

TENSEGRITY 2: CORTISOL = POWER OF AM SUNLIGHT

READERS SUMMARY:

1. How does cortisol tie to a cell's mass equivalence?
2. How does cortisol work daily in a human to wake us up?
3. How are the effects of cortisol stopped by AM sunlight?
4. How does cortisol link to NAD^+ and circadian gene expression?
5. How does cortisol link circadian biology to mitochondrial handling of electrons in the cytochromes?

The circadian cycle of cortisol is critical in understanding optimal energy balance. Cortisol directly alters a cell's mass and volume. Mass is directly tied to energy balance by $E=mc^2$. When you understand circadian biology, you get a much more complete picture of how the system works on a 24 hour basis. It turns out electrons control the coupling of biochemistry in life and understanding this helps making sense of why hormones become disrupted when electrons are not handled correctly. I became a student of circadian biology when I saw the entire view from a 30,000 foot level.

Our brain wakes up with a morning surge of cortisol. That is what turns our brain on at 6AM. VIP helps do this in long light cycles. VIP is highest at 6 AM and lowest at 6 PM when light levels are low. Leptin sensitivity directly regulates VIP production. VIP regulates the circadian rhythm and entrain the SCN to light. When it is cold, leptin is released from fat cells in large amounts, and we begin to use e-NOS to entrain

our SCN to cold cycles and we should avoid carbs like the plague then. In the late night (4AM) when temperature levels are lowest, cortisol begins to rise in the blood while leptin levels in the blood are falling. Leptin levels peak in the blood at midnight to 2 AM. They are lowest in the morning when cortisol is rising fastest.

It seems cortisol action in the morning then must be yoked to sunlight for a reason. What might that reason be?

Just think for a moment about the involuntary isometric muscle contraction stretching we do in the morning after awakening. We see cats and dogs do it as well. They stretch out and contract every muscle in their bodies it seems. Does this isometric muscle contraction produce an energizing "let's get rolling" signal to the entire body from the mechanically induced piezoelectric effect? It does seem to me from my own N=1 that it does produce an energy jolt to get things going. If not piezoelectric in nature, what biochemical purpose does this activity serve?

Might it control an ultradian 12 hour cycle of our proteins?

When you rise what happens? Cortisol spikes in the AM. What does cortisol do? It makes collagen helices swell slightly because it unzips part of it by removing electrons from it.

What does this do? It cause cells to swell because they become more loose in their tensegrity system and lose their electric and magnetic charge. This process is usually self-limited, by your own redox state, but if it is low to begin with it can lead to more serious medical problems over time.

This process is lost in cancer states. This is why more heart attacks occur in the AM than any other time in the day as well. No one in medicine has a clue why this happens but this is why it does. It is a piezoelectric effect in collagen gone awry. Getting the AM sunlight on your skin builds a massive EZ inside your blood vessels to create a huge flow of

protons within the center of your blood's plasma to augment flow. Protons recycling in the mitochondrion is also a critical simultaneous step. This helps you decrease your AM blood pressure and make water from foods. Sunlight lowers your blood pressure and re-zips your collagen architecture.

Fundamentally this is why dermatologists are clueless about the sun's benefits, especially in the AM. The reason they think the sun is bad is because they do not realize they people in their waiting rooms all have a low redox state.

This is when the dose response curve of sunlight might hurt you.

Why is morning stretching done by humans and most other mammals? To contract the more loose collagen and thereby send the piezoelectric signal in it to the autophagy programs in us to re-tighten the tensegrity system during the day. It is like wearing a compression garment.

When you tighten up your muscles, what does this mean to Einstein's mass equivalence equation? As cell mass/volume decreases, we become more energy-efficient, when it enlarges light energy is lost. In the late afternoon, the changing diurnal variation of the days sun stimulate protein synthesis in controlled fashion. So you might ask what re-zips our collagen in the AM? We use the "massless energy" to re-zip the collagen to a more tight confirmation like a zipper.

