with more than one orientation. When electrons are captured on DHAs, they will repel the water molecules' negative ends (oxygen) and attract their positive ends (hydrogen end), effectively screening itself in the positive charge. The result of the polarization is to reduce the electron's charge in the brain to allow light to take control of the neural network. In an electromagnetic wave, such as light, both the electric field and magnetic field are oscillating, but in different directions. Both the

electric and magnetic fields are perpendicular to the wave's direction of travel. This allows the white matter tracts, filled with microtubules and water, to act like magnetic flux tubes in the brain. The oscillation of these fields may be in a single direction (linear polarization), or the field may rotate at the optical frequency (circular or elliptical polarization). In that case, the direction of the fields' rotation, and thus the specified polarization, may be either clockwise or counter clockwise. This is referred to as the wave's chirality, or handedness. This change in prion disease is manifested in the green gold birefrigence change mentioned above. The polarization process always involves the use of a charged object to induce electron movement or electron rearrangement.

Polarization can occur within insulators (think myelin or topologic insulators), but the process occurs in a different manner than it does within a semiconductor. In a semiconducting object, electrons are induced into movement across the surface of the conductor from one side of the object to the opposite side. In an insulator, electrons merely redistribute themselves within the atoms or molecules nearest the outer surface of the object. To understand the electron redistribution process, it is important to take another brief excursion into the world of atoms, molecules, and chemical bonds.

The electrons surrounding the nucleus of an atom are believed to be located in regions of space with specific shapes and sizes. The actual size and shape of these regions is determined by the high-powered mathematical equations common to quantum mechanics. Rather than being located a specific distance from the nucleus in a fixed orbit, the electrons are simply thought of as being located in regions often referred to as electron clouds (think DHA). At any given moment, the electron is likely to be found at some location within the cloud. The electron clouds have varying densities. DHA has a

very dense pi electron cloud because of its 6 allylic double bonds; the density of the cloud is considered to be greatest in the portion of the cloud where the electron has the greatest probability of being found at any given moment.

Conversely, the electron cloud density is least in the regions where the electron is least likely to be found. In addition to having varying densities, these electron clouds are also highly distortable by electric and magnetic fields. The presence of neighboring atoms with high electron affinity can distort the electron clouds around atoms. This is why transition metals are tightly coupled to DHA. PrP^{c} is that coupling agent in the brain.

Rather than being located symmetrically about the positive nucleus, the cloud becomes asymmetrically shaped. As such, there is a polarization of the atom as the centers of positive and negative charges are no longer located in the same place. This is how shape change occurs in lipid rafts and proteins. As the shape changes, the energy characteristics of the atoms can radically change. If the atom is still a neutral atom, it has just become polarized.

The discussion becomes even more complex when we consider molecules (combination of atoms bonded together). This is what is happening in the brain. In molecules, atoms are bonded together as protons in one atom and they attract the electrons in the clouds of other atoms. This electrostatic attraction results in a bond between the two atoms. Electrons are shared by the two atoms as they begin to overlap their electron clouds. If the atoms are of different types (for instance, one atom is Hydrogen and the other atom is Oxygen), then the electrons within the clouds of the two atoms are not equally shared. The clouds become distorted, with the electrons having the greatest probability of being found closest to the more electron-greedy atom. The bond is said to be a “polar bond”. The distribution of electrons within the cloud is shifted more

towards one atom than towards the other atom. This is the case for the two hydrogen-oxygen bonds in the water molecule.

Electrons shared by these two atoms are drawn more towards the oxygen atom than towards the hydrogen atom. Subsequently, there is a separation of charge, with oxygen having a partially negative charge and hydrogen having a partially positive charge. This is how charge separation of water is maximized in a microtubule to generate maximum energy. This is where Pollack's work should resonate for you. In a cell, infrared heat does this trick, but in the brain the polarization of light does the trick. In prion diseases, the loss of the polarization of light causes massive energy failures that first lead to rapid cognitive decline, but eventually lead to death. All other diseases that affect this mechanism cause disease on a continuum and lead to their classic symptoms. I've mentioned them in OSF 3, 4, and now here.

It is very common to observe this polarization within molecules. In molecules that have long chains of atoms bonded together (like DHA), there are often several locations along the chain, or near the ends of the chain, that have polar bonds. This is another reason that, at synapses, we see massive infusion of iodine and PrP, which controls copper ion flow. Iodine is the one halogen that can give up its 7 electrons to the pi electron cloud of DHA to increase the electric field, leaving behind a large nucleus with a positive charge. The reason is because iodine's atomic nucleus is so big it limits its control over these electrons in the strong electric fields created by DHA. Copper can add or subtract electrons to proteins with its 4 valence electrons depending upon the redox situation around the neuron. This allows for forward progression or reversal of flows in neural circuits during wakefulness and sleep.

