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Review Article

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From Coral to Coal

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Abstract

From coral to coal. A course from life to decay; from fluid plasticity to sedimentary rigidity; from tropical reef polyps expansion to combustible oxidative free radicals reaction; from expression to suppression; from flourish to perish. All these contrasting dynamics, this vivid antithesis of an integer ecosystem to a compressed peat bog, this endless colorful miracle of delight to a blackish stratum shell of scorn, get to be defined by a single letter; an "r".

The reflection that stands in between and makes all the difference; the reflection that stands in between and as a pivotal criterion reverberates opposed conditions and qualities; the reflection that stands in between and as a sword decides and divides. Which way it will be: light or darkness, real or unreal, allostasis or allostatic load, neuroinflammation or neuroprotection, progressive neurodegeneration or cell control and communication, gamma synchrony or hyperexcitability, well-being or mind-body infirmity, is determined by a serious consideration and careful thought. The conceptual operation of inverting a system or event with respect to a plane. A level of existence and cogitation, or development in accordance with a vectoring, a direction, orientation and attention. And for all the above, the mind-eye mirror connection which perceives and interprets, and creates a neuronal abstract substrate of bound distinct circuits that form as a whole memory, undoubtedly holds the key.

Keywords: Reflection

Traumatic brain injury (TBI) is usually caused by a blow or other traumatic injury to the head or body. The degree of damage can depend on several factors, including the nature of the injury and the force of impact. Collisions involving cars, motorcycles or bicycles — and pedestrians involved in such accidents — are a common cause of traumatic brain injury.

Several complications can occur immediately or soon after a traumatic brain injury. Severe injuries increase the risk of a greater number and more-severe complications. Serious traumatic brain injury can result in bruising, torn tissues, bleeding and other physical damage to the brain. These injuries can result in long-term complications or death. Traumatic brain injury can have wide-ranging physical and psychological effects. Some signs or symptoms may appear immediately after the traumatic event, while others may appear days or weeks later.

Moderate to severe traumatic brain injury can result in prolonged or permanent changes in a person's state of consciousness, awareness or responsiveness. Different states of consciousness include: A person in a coma is unconscious, unaware of anything and unable to respond to any stimulus. This results from widespread damage

to all parts of the brain. After a few days to a few weeks, a person may emerge from a coma or enter a vegetative state. Some people with traumatic brain injury will develop seizures. The seizures may occur only in the early stages, or years after the injury. Recurrent seizures are called post-traumatic epilepsy.

Widespread damage to the brain can result in a vegetative state. Although the person is unaware of surroundings, he or she may open his or her eyes, make sounds, respond to reflexes, or move. It's possible that a vegetative state can become permanent, but often individuals progress to a minimally conscious state.

Several small or large blood vessels in the brain may be damaged in a traumatic brain injury. This damage could lead to a stroke, blood clots or other problems. Neuroinflammation occurs within seconds to minutes after a TBI, and involves a complex cascade of microglia activation, pro- and anti-inflammatory cytokine release, and up- and downregulation of neurotrophic factors. Neuroinflammation may cause acute secondary injury after TBI, and it has been linked to chronic neurodegenerative diseases. The inflammatory reaction to TBI was thought to occur solely through peripheral immune mediators entering via a disturbed blood brain barrier (BBB); it is now recognized as a robust and complex interaction between central and peripheral cellular and soluble components influenced by patient age, sex, mechanism of injury (focal, diffuse, blast), degree of injury (mild, repetitive mild, severe), secondary insults (hypoxemia, hypotension), therapeutic interventions, and genetic variability. Post-traumatic inflammation may be beneficial, by promoting clearance of debris and regeneration, and/or harmful, mediating neuronal death and progressive neurodegeneration.

Frequent headaches are very common after a traumatic brain injury. They may begin within a week after the injury and could persist as long as several months. Many people experience vertigo, a condition characterized by dizziness, after a traumatic brain injury.