The answer: The sun's AM light is the key stimulus; this occurs by way of the dawns photons....photons can do what electrons do.....the photoelectric effect has the power to re-zip collagen that cortisol unzipped and lower your cell mass and control volumes carefully. Staying inside all morning is not a good answer for humans. Wearing clothes is a big problem too. This creates a circadian mismatch and does not allow the power of light to re-zip your collagen.

As we get those photons over the day we keep tightening our tensegrity system and become more energy-efficient. This is

why the best time to work out is late afternoon. At 5 PM humans show their greatest cardiovascular efficiency allowing for maximal exercising or hunting. This also occurs during a time when we have our best rates of protein synthesis in our body. This is why exercise should optimally be done in this window. It increases protein synthesis in us 200-400% because cortisol levels are lower and cell volumes are better controlled. The mass equivalence equation then becomes more favorable to our ubiquitination and autophagy programs and we conserve even more energy.

You heard this long ago in point 7 in this blog: I just never mentioned why back then. Back then, you knew little about the photoelectric effect. Now you do.

In the very same blog, I later wrote this: To show you how important meal timing to the light cycles are to us humans, consider these facts about exercise and meal timing. If you can yoke your workout to your evening protein dinner meal (within 30-45 minutes) you actually "triple the amount of protein synthesis" that occurs compared to those who do not. This is how a hunter gatherer attained their ideal body comp without having to do huge amounts of exercises. Moreover, if this is also yoked to the light cycle in winter when the temperatures are below 40 degrees, you can **increase protein synthesis to 400%** while inducing uncoupling proteins to burn fat at a tripled rate to baseline. This allows you to increase fat burning to shred body fat further faster while you are increasing lean muscle mass. Using fat also facilitates proton recycling in our mitochondria. Circadian timing is more important than the food we eat.

NASA uses this optical technology for the astronauts that space walk to maximize their metabolic efficiency in space. The problem is in space is cortisol is always raised and it causes massive optic nerve swelling. There is no grounding in space. The physics of space imply you cannot ground yourself in space. These conditions have massive effects on cell size

in space. These conditions of existence imply there would always be a potential difference; there would always be electric currents present in space and the ships humans are in; there would always be induction currents; these currents would always have massive dynamical swings because of the lack of protection from the magnetosphere and this is what leads to cell volume instability and circadian and ultradian mismatches.

This is why space ages humans. Mitochondria hate this situation because of their bacterial origins. The cell cannot equalize the charges in space so the mitochondria constantly swells – it's just the way it is in this environment. Humans are adapted to Earth not space. This is also why prokaryotes grow massively in space when NASA has tested it. Astronauts face something more daunting simultaneously too. Astronauts in space see 19 sunrises and sunsets per day so they can not use the sun as we do to rezip collagen and shrink their brain and decrease their intracranial pressure. Spacewalks are yoked to sunlight to offset the risk. The other problem is that the sunlight in space is much brighter than it is on Earth and this can damage their retina. Life is not designed to leave this planet because it was organized on Earth's surface. That reason is tied to how our mitochondria fundamentally work.

CORTISOL LINK TO THE EPIGENOME

If you're sick from stress, research reports appearing in the August 2012 issue of *The FASEB Journal* suggests that what your mother ate – or didn't eat – may be part of the cause. The report shows that choline intake that is higher than what is generally recommended during pregnancy may improve how a child responds to stress. Foods with choline tends to be high in cysteine which is loaded with electrons. These improvements are the result of epigenetic changes that ultimately lead to lower cortisol levels. Epigenetic changes affect how a gene functions, even if the gene itself is not changed. Lowering cortisol is important as high levels of cortisol are linked to

a range of problems ranging from mental health to metabolic and cardiovascular disorders.

Cortisol removes electrons from collagen to decrease its electric charge. This decreases its piezoelectric potential. This diminishes its ability to act as a semiconductor. Cortisol also removes electrons from a fetal and newborn genes in humans. The removal of these electrons causes a change in the epigenetic methylation status of a human gene called NR3C1.