This "optical polarization" leaves the molecule with areas that have a concentration of positive charges and areas with a

concentration of negative charges. This principle is utilized in the manufacture of certain commercial products that are used to reduce static cling when you wash clothes. Your brain uses iodine and copper to reduce this static cling in your adjacent neurons to maintain its DC current flow. This is where Becker's DC current begins. This charge maintains the brain's optical network by not allowing it to collapse.

The centers of positive and negative charges within a regular product are drawn to excess charge residing on the clothes. There is a neutralization of the static charge buildup on the clothes, thus reducing their tendency to be attracted to each other. During manufacturing, a thin sheet is soaked in a solution containing positively charged ions. The sheet is tossed into the dryer with the clothes. Being saturated with positive charges, the sheet is capable of attracting excess electrons that are scuffed off of clothes during the drying cycle, reducing the static cling.

Perhaps the biggest misconception that pertains to polarization is the belief that polarization involves the charging of an object. Polarization is not charging! When an object becomes polarized, there is simply a redistribution of the centers of positive and negative charges within the object. Either by the movement of electrons across the surface of the object (as is the case in conductors) or through the distortion of electron clouds (as is the case in insulators), the centers of positive and negative charges become separated from each other.

Polarization is an important parameter in areas of science dealing with transverse wave propagation, such as optics, seismology, radio and microwaves. Especially impacted are technologies such as lasers, wireless and optical fiber telecommunications, and radar. Here you can begin to see why non native EMF waves can easily disrupt the brain's native polarization.

The process of polarization is often used in many charging methods. Your brain uses light and water to recharge its DHA network. This is why the brain can work on just 20 watts of power.

The most common optical materials (such as glass) are isotropic. They simply preserve the polarization of a wave, but do not differentiate between polarization states. There are, however, important classes of materials classified as birefringent or optically active in which this is not the case. A wave's polarization will generally be modified or will affect propagation.

Water in the brain acts as an anisotropic polarizer. Liquid crystals are examples of anisotropic liquids. Water can be ordered to be a liquid crystal when it is bound to proteins in hydration shells. Anisotropy is the physical property of being directionally dependent, as opposed to isotropy, which implies identical properties in all directions. Water controls the flow of currents using this physical property it contains when it is in a liquid crystalline form. Hydrogen and oxygen alone do not have this ability, so anisotropy is an "emergent" property of water molecule. Life takes massive advantage of this. Prion diseases destroy this mechanism in the brain and the immune system. This is why they are so clinically devastating when they occur.

Water is an anisotropic filter. It is used to filter electrons and photons in the brain. It is a filter built with increasingly smaller interstitial spaces within the decreasing caliber of microtubule diameters in the white matter tracts of the brain in the direction of the DC current. The action occurs so that the proximal regions filter out larger particles and distal regions remove smaller particles, resulting in greater flow-through and more efficient development of current or power. This is how a laser gains its power by focusing the power of photons. Anisotropic lasers are extremely efficient in creating 3D memory

holographically. In prion diseases, memory is destroyed because tissue optics in the brain are destroyed.

Diffusion tensor imaging is an MRI technique that involves measuring the fractional anisotropy of the random motion (Brownian motion) of water molecules in the brain. Water molecules located in fiber tracts are more likely to be anisotropic, since they are restricted in their movement by microtubules. Water molecules dispersed in the rest of the brain have less restricted movement because the water is outside microtubles bound to proteins and therefore display more isotropy.

It also turns out that cosmic background microwave radiation maps exhibit anisotropy throughout, providing the same pattern that the human brain has.

MACROCOSM MEETS MICROCOSM

Here, the quantum blueprint of a red giant's lifespan can be scaled and reproduced in our brain if the PrP^{c} is working well. If PrP^{sc} is present in brain tissue, this cannot work because the optics of the brain are thermodynamically ruined. The same exact mechanism that is built into a red giant is built into your brain. The foundations of it all are quantized.

Prion diseases show that this normal red light is not retained in the brain's tissue because Congo red stain is avidly bound to amyloid prions. When this tissue is looked at under a microscope's light and is polarized, the tissues are birefringent with green/gold colors. Moreover, this color change is associated with vacuolization in the brain. Vacuolization is evidence of an increase of mass and loss of energy in neurons. It is associated with this color change.

Color change is also indicative of protein shape change. Here you see mass equivalence working its thermodynamic brew.

Prions directly change the optics and density of brain tissue. When the optics are altered, so are the tissues' ability to transform energy from electrons to photons in microtubules.

When Congo red stain was used in AD and kuru infected brains, it produced a deep red stain under the light microscope. Congo red binds avidly to the amyloid fibrils in both diseases. When polarized filters were added to the scope, the amyloid displayed characteristic birefringence in green and gold. This signifies a low pH and high salinity in cellular water, as well as a poor EZ in the brain. Here you see Prusiner's and Pollack's work merging. Both of these effects lower energy transformation of water inside the micotubules.