The events leading to traumatic brain injury (TBI) are often psychologically traumatic (e.g. motor vehicle accidents) or occur within a broader context of psychological trauma, such as military combat or recurrent interpersonal violence. In such cases, posttraumatic stress disorder (PTSD) may develop and serve to complicate TBI recovery. Likewise, brain trauma may impede emotional resolution following psychological trauma exposure.

Psychological stress and traumatic brain injury (TBI) can both result in lasting neurobehavioral abnormalities. Some of the behavioral changes that can be associated with TBI may include, but are not limited to, increased impulsivity, increased agitation, dysregulated emotional responses, and social inhibition.

Stress is becoming a public health crisis, according to the American Psychological Association. Most Americans are reporting more stress, depression and anxiety. Physical symptoms include difficulty sleeping, headaches, stomachaches and weight gain. Stress is being associated with more health problems, such as hypertension, obesity, diabetes, depression, alcoholism and gastrointestinal problems.

"Without the vital force the material organism is unable to feel, or act, or maintain itself […]. Without the vital force the body dies."
- Samuel Hahnemann

Life emerges when biological structures are animated by energy. Energy is defined as a fundamental entity of nature that is transferred between parts of a system in the production of physical change within the system, and usually regarded as the capacity for doing work (Merriam-Webster, 2017). Without energy, there is no life – molecules alone do not interact in meaningful ways, and complex structures do not assemble nor replicate. A crucial factor that distinguishes the breathing-living organism from the inanimate body (i.e., cadaver) is the flow of energy. There are no intrinsic differences in the molecular composition of dead and living organisms. But it is a required quality of living organisms to experience a constant flow of energy through and between their different parts. This flow of energy sustains the movement, chemical reactions, and dynamic changes in the position and organization of molecules that is required to think, feel, move, and execute every element of the stress response.

Without energy, stress adaptation is not possible, and the body dies. In human/mammalian cells, a substantial fraction of energy flow occurs through a specific endosymbiotic organelle, the mitochondrion. Mitochondria are the only organelles that contain their own genome – the mitochondrial DNA (mtDNA). The mtDNA encodes proteins essential to electron flow through a series of protein complexes called the respiratory chain (also known as electron transport chain, or ETC) (Wallace, 2015). As its name implies, the respiratory chain consumes oxygen. Mitochondrial respiration channels high-energy molecular intermediates through a series of enzymatic reactions, transferring chemical energy from food substrates and oxygen into a trans-membrane electrochemical potential ($\Delta\Psi m$) (Nicholls and Fergusson, 2013). This stored energy is then used to power various mitochondrial functions, including adenosine triphosphate (ATP) synthesis, calcium uptake, protein and molecular import, biosynthesis of macromolecules and hormones, among others. Thus, the breath provides oxygen to the mitochondria, sustaining membrane potential to ensure energy flow through the cell and the whole organism. Mitochondrial energy production then powers growth, healing, as well as the complex processes required for adaptation to the changing environment.

In cases where there are defects in the energy flow, life is disrupted and shortened by disease. Stress – defined as a brain and body response aimed at promoting adaptation on the face of real or imagined threats to the organism's homeostasis – cannot occur without energy. As discussed below, every aspect of the stress response requires energy: energy-dependent enzymatic reactions, transcription and translation enabling gene expression and protein synthesis, neurotransmitter release and reuptake, hormone biosynthesis, sympathetic activation, behavioral adaptations, and long-term structural remodeling of organs and tissues are all "powered", and to some extent regulated, by cellular energy levels. Even though, in fact, basic life-sustaining biological functions also require energy for their maintenance, the energy requirement for stress responses is above the basal needs of the organisms; hence, the emphasis here on the link between stress and energy.

"Stress" is a term often used loosely given the difficulties experienced by scientists from the stress field to arrive at a consensus on its definition. Some authors have proposed to restrict the use of the term to conditions in which environmental demands exceed the natural regulatory capacity of an organism, particularly when the stressor is unpredictable and uncontrollable (Koolhaas et al., 2011). Other authors – particularly those concerned with the impact of stress on

human mental and physical health – make a distinction between positive and negative ('toxic') stress conditions (Shonkoff and Garner, 2012, Johnson et al., 2013).