Methylation status of the human NR3C1 gene in newborns is sensitive to prenatal maternal mood and offers insight just how electron withdrawal can cause massive alterations to epigenetic processes that links ante-natal maternal mood/stress to directly alter HPA stress reactivity during infancy of her child. This is why I believe space travel will induce methylation defects and up-regulate protein synthesis, expansion of the subcutaneous fat mass, while sarcopenia increases, and the more chronic the altered light frequencies remain, will eventually lead to cancer. I believe this is the key mechanism to space diseases and all mitochondrial disease in modern Earth. I also think these mitochondrial changes in the brain and body clocks are active in **eating disorder development**. Moreover, if the defect occurs in the hypothalamic region of the fetus or child, the disease will manifest in the first three decades of life when our mitochondria should be in its best shape. This is why eating disorders are signs of mitochondrial damage.

Elaborate on the connection of methylation and acetylation to cortisol:

These are the two major pathways that are working in DNA/RNA epigenetic changes. Methylation pathways are highly dependent on water chemistry in the cell. Another layer of complexity are SNP's in the MTHFR genes; histone acetylation is critical part of epigenetic signaling from the beta oxidation of animal fats in mitochondria. These signals, along with the

intracellular redox state are critical in turning the epigenetic expression of genes on and off at the correct times. They are both intimately tied to the metabolic shift that occurs in mitochondria with respect to which cytochrome is being fed in mitochondria. If you are a carbophile you are mostly delivering food electrons to cytochrome 1, while depleting yourself of NAD⁺ there. Excessive carbohydrate use is also linked to excessive cortisol levels. If you are eating animal fats you are primarily delivering electrons to cytochrome 2, while creating a lot of NAD⁺ there. It turns out NAD⁺ are critical in epigenetic regulation pathways. NAD⁺ activates sirtuins, a family of deacetylase enzymes. SIRT1 happens to regulate the activity of BMAL1 and CLOCK, two circadian transcription factors. This is how your epigenetic switches are first altered. It also turns out that CLOCK and BMAL1 enhance SIRT1 expression. Moreover, genetic deletion of any of these players induces insulin resistance. IR also is critically linked to abnormal cortisol levels as you saw in the hormone 101 and 102 blog posts.

Most people think genes cause disease today. It is just a belief and it is not a 100% truth. I think 85% of the diseases of aging are mitochondrial. The altered expression of your genes by the environmental changes is the real problem. The signature at any time of your redox state tells you about diseases you already own that you do not know about, like silent cancers. This is where DNA methylation comes in, a process by means of which sites next to genes on chromosomes, called promoter regions (dark matter of the genome), are chemically methylated after a cycle of DNA replication.

<http://genesdev.cshlp.org/content/16/1/6.full>

Histones are spindle molecules in a cell's nucleus around which DNA is wrapped; they play important roles in gene activation and expression. Histones are made active or inactive when the tensegrity system surrounding the nucleus is altered. What is the signal? The volume change brought about

by the loss of collagen cytoarchitecture. Here is where cortisol plays another role in stress signaling. Histone acetylation is a chemical modification of a portion of a histone which leads to selective unwrapping of the DNA making the exposed genes amenable to activation and expression. Histone deacetylation is the opposite. It is done by histone deacetylases and wraps up the DNA making the associated genes unreachable by activating proteins and therefore less amenable to expression. Gene transcription is repressed. Histone deacetylase inhibitors prevent the actions of histone deacetylases, that is, they keep the DNA unwrapped and available for gene expression. The enzymes controlling the state of histone acetylation in vivo are histone acetyltransferase (HAT) and histone deacetylases (HDAC). HDAC enzymes catalyze the removal of acetyl groups from the amino-terminal lysine residues of core nucleosomal histones. The result is gene silencing. Exactly how the HAT and HDAC enzymes work today is complex and only partially understood because people remain unaware of how the addition and subtraction of electrons from these proteins alters their function. The magnetic moments of these proteins appears to be even more important in embryologic development because this is when genes are turned off and on many times rapidly in key sequences to form a species.