The story gets worse for energy transformation because the optical orientation of amyloid deposited in blood vessels from subjects having cerebral amyloid deposition is 90° out of phase from that in the plaques, suggesting that the fibrils run tangentially with respect to the circumference of the blood vessels. This optical change also limits transformation of energy from photons to water. This means the brain is losing the optical energy it relies on for cognitive function and memory when it transfers energy from electrons to photons in the brain. *This implies that size, shape, and orientation of the fibrils dictate the disease onset and rapidity of how it overtakes a person.*

It also meant that researchers should have been able to **see light loss** from the surface of the brain in animals with protein prion diseases. PrP seems to act as a rectifier of optical information in neurons. This is akin to what Becker found in bone with respect to copper atoms. The copper ions in bone acted as a Josephson junction (J-J). Copper and iodine act as a J-J junction in the brain as well. In neurons and synapses, the weak link can consist of a thin insulating

barrier known as a superconductor–insulator–superconductor junction (S-I-S)(iodine), or a short section of non-superconducting metal (S-N-S) (copper), or a physical constriction that weakens the superconductivity at the point of contact (S-s-S) (node of Ranvier or synapse).

In the physics of semiconduction, J-J is used as a frequency-to-voltage converter with a conversion factor equal to 2e/h. The adopted value of 2e/h is 483593.420 GHz/V(NBS).

This relationship is critical for the photoelectric effect in the brain to work optimally. Prusiner never realized this because he is not a physicist. Taubes, however, was a physicist. He never realized its critical importance before he threw the baby out with the bath water in Prusiner's work.

Prusiner never realized the importance because he did not understand the quantum mechanism built into proteins. He didn't understand their shape change with the addition or subtraction of electrons. This situation is akin to what occurred to Max Planck pre-Einstein circa 1905. Planck should have discovered relativity because he found the key point in Planck's constant, but he did not. Einstein, however, did understand its relevance, and he innovated the Theory of Relativity.

Ironically, Prusiner *did show experimental bio-luminescent loss in all prion diseases* in his transgenic mice. Today, he and other neurobiologists have remained in the dark about the significance of what this implies with respect to red light energy loss in the human brain.

Once again, you see that a loss of optics correlates with energy loss and loss of cognitive power. ***Lady evolution has re-created the thermodynamics seen in a red giant and placed it in your skull.*** It is not an exact replica, but it works on the same thermodynamic principles of metastability. Your cells, brain, tissues, stars, and the cosmic microwave

background radiation are all metastable. Here we see fractal design.

Transgenic mice with A/beta amyloid deposits were shown to emit light from their brains even before clinical disease was present. They began measuring light emission from the brain through the skull during prion replication. This showed that light was released several months before any signs of neurologic disease manifested. The same findings have been reproduced in mice with Alzheimer's plaques. This was also reproduced in brain homogenates in transgenic mice in a Parkinson's disease model, which showed light was emitted by a brain littered with the prion alpha-synuclein. It turns out Lewy bodies turn into alpha-synuclein prions when the local redox potential is chronically lowered. This leads to Parkinson's disease. Shy Drager disease is also associated with alpha-synuclein prion accumulation. The difference in both is their size, length, and shape.

When the normal PrP^{c} in a Lewy body is transformed by addition or subtraction of quanta from any cause, the Lewy body becomes a disease causing prion (PrP^{sc}) because of an alpha to beta sheet transition. This was found during autopsy in these animals.

These proteins are also found in all humans with these diseases.

So how do these proteins form in us to cause disease?

When DHA semiconduction is broken in the brain for any reason, the normal PrP^{c} becomes ionized to form PrP^{sc}. This change allows all neuro-degenerative diseases to manifest by altering the shape and length of this protein. It is critical to remember that Prp^{c} prion is made from all of the known 20 amino acids that occur naturally in humans as a normal membrane bound protein. This protein molecule consists of 3 alpha

helices (type H) and 2 anti-parallel beta-pleated sheets. Scientists have observed and named the alpha helix 1 (H1) , alpha helix 2 (H2) , and alpha helix 3 (H3) of the human prion protein. There are also two shorter type-G helices called G1 and G2. Their loop diameters are significantly smaller than type H helices. There are also 2 anti parallel beta sheets called B1 & B2.

The Prp^{c} protein is normally found in the neocortex, but PrP^{sc} is found in all brains with prion diseases. In fact, it is used to direct normal neocortical migration and function. From this normal amino acid template, it seems to act like a DNA or RNA chaperone protein. All prion diseases transform when quanta is added or subtracted under the power of native electromagnetic forces acting upon the PrP^{c}. If redox power is lost, the result is an abnormal PrP^{sc}. This is another example of how the quantum mechanism, which you learned about in OSF 3, innovates or extinguishes physiologic abilities within the neocortex as it evolves. The mechanism does not require the use of nucleic acid chemistry at all. This protein was critical in forming the human brain from the chimp brain without much direction from our nucleic acids. This is why chimp DNA and human DNA are close to identical, but the capability of their brains are vastly different.

