SLEEP YA BIG DUMMY

READER'S SUMMARY:

1. Ever wonder why we use blankets to sleep?
2. A quick overview of how the hormones of sleep and metabolism are yoked.
3. See how several levees of the quilt are tied together.
4. Why the brain really needs to be understood for optimal health.

Today is a quick hitter post for those who are trying to regain leptin sensitivity. I have gotten many PM's on my Dr. Jack Kruse Facebook page and a ton of requests via Twitter (@DrJackKruse) and by email to post a synthesis post on how several levees tie together for metabolism and sleep. I would strongly recommend that you re-read the first leptin and sleep blog to review some of the material as a nice primer.

Ok, so off we go.

1. Body temperature is controlled by the hypothalamus in the brain and it also controls leptin status and sensitivity centrally via the hypocretin neurons (HCN). The HCN also help modulate our sleep by keeping the stages in order and working optimally. Cold temperatures are linked to magnetic effects of oxygen in our blood at night. It raises the question of how do sunlight and higher temperatures wake us up when the sun is out?

2. So you ever wonder why we "like" to wear blankets when we sleep? Well, the reason is simple. Melatonin is made from our brain's pineal gland from stored serotonin after four continuous hours of total darkness (the reason you need to avoid light after sunset). One of melatonin's functions during sleep is to lower our body temperature. This lower temperature increases the energy in electrons in our body to replenish us. Why do you guess the brain would want to do this when we

sleep you ask? The key protein, however, is something called melanopsin. Melanopsin is the dimmer switch for the eye as light drops to darkness and works with the sun's photoelectric effect. When I get to the quantum sleep blog you will see just how sleep is induced and how wakefulness evolved.

3. After the 4 hours of darkness, melatonin secretion increases and this allows plasma leptin to enter the hypothalamus if we are sensitive to its receptor. If we are resistant, this process can no longer occur. Once leptin enters and binds to its receptors, it affects the lateral hypothalamic tracts to immediately send a second messenger signal to the thyroid to signal it to up-regulate thyroid function and efficiency. This specifically is how we can raise our basal metabolic rate when we are leptin sensitive. The leptin receptor is an electron accountant. These coupled events, matched with leptin's actions peripherally in muscles, occur at the UCP3 sites to burn fat as we sleep at a higher basal metabolic rate. Energy added to the system changes how the biochemistry actually works by compliant design mechanisms. This is a macroscopic quantum effect in sleep. This means electron chain transport does not make ATP as usual, but electrons are still reducing oxygen. ATP is not made because the ATPase loses its ability to rotate within the inner mitochondrial membrane.

When uncoupling occurs, we make heat and not energy from normal metabolism. This heat a part of light in the infrared part of the spectrum. This means we will burn off our excess calories as pure heat. In other words, we lose light and it affects water adjacent to the mitochondrial surface. This is one reason why calories in and calories out argument makes no biologic sense once you understand how leptin works. Humans are built to burn fat at night as we sleep to lose excess weight we don't need. The timing of the leptin action is also critical. It usually occurs between 12-2 AM and is tied to when you last ate and how much darkness your eyes have seen.

This generally occurs soon after our hypothalamus releases another hormone called prolactin from our pituitary gland in the brain. The prolactin surge does not happen if the patient has sleep apnea or ate some carbs to close to bedtime. If you eat any carbs and protein within 4 hours of sleep you will never see the prolactin surge because any spike in insulin turns off this critical release. Ok, you must be asking why is this prolactin hormone so important? Is not prolactin just a hormone to secrete human milk, doc? That is not the only action of prolactin. Immediately after prolactin is released at this time, another signal is sent to the anterior pituitary to release Growth Hormone (GH). GH is stimulated only during autophagic sleep cycles in stage 3 and 4 to increase protein synthesis for muscle growth all while you're dissipating heat. This is the major release of GH in humans post-puberty. The implications here are huge. If you are LR and have sleep apnea you will have an altered body composition because of a low GH level. This occurs because you are losing electrons from your tissues during sleep and they swell. This impedes your ability to breathe well to get oxygen to your mitochondria. It means as you age you have higher body fat and lower muscle mass. This is precisely what we see in humans as they age and invariably their sleep is also poor.

The reduced temperature induced by melatonin in sleep is needed for Central Nervous System autophagic repair for another less well-known reason. The lowered temperature sets the stage for the biologic quantum effects to be optimal on neuron microtubules that facilitate learning and neuronal spouting that occur brain-wide. This is why if you don't sleep well you feel bad and your performance suffers in the next few days on tasks. Research also shows your learning is severely impaired. This is tied to a breakdown of cell membrane electron/proton transfer to microtubules. The mechanism involves alteration of small proteins connected to the cell membrane of neurons. This is why we monitor truck drivers and airline pilots sleep and wake cycles by law! Moreover, for

hospitalized patients or the elderly when this occurs, it sets the stage for the appearance of acute onset delirium. We see this often in hospitalized patients who can not sleep well in ICU's. Acute delirium states very much look the same as chronic sleep deprivation patients we see clinically as well.

4. Simultaneously, while sleep is rebuilding our cellular terroir (think levee one), the immune system is also undergoing autophagic repair……..that is another reason why the temperature has to fall. Usually, when the temperature rises, it causes the immune function to stimulate its response by inducing fever, caused by stress and infections. This "turning on", deplete our immune system of its reserves by depleting us of electrons. Dropping our temperature as we sleep allows us to repair it. During sleep, this is when the body retools our immunity to function optimally the next day by engaging a process called autophagy to recycle misfolded proteins. What controls this entire orchestra of hormonal regulation? Its all leptin-mediated, and the brain is the master receptive organ to its function because of how it accounts for the ratio of protons to electrons.