There is a whole mammalian HDAC gene family and a corresponding HAT gene family. Patterns of histone acetylation are part of the epigenomic history of a cell.

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2248733/>

Your epi-genome knows much more about your medical history than your doctor or you will ever know about you. In fact, this science is telling us our "zip codes" are more important than our genetic code in our nucleus. You'd be wise to brush up on that science if you want to avoid guys like me. There is a simple analogy to make the point how important epigenetics is. If your computer breaks down do you toss it out or do you

just fix the software? You fix the software to recover the function of the laptop. It is more economical. Biology works the same way because the second law of thermodynamics is how cells are organized. Managing energy properly is critical function of the epigenome. This is why leptin controls fecundity and oocyte selection in all mammals.

By being able to write a genome (DNA/RNA) and plug it into the cell of organisms, the software or epigenome, if you will, changes the hardware when the environment calls for it.

Medicine and researchers are clueless about this today. Nutrition researchers even more clueless.

Most have no clue how biochemistry creates and manages energy. It is quantized and all controlled by the redox potential and the epigenome.

You never hear that from them, do you?

Why?

They think they know the answers of how cells generate energy because they read biochemistry books that told them ATP hydrolysis is where the energy comes from.

One small problem with their belief.

The ATPase in their biochemistry books breaks the Second Law of thermodynamics by a wide margin.

What does that mean?

Any thing that breaks the second law is a falsehood.

Feynman said when your experiment does not meet your theory, no matter how smart you are, or how elegant your beliefs sound to the masses.....they are wrong.

Bread crumb alert: The medical and ancestral community does not know any of this because they do not understand how the work of Ling or Pollack directly tie to how biochemistry

proceeds.

They know even less about how the 3 legged stool of nature works in quantized fashion.

Living in your epigenome is the history of our species. Mother Nature has provided us a spectacular epigenetic toolbox to recreate wellness from illness. The toolbox exists in our DNA but is expressed in a much more sophisticated pattern. This is where methylation and acetylation play their roles. Proper circadian control of cortisol is incredibly important in these systems. Cortisol works in quantized fashion.

The genome is the first draft of what we might be, but our life experience is the final draft and is made by the choices we made in our the time we were given based upon the electronic induction of these proteins. An architect far better and smarter than us has given us that epigenetic toolbox, and we now have the ability to use it by altering our behaviors to change our lives. We just need to understand how choices marry to electrons. That is the story being written by modern epigenetics. It will soon be codified in quantum biology.

Cortisol wakes you up every AM because it unzips collagen by removing electrons. The effect is designed to be short lived until you are out in the sun under the power of sunlight photons that replaces those lost electrons to re zip collagen.

Cortisol makes your cells swell and increase its mass. Sunlight binds you cell and lowers its mass. Cortisol raises blood sugar. When cortisol is released from the PVN the more immediate players in circadian biology is the coenzyme nicotinamide adenine dinucleotide (NAD). Cortisol lowers NAD⁺.

NAD⁺ participates in a variety of redox reactions that control you circadian cycles. NAD activates sirtuins, a family of deacetylase enzymes (think SIRT 1). This is fundamentally how epigenetic switches are altered by circadian mismatches.

Cortisol's connection to NAD⁺ is a big missing piece for alternative medicine and allopathic medicine.

SIRT1 regulates the activity of BMAL1 and CLOCK, two circadian transcription factors, which target NAMPT, an enzyme that synthesizes NAD⁺.

Fasting increases the intracellular NAD⁺/NADH ratio, setting off a cascade of events involving epigenetics and regulation of metabolism. Cortisol's ability to remove electrons from collagen plays a major role in altering your epigenetic programs that control your genes. Few people understand how this dance happens. Cortisol can both help or hurt you depending upon the context its release from your PVN happens.

For example, to generate memories in the hippocampus it requires the initial swelling in those neurons to allow the process to begin. The swelling however, is self-limited when the person is healthy with a good redox. When someone is not well and has a poor redox they can not generate the first surge of cortisol, or the initial surge surpasses the normal pathway and does not turn off and a bunch of neural connections are made that can not be wired properly to form a lasting memory. This require optimized melatonin and BDNF.