When humans move away from diets deep in DHA, they move away from their best way to collect electrons to avoid PrP transformation to PrP^{sc}. DHA allows us to collect, assimilate, and direct electrons in the proper neural circuits of the brain. DHA metabolites also form resolvins and protectins that are anti inflammatory by nature. This means they allow for more electrons and less protons. It means it keeps the delta psi of cell membranes and mitochondrial membranes robust wherever they are found. In a person without DHA, the redox

potential is destroyed, so no resolvins or protectins are present. They also control macrophage transition in the immune system to keep Nfr-2 pathways quiescent. When these chemicals are missing, neuro-degeneration and neuro-immune diseases explode. This is all tied to the innate ability of the electron to build cellular complexity in the brain and immune system by changing the ionization and redox potential of PrP. The small amount of quanta in electrons carries a large swing in power for the conformational shape changes in these prions.

The 1997 Nobel winner also went the extra step of definitively showing that alpha/beta amyloid prions stimulate the transformation of normal tau proteins into a prion that can spread all over the CNS *without the help of any nucleic acid*. This mechanism blows Darwin and biology's Central Dogma to hell. The same results were found in rabies, post-encephalitic Parkinson's disease, and Niemann Pick disease. In familial Alzheimer's and ALS, they even showed that mutant human CJD prions induced large numbers of neurofibrillary tangles.

We also know that as tau proteins lose electrons and become oxidized, they become ***more dense*** because they have more protons than electrons within them. This immediately *changes their shape and length* to form beta sheets and lose their alpha conformation. We also know that when water loses its normal dielectric constant (think Br^- or F^-), alpha sheets transform to beta sheets because of the massive shifts in the magnetic moment of the PrP^c protein. *When electrons are lost for any reason at all, the dielectric constant drops and the magnetic moment of the prion protein changes dramatically.* When a magnetic moment or field is lost on a protein or a planet, energy drops. This drop also causes alpha sheets in prions to transform to beta sheets. **This is where neuro-degeneration and autoimmunity begin in all cases.**

Normal magnetic fields around Earth connect and disconnect,

explosively releasing energy to and fro in a process known as magnetic reconnection. This can accelerate electrons to nearly the speed of light and release them to the system. In our brain, this energy is released to water in the smallest parts of microtubules below 1.4 nm in diameter. The same action occurs around prions, but on an atomic scale. The dielectric point of water is critical to a healthy life. The dielectric constant measures how effectively the cell or protein in question shields the negative and positive charges from one another. It reduces the force between them. Water is a major constituent of living systems, and it is an excellent dielectric facilitator because of its large effective magnetic dipole moment. A biological cell is a dissipative system by its very nature. A dissipative system is a thermodynamically open system which is operating far from thermodynamic equilibrium in an environment with which it constantly exchanges energy and matter. This implies it has the purpose to break symmetry and create a metastable system to react to all environmental possibilities that the cell may face. Water is the molecule that allows a cell to break symmetry. Water is confined in microtubules to maximize its ability to transfer energy to the surrounding neurons.

This effect also scales to the Earth because of all the water it has on its surface. The planet has its own dipole moment because it has a magnetic field. What many do not appreciate is how light and the magnetic dipole of the Earth scales to your cellular level. When we lose electrons, we lose our magnetic moments on proteins, causing prions to transition from alpha helices to beta sheets.

This can occur in multiple ways depending upon how electrons are lost in the system. When the redox potential is low, DHA is slowly degraded to protect the cell. If it is not replaced, a deficit results in cell membranes and organelles. In the neocortex, we use DHA to capture electrons from the environment. At the brain’s surface, it couples the magnetic

moment to the water interface to transfer energy to the neocortex below. If this system is altered in any way, it won't work well, causing the redox potential to fall. The Prp^{c} is normally found in the neocortex, but when electrons are sparse for any reason, energy drops. When a prion is present, this energy drop becomes constant and quickly builds to a crescendo across the entire system. This is why it mimics an "infection" but has no evidence of immunologic up regulation. This redox change facilitates the transition from the normal Prp^{c} to the Prp^{sc}, which can cause many different forms of neurologic prion disease based upon how the shape, length, and mass of the PrP protein is altered.

Water molecules can surround the ions in solution and effectively neutralize charges just like the magnetosphere does to sunlight in space around our planet. In prion protein transitions, this change in water structure is altered, and these transfers cannot happen naturally. The critical event in the pathogenesis of transmissible spongiform encephalopathies is the conversion of the prion protein from an alpha-helical form, PrP^{c}, to a beta-sheet-rich conformer, PrP^{sc}.

Recently, it has been shown that incubation of the recombinant prion protein under mildly acidic conditions (pH 5 or below) seriously alters water structure facilitating beta sheet formation. Acids, a lower pH, or excessive protons cause the exclusion zone (EZ) of water to become smaller, diminishing the water's ability to transfer energy within the CSF as well.