5. In children, OSA is associated with ADH release from the posterior pituitary.

ADH deficit is a lot like peripheral neuropathy development in nerves, but it ultimately begins in the brain at the leptin receptor which is an electron counter for neurons. It turns out peripheral neuropathy is also tied to the development of OSA. ADH is primary central neurons cells, but all neurons have the same general features which include cell membranes that need DHA, iodine, water, and microtubules to function.

Myelination is a ketosis seafood story: The other key linking OSA and neuropathies are tied to the altered functioning of the microtubules in neurons. Robert O. Becker associated the mammalian life system with DC currents in neurons in his work to show that all mammals use carbon-based

semiconduction to generate energy for uses in bone, regeneration, and in wakefulness. Regeneration occurs during sleep when autophagy is the key way we recycle proteins. In OSA and ADH case, it is also needed to make this posterior pituitary hormone and shuttle it down from the stalk to the pituitary via microtubules. Children with apnea often suffer from enuresis. The more blue light children face the more likely they will be bed wetters. This occurs right below the myelin layer in neurons. So any kind of central or peripheral neuropathy destroys this semiconducting ability and directly affects the ability for microtubules to work. Today, I believe those with these neuropathies and OSA have lost their effective semiconductor property in peripheral nerves but it begins in the brain be due to the fact that the transfer of charge carriers from DHA to water occurs in microtubules. A lack of *ADH, OSA or even adrenal fatigue* is a story of bad semiconduction and electron steal syndrome. Remember, water is naturally confined by microtubules in the brain and peripheral nerves. This is critical in sleep apnea cases. The brain acts by first accelerating electrons in the pi electron electric fields in DHA to shuttle them down the cell membranes of peripheral nerves. This electronic current can alter protein size and shapes of the upper airways in people with apnea. This tells you all forms of OSA/neuropathy begins in the brain secondary to a lack of DHA in the cell membranes to generate the DC current. Paleo meats are not going to help this at all because it has very little DHA in it and ***even less iodine*** that allows for the generation of the DC current to alter protein shape. Moreover, eating meat to excess will dehydrate you too. This is why the "paleo solution" falls way to short in sleep apnea reversals in my opinion.

This becomes analogous to the transfer of electrons between conduction bands in a semiconductor junction called the Josephson junction. The semiconductor property of DHA, iodine, and water would be a direct signature of the realization of how metabolic energy quanta is transformed to a kinetic energy as water moves down a microtubule. When energies are lost

proteins are misfolded, and autophagy is destroyed in poor sleep to remove these proteins. The result is altered tissues in the upper airways in those with apnea by causing tissues to alter their masses due to drops in energy. Certain cell membrane proteins are critical in the cell membranes in neurons in the brain and peripheral nerves to allow the transfer of electron's energies in this process.

The ability to change the power or energies of small proteins is the critical force that has allowed us to build our complex human nervous system. This protein can open the door to heaven or hell based upon the redox potential. The redox potential in sleep apnea is directly tied to the balance of electrons over protons in cell membranes and in our mitochondria. The energies in these particles can change the shape of these proteins. OSA is directly tied to a slow failure in this mechanism. This very same protein can lead to OSA/neuropathy when the redox potential in low in the cell membrane or our mitochondria. If you have OSA, your redox potential is low by definition. Today, it is the loss of power that is robbing of us of nerve function today. A power loss that causes the proteins in our upper airways to lose its piezoelectric ability due to electron/photon loss in DHA in cell membranes of nerves. When this happens to collagen in out tissues they get larger and obstruct our airway. In physics, energy is power and power comes in many different forms. This mechanism provides proteins with small quanta of energy that can lead to massive alterations in function. This is why poor sleep is always tied to poor autophagy, and we lose the ability to clear these larger proteins blocking airflow in us. Replacing DHA, iodine, and water is critical to lowering the upper airway tissues mass to improve oxygen delivery to restore autophagy. This is how OSA is best dealt with in my opinion.

This is why sleep gets its power to change and adapt proteins for good or bad or to open the gates to heaven or hell if you like. Proteins are the basic unit of what the matter is, and

what we are fundamental. This is why sleep is primordial to health reversals within each one of us. In order to see the true effects of sleep, we need to stop looking at morphologic changes in the upper airway and focus on why the tissues get larger because they are losing electrons and photons in our proteins to alter the shape of our proteins. We need to begin to study apnea differently and start looking at protein shapes changes in 3-D molecular simulations of how atoms interact as energies are changed by electrons or photons.

6. OSA is all tied to small protein interactions with quanta of energies changing the shape of proteins. All life is based upon protein shapes being able to morph under native forces to change tissue optics and electronics. OSA is a failure of this mechanism. My blog will be painting that picture on a canvas few see. The ability to alter protein geometry in real time allows life to innovate rapidly with utmost energy efficiency…….OSA patients have lost that innovative ability. AGE, lipid peroxidation, altered chromophore function in the brain are all the mechanisms that allow for optimal tissue morphing to save energy. Few realize that protein shape is 100% tied to mass equivalence. This means at its core, OSA is a thermodynamic problem and not a biologic one. You won’t and cannot reverse it until you see what I see.

That is why brain surgery is cool. It’s my experimental lab and helps explain why disordered sleep and metabolism are coupled at multiple levels in the human brain.

Question