Neither work well when the BMAL1 and CLOCK genes have been altered by low levels of NAD⁺ or abnormal cortisol secretion from the PVN. When the process works by evolutionary design, it can lead to a person with a great working memory. When it does not, it can form a path work array of connections that make no sense. This is what happens in schizophrenia.

Moreover, rapid excessive cortisol release in the hypothalamus of a young child can lead to anorexia or hypothalamic amenorrhea and cause massive circadian mismatches to cause life long alterations. Understanding how cortisol can open the door to heaven or hell is critical in the quantum health model.

CITES

X. Jiang, J. Yan, A. A. West, C. A. Perry, O. V. Malysheva, S. Devapatla, E. Pressman, F. Vermeulen, M. A. Caudill. **Maternal choline intake alters the epigenetic state of fetal cortisol-regulating genes in humans.** *The FASEB Journal*, 2012; 26 (8): 3563 DOI: 10.1096/fj.12-207894

<http://www.ncbi.nlm.nih.gov/pubmed/18536531>

- <http://www.nature.com/nature/journal/v226/n5252/abs/2261261a0.html>
- Moore JG, Halberg F. Circadian rhythm of gastric acid secretion in active duodenal ulcer: chronobiological statistical characteristics and comparison of acid secretory and plasma gastrin patterns with healthy subjects and post-vagotomy and pyloroplasty patients. *Chronobiology International*. 1987;4:101-110.
- Moore JG, Merki H. Gastrointestinal tract. In: *Physiology and Physiology of Biological Rhythms*. Peter H. Redfern and Bjorn Lemmer, Editors. Berlin, Springer, 1997, pp.351-373.
- Haus E. and Smolensky M.: Development of circadian time structure and blood pressure rhythms. In: Portman R.J., Sorof J.M., Inglefinger J.R. (eds). *Clinical Hypertension and Vascular Disease: Pediatric Hypertension*. Humana Press Inc., NJ, pp 45-73, 2004.
- Haus E., Nicolau G., Lakatua D.J., Sackett-Lundeen L.: Reference values for chronopharmacology. *Ann. Rev. Chronopharm.* 4:333-342, 1988.
- Rivkees S.A. and Hao H.: Developing circadian rhythmicity. *Seminar in Perinatology* 24(4):232-242, 2000.
- Sara Mednick , Ken Nakayama, Jose L. Cantero, Mercedes Atienza, Alicia A. Levin, Neha Pathak & Robert Stickgold (2002). The restorative effect of naps on perceptual deterioration. *Nature Neuroscience*, published online May 28, 2002

- Aeschbach D, Matthews JR, Postolache TT, Jackson MA, Giesen HA, Wehr TA (1999). Two circadian rhythms in the human electroencephalogram during wakefulness. *American Journal of Physiology*, 277, R1771-9
- Meneses Ortega S, Corsi Cabrera M (1990). Ultradian rhythms in the EEG and task performance. *Chronobiologia*, 17, 183-94
- Rector RS, Uptergrove GM, Morris EM, Borengasser SJ, Laughlin MH, **Booth FW**, Thyfault JP, Ibdah JA., Daily exercise vs. caloric restriction for prevention of nonalcoholic fatty liver disease in the OLETF rat model., *Am J Physiol Gastrointest Liver Physiol*. 2011 May;300(5):G874-83. Epub 2011 Feb 24. PMID:21350190
- Bjorntorp P. 2001 Do stress reactions cause abdominal obesity and co-morbidities? *Obesity Reviews* 2: 73-86.
- Cota, D et al. 2006 Hypothalamic mTor signaling regulates food intake. *Science* 312:927-930.
- Woo, R. et al. 1982 Effect of exercise on spontaneous calorie intake in obesity. *Amer. Journal of Clin. Nut.* 36: 447-47
- <http://www.ted.com/talks/view/lang/en//id/184>