Water has a normal magnetic dipole moment of 1.85 Debye. Polypeptide chains in the α-helical configuration normally have enormous dipole moments. They are upwards of 500 Debye because the individual moments of the peptide bonds are all aligned properly. Beta sheet amyloids do not have a large dipole moment because they are smaller in size and shape and their bonds are not aligned properly. This improper bond alignment is what changes the protein length, shape, and mass.

This is why energy is lost on the neocortex. This is why cognition suffers and disease ensues when the beta sheet predominates.

It turns out that beta sheet transformation is directly linked to protein size, length, and mass. Here we scale back to Einstein's mass equivalence equation yet again.

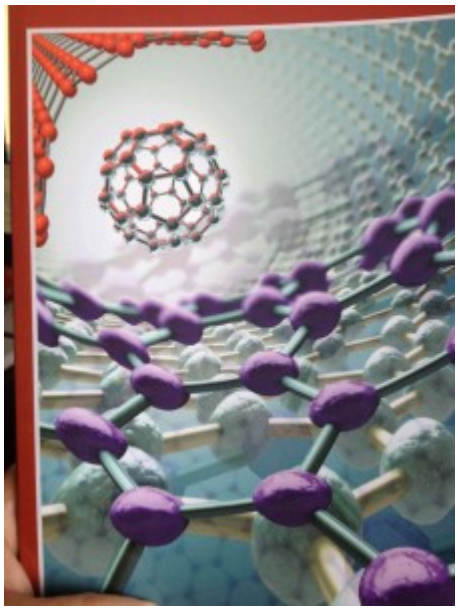
TAU PROTEINS AND FRONTO-TEMPORAL DYSPLASIA

When tau proteins lose electrons, they change shape and cause diseases at earlier ages. In fronto-temporal dementia (FTD), more and more causes of shape change show up daily. Six other tau protein prions have been found to cause FTD. FTD diseases include progressive supranuclear palsy, corticobasal degeneration, Pick's disease, and argyrophilic grain disease. Each are caused by a separate tau prion that has an identical molecular formula but carries a different magnetic moment and energy profile. Two prion proteins associated with FTD most commonly are TDP43 and FUS. They are critical in forming the prion that causes FTD. These two proteins are RNA binding proteins that are loaded with glutamine and asparagine amino acids like those seen in ***yeast proteins*** that form prions. If you have SIBO or candida in your gut, you better pay attention to this.

NFL Hall of Famer John Mackey died from FTD. I believe Michael Jackson died of a *yeast-like prion disease* because he also suffered from vitiligo (an AI) and poor sleep. Since he was unable to sleep well, he could not clear abnormal tau proteins in his brain because, without sleep, there is poor autophagy. This alters the optics of the brain to result in bizarre behavior over the last 25 years of his life. This is why I believe they buried him without his brain and repeated his brain autopsy twice.

HOW SHAPE CHANGE CAUSES PRION DISEASE

Our neocortex is un-myelinated and is supposed to be filled with DHA to capture electrons from our environment. It is designed to act as the perfect antenna for the native electromagnetic force on Earth. When the environmental non native electromagnetic force is increased in our atmosphere, it lowers the energy our neocortex senses, and its energies alter proteins normally found in our neocortex. This altered PrP^{c} is a 27-30k Dalton. It becomes smaller in size,length, and mass. It is cleaved at the N terminus based upon the energy in the environment. The smaller the Prp^{c} 27-30 is cleaved, the more aggressive the prion-like disease becomes. Once prions accumulate in the neocortex, they cause disease by spread along the microtubules. This spreading of the prion proteins simulates an infection. This is why so many cases of neuro-degeneration, mental illness, and brain gut disorders appear to be infectious on the surface. They are not. The mechanism of how they occur is why this appearance seems familiar. These proteins block water from filing the smallest spaces in microtubules and energy is lost. I went over how water generates massive energy in the brain microtubules in OSF 3. All prions are small enough to fit within the microtubules of the brain to affect the magnetic dipole of water. This diminishes energy transfer to the neurons mentioned above.



Prion protein in a microtubule

Microtubules diameter below the neocortex goes from 30 nanometers and narrows to 15 nanometers and ends in spaces below 1.4 nanometers. The more water is restricted in space the more energy it is capable of generating in the brain. This energy is sent to neurons, white matter, to DHA electrically and magnetically. It is evenetually sent over collagen and fascial network throughout the cell. These small prions block water from entering the critical parts of microtubules below 10 nanometers.

As the prions get smaller in size they cause more aggressive disease because water transfers its energies best when confined in the smallest spaces in microtubules. When water can not enter the smallest spaces, electrons can not fall back to their ground state and release red light to the brain to energize it optically. Most quantum energy transfers with water occur optimally when water molecules movements are restricted to 1.4 nm in a microtubule.

When the prion amyloid plaques are present it forces electrons

to unload its photons at a different energy state in the green/gold range in space where water is not confined or present. If water is absent we see vacuolization due to dissipation of energy due to a lack of coupling. These photons do not carry the energy that the red photons do, and cognitive function slowly declines. As red light declines steadily the redox potential is assaulted and declines further fast forwarding the process. As electrons are chronically lost autophagy efficiency declines and more prions persist and neurologic decline quickens. In this way it simulates an infectious process without the immune system activation.

Normally autophagy can go on indefinitely and get rid of mis-folded proteins if the redox potential stays high. The redox potential is 100% tied to electron collection and assimilation. As the redox potential declines, DHA levels are lowered in all neural circuits, cell membranes and mitochondria. DHA is the only lipid capable of collecting electrons. If you do not out supply DHA in your brain from seafood compared to match your losses from oxidation, disease ensues as the ratio of electrons declines compared to protons.

Autophagy constantly recycles and refurbishes mitochondria to work well and give us longevity when electrons are plentiful. This implies that the same principles in a red giant are built within us, but keeping this system up and running requires continuous electron input. The key to mitochonria constantly recreating themselves using autophagy is maintenance of their redox potential. The redox potential is 100% tied to how many electrons are delivered to the inner mitochondrial membrane. This is why mitochondria input uses electron chain transport to create energy and signaling molecules.

In the normal functioning brain this process is “pruned” daily by autophagy and the the ubiquination pathways. In patients who are afflicted by excessive non native EMF’s are constantly losing electrons from their brain’s mitochondria. The result of electron loss is excessive generation of Fenton reaction

hydroxyl free radical and nitric oxide.

Recall that OSF 3 had the key mechanism in this game plan:

“Mitochondria use electron losses to signal things to a cell over its collagen cytoarchitecture network. A one-electron reduction of oxygen forms superoxide (0_2-), a two-electron reduction forms hydrogen peroxide (H_2O_2), and a three-electron reduction forms the hydroxyl radical (-OH Fenton free radical).

Today, the dominant paradigm in the process of aging is that antioxidants decrease aging. This paradigm vilifies superoxide as the card carrying cause of disease and aging. There is a big problem, however. Superoxide is associated with health and longevity and not disease. Why? Superoxide acts not as a destructive molecule, but as a protective signal in our bodies, turning up the expression of genes that help to repair cellular damage during autophagy. Why? Because we have a special enzyme in us called superoxide dismutase 1 (SOD 1) that protects us from one or two electrons loss normally. **We have no protection when we lose more than two electrons from electron coupling.** When we lose more than two electrons, voltages change in a cell (called lower delta psi) and calcium is released from voltage gated channels. This is where electron steal syndrome because very destructive. If it continues chronically, NMDA receptors become active and more calcium is released to cause widespread peroxidation of all cellular components by the -OH free radical and nitric oxide.

So why does **non native EMF** cause us a massive loss of autophagic efficiency? (-OH and Nitric oxide release)

Reactive oxygen species (ROS) and reactive nitrogen species (RNS) play important roles in regulation of cell survival. In general, moderate levels of ROS/RNS may function as signals to promote cell proliferation and survival, whereas severe increase of ROS/RNS can induce cell death. Under physiologic

conditions, the balance between generation and elimination of ROS/RNS maintains the proper function of redox-sensitive signaling proteins.

When **we lose three or more electrons** our mitochondria begin make Fenton reactions due to the more positively charge mitochondrial membrane that lowers our charge (delta psi) that lead to the generation of massive amounts of nitric oxide and cause calcium release from calcium voltage gated channels initially. Nitric oxide begins to quickly accumulate due to massive electron loss and it increases peroxynitrite production (PRN). Peroxynitrite (sometimes called peroxonitrite) is an anion with the formula $ONOO^-$. It is an unstable structural isomer of nitrate, NO_3^-. PRN is a potent oxidant that steals electrons from proteins. As this fast forwards due to the unending assault of non native EMF and calcium release within a cell, PRN stimulates massive oxidative stress by activating NF-kappa Beta. Review CPC #8 to see how this cause massive intracellular failure by increasing mitochondrial size in neurons activating cytochrome c, which is also known as cytochrome #4 in the mitochondria. When a mitochondrial swells it is a sign of energy inefficiency within it because electrons are being lost and more protons are being generated.

PRN can not be cleared well when the intracellular charge is lost and it quickly increases the production of iNOS which can, in turn increases more nitric oxide (NO) release. When enough NO is released NMDA receptors become leaky to calcium and mitochondrial failure occurs because cytochrome 4 is activated to undergo cell suicide. Cytochrome c (cytochrome 4) is a small-molecular-weight heme-containing protein, which participates in an electron transfer from complex III to complex IV in mitochondrial electron-transport chain. It's mode of action is very similar to the membrane bound prion PrP^c. Because of the nature of cytochrome c as a reversible electron donor/acceptor, this protein is highly redox-

sensitive. Cytochrome c locates in inner mitochondria membrane and interacts with the membrane phospholipid cardiolipin. This has a lot of homology with PrP mechanistically. It appears to be self similar design at work. The release of cytochrome c is considered a hallmark of mitochondrial-mediated apoptosis. When it is activated it allows calcium to rush inside the mitochondria to cause it to increase its mass and swell.

Under physiologic conditions, cytochrome c plays an essential role in oxidative phosphorylation and production of ATP, which allows for water binding to occur on protein side chains. Dr. Gilbert Ling experiments were exquisite in showing this mechanism of action.

Generation of superoxide in mitochondria occurs mainly through electron leakage from complex I and complex III. Therefore, cytochrome c, which accepts an electron from complex III and donates it to cytochrome c oxidase, also functions to prevent the electron outflow and superoxide generation. Because of this function, cytochrome c plays a critical role in keeping ROS/RNS; Generation at a low level is optimal for cell survival normally.

Besides its function as an electron carrier, cytochrome c may act as a peroxidase. During the cell-death process, the released cytochrome c in the cytosol binds and exerts a peroxidase activity on plasma membrane lipids, especially phosphatidyl serine, leading to lipid peroxidation. This structural modification of the plasma membrane can lead to increases in cell mass, that expose the cell to signals recognized by the macrophage to engulf the cell corpse and eliminate it.

When this loop is up-regulated it constitutes a potential vicious cycle and there are a number of other loops activated in the NRF-2 pathways. The Nrf2-signaling pathway mediates multiple avenues of cytoprotection by activating the transcription of more than 200 genes that are crucial in the

metabolism of drugs and toxins, protection against oxidative stress and inflammation, as well as playing an integral role in stability of proteins and in the removal of damaged proteins via proteasomal degradation or autophagy. Nrf2 interacts with other important cell regulators such as tumor suppressor protein 53 (p53) and nuclear factor-kappa beta (NF-κB) and through their combined interactions is the guardian of healthspan, protecting against many age-related diseases including cancer and neurodegeneration

The NFR 2 pathways activate NMDA calcium receptors which bypass normal autophagy recycling that salvages mitochondria and instead force cells to undergo apoptosis and they die. If this goes on long enough you deplete yourself of stem cells and your life force. When this depletes stem cells you are left with cells that have poor functioning mitochondria that have undergone the redox shift I mentioned in EE 12. The redox shift signifies a loss of electrons, DHA, and loss of cysteine with in the proteins that make up your cells framework. Alterations in how your cells are made is at the root of all disease.

The worse the depletion of DHA and cysteine in cell membranes, the faster the decline of mitochondrial efficiency, the faster the cell turns senescent. This occurs in metabolic syndrome quite often. If it goes on chronically, oncogenesis is favored because the p53 gene becomes altered by a chaperone protein to change its physiologic role.

In this way, cancer can also be thought of as an "*emergent disease*" due to protein mis-folding: Where have you heard that before? #CPC 8.

BRAIN CANCER: GBM = Glioblastoma multiformans

If this redox assault continues, the p53 gene is altered by a very small prion protein acting like a chaperone protein and

aggressive oncogenesis can result in the brain. The **most aggressive brain tumor is GBM** and it has been associated with the generation of a small prion protein. This is a tumor I deal with all the time. It is devastating for the patient. GBM shows up quickly and is ultra aggressive. In other words, this form of brain cancer is emergent, shows up out of the blue and kills swiftly. We are powerless as neurosurgeons with this tumor because we are not thinking outside the box in how to deal with the quantized prion that causes it.

Researchers at Karolinska Institutet have discovered that a prion substance called Vacquinol-1 makes cells from glioblastoma, literally explode into more GBM cells throughout the cerebral hemispheres. There mechanism of action also follows microtubules as other prions do. Today in humans, the average survival is just 15 months for GBM. This tumor is now more common that it was 50 years ago when modern technology was being developed.

GBM was not linked to a "***prion like mechanism***" until recently. I went over many of the molecular details in brain cancer in CPC #8. It was found that the prion protein molecule gave the cancer cells an uncontrolled vacuolization, a process in which the cell carries substances from outside the cell into its interior. *Vacuolization is seen in every known prion neurodegenerative disease in humans.* This carrying process is made via the vacuoles and transferred across neurons and synapses to rapidly and locally invade the brain. This process can roughly be described as blisters or bags consisting of cell membranes within the neurons that is serially spread and infected to all parts of the CNS via microtubules. The process is similar to what was behind the recent 1997 Nobel Prize in physiology for medicine on prions. The discovery that describes how cellular vesicles move things from the interior of the cell to its surface where the collagen network becomes altered by electron loss or a loss of redox potential. This vacuolization increases a cell mass and causes energy levels

to drop and death to come fast. This is clear evidence that mass equivalence is under direct assault using the quantum mechanism found in OSF 3.

This mechanism is involved in autism, AD, PD, FTD, concussion, CTE, ALS, schizophrenia, PTSD any many others……like multiple sclerosis. Yes, Multiple Sclerosis is also a myelin vacuolization disease similar in many respects to NF-1 and NF-2.

You might be shocked to find out this exactly mechanism is found in yeast and is transmitted via the gut via the vagus nerve to the brain in the fourth ventricle via the CSF.

Yes……..this is how the King of Pop really died. Candida (a yeast) uses this exact quantum mechanism in the brain gut axis.

So if you have candida in your gut you can bet your environment is loaded with non native EMF that causes a spike of Fenton reaction to stimulate massive uncontrolled amounts of nitric oxide production in your gut. This is what also happens in many cases of SIBO with respect to organisms that should not be in your gut. This can alter the normal PrP in your gut's immune system's cell in the GALT. Once this protein transformation happens in the gut the proteins access your brain directly via the microtubules in your vagus nerve to enter area postrema and your CSF to cause their global effects. This is how a gut issue can get through the blood brain barrier. It never has to penetrate it as most believe because it follows microtubules in the neurons. It is also why many gut issues are associated with changes in cognition. This is why many mental illnesses appear like brain infections to cause mental illness, neuro-degeneration, and autoimmunity. Counter intuitive, but then again things that work by a quantum mechanism often are, like photosynthesis.

Become aware of what you might not know. It might save your life.

Science works by iteration but biology works by quantum mechanisms which favor N=1 locality. It has to make you ask yourself is scientific progress declining on a massive scale because we are missing this key link?

Is life currently dying faster for some reason no one seems to see?

CITES

DebBurman, S., Raymond, G., Caughey, B., and S. Lindquist. 1997. “Chaperone-supervised conversion of prion protein to its protease-resistant form.” Proceedings of the National Academy of Science USA. 94: 13938-13943.

Haire, L.F., Whyte, S., Vasisht, N., Gill, A., Verma, C., Dodson, E., Dodson, G., and P. Bayley. 2004. Journal of Molecular Biology. 336: 1175-1183.

Jones, C. E., Abdelraheim, S., Brown, D., and J.H. Viles. 2004. “Preferential Cu 2+ Coordination by His 96 and His 111 Induces B-Sheet Formation in the Unstructured Amyloidogenic Region of the Prion Protein.” Journal of Biological Chemistry. 279(31): 32018-32027.

Kaneko, K., Zulianello, L., Scott, M., Cooper, C., Wallace, A., James, T., Cohen, F., and S. Prusiner. 1997. “Evidence for protein X binding to a discontinuous epitope on the cellular prion protein during scrapie prion propagation.” Proceedings

of the National Academy of Science. 94: 10069-10074.

Layne, E. Spectrophotometric and Turbidimetric Methods for Measuring Proteins. Methods in Enzymology 3: 447-455. 1957.
Stoscheck, CM. Quantitation of Protein. Methods in Enzymology 182: 50-69. 1990.

Knaus, K.J., Morilla, M., Swietnicki, W., Malone, M., Surewicz, W., and V. Yee. 2001. "Crystal Structure of the human prion protein reveals a mechanism for oligomerization." Nature Structural Biology. 8: 770-774.

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2850414/

Guyer RL. Prions: Puzzling Infectious Proteins. NIH – Research in the News. (http://science-education.nih.gov/nihHTML/ose/snapshots/multimedia/ritn/prions/prions1.html)

Taubes G. The Game of the Name is Fame. But is it Science? Discover. December, 1986. Reprinted at: http://www.slate.com/id/2096/sidebar/42786/.

Prusiner SB. (1995).The Prion Diseases. Scientific American. (http://www.sciam.com/article.cfm?articleID=0009FD80-C3C6-1C5A-B882809EC588ED9F&pageNumber=1&catID=2)

Whitmore L, Wallace BA (2008). "Protein secondary structure analyses from circular dichroism spectroscopy: methods and reference databases". Biopolymers 89 (5): 392–400. doi:10.1002/bip.20853. PMID 17896349.
Greenfield NJ (2006). "Using circular dichroism spectra to estimate protein secondary structure". Nature protocols 1 (6): 2876–90. doi:10.1038/nprot.2006.202. PMC 2728378. PMID 17406547.Prusiner SB. (1982). Novel proteinaceous infectious particles cause scrapie. Science. 9;216(4542):136-44.

Legname G, Baskakov IV, Nguyen HB, et al. (2004). Synthetic Mammalian Prions. Science. 7;305:673-676.

Johnston N. (2004). Clearing Hurdles: Prions Know How to Do It. The Scientist. 18(11):18. (http://www.the-scientist.com/yr2004/jun/feature_040607.html)

Bastian FO, Foster JW. Spiroplasma SP. (2001). 16S rDNA in Creutzfeldt-Jakob disease and scrapie as shown by PCR AND DNA sequence analysis. J Neuropathol Exp Neurol. 60:613-620.

Aguzzi A, Polymenidou M. (2004). Mammalian Prion Biology: One Century of Evolving Concepts. Cell. 116(2):313-327. (http://www.sciencedirect.com/science/article/B6WSN-4BJK582-H/2/70143387edac888b362f04571b6a7988)