Energy is present in two main forms in living organisms: (i) as heat, which permeates all structures of living organisms; and (ii) as chemical energy, in the form of chemical intermediates such as ATP that fuels specific enzymatic or biophysical reactions. Consider for example the increase in heart rate associated with the stress response. The heart is a collection of different cell types where each contraction involves the hydrolysis of billions of ATP molecules, which are required to unidirectionally transmit the wave of depolarization/repolarization, for the actin-myosin cross-bridging that provide the power stroke of contraction during systole, and for Ca2+ pumping enabling relaxation during diastole (Suga et al., 1993). Psychological stress alone increases heart rate and blood pressure by >10–20% (Schubert et al., 2009), which would correspond to an equivalent increase in cardiac energy demand. In turn, this increase in cellular energy demand also results in systemic changes such as increased breathing rate and minute ventilation, which also further increase total energy consumption. These systems-level physiological changes ultimately reflect – or are subservient, to some extent – the increase in ATP-consuming reactions within cells, and the corresponding increase in oxygen consumption within the mitochondria of the contracting heart. The same is also true of other metabolically active organs such as the brain. Stress in particular increases cerebral energy demand including oxidation of glucose and oxygen consumption (Bryan, 1990), reflecting increased mitochondrial activity within the brain. Interestingly, the stress-induced increase in cerebral energy demand may require adrenergic signaling by catecholamines (Bryan, 1990, Carlsson et al., 1977), indicating the interaction of stress mediators and mitochondrial metabolism.

Overall, biological processes starting from the basic sustenance of vital functions, acutely responding to daily stressors, all the way to the permanent adaptation to chronic stress, require substantial amount of energy. This is in part achieved by the production of broad acting hormones like glucocorticoids and catecholamines. Glucocorticoids synthesis takes place in the zona fasciculata of the adrenal cortex, and occurs via a series of molecular reactions catalyzed within the mitochondrion. Another important class of hormones released in response to certain stressors are catecholamines, particularly norepinephrine (NE) and epinephrine (E). Both are derived from the neurotransmitter dopamine, itself generated from the amino acid tyrosine.

Mitochondria fuel the stress response in two main ways: they use substrates (glucose, lipids, amino acids) and oxygen to provide energy intracellularly, via the transformation of energetic substrates and oxygen into ATP; in a related way, they also contribute to the synthesis of stress hormones, which mobilize these same energetic substrates into the circulation.

In the absence of real stressors and without the need to engage in physically demanding behavioral responses such as running away or fighting, stress hormones can dysregulate metabolism. In humans, individuals with higher circulating levels of cortisol under resting (non-stressed) conditions also have higher levels of glucose and triglycerides, and a higher score reflecting insulin resistance and a pre-diabetic state (Phillips et al., 1998). Likewise, in mice, chronic glucocorticoid administration results in elevated triglycerides, glucose intolerance, and weight gain (Karatsoreos et al., 2010). These metabolic changes are associated with higher levels of insulin and leptin hormones, indicating cross-talk between these systems. And in addition to their effects on metabolism, chronic glucocorticoid administration can also induce physical inactivity and depressive-like behavior (Karatsoreos et al., 2010, Gourley and Taylor, 2009).

Glucocorticoid secretion has important functions other than responding to stressors, namely, coordinating waking and sleeping functions during the diurnal cycle. Consistent with the role of glucocorticoids on the brain, plasticity of the brain extends to the day-night (diurnal) cycle of waking and sleeping. Some, but not all, synapses in many parts of the cerebral cortex turn over during the diurnal cycle due to the ultradian fluctuation of glucocorticoids, and interfering with that cycle by elevated glucocorticoids at the wrong time of day interferes with motor learning.

Stress increases energy demand at the cellular level and activates systemic mitocrine and endocrine processes to sustain this increased demand. Based on the increased energetic needs associated with stress responses and allostasis, and the energy-mobilizing effect of mitochondria-derived glucocorticoids, it is logical to expect that stress would be coupled to changes in food- and energy-seeking behaviors. Indeed, there is evidence, described below, that psychological stress triggers eating behavior, shifts macronutrient preferences to denser calories, and shift fat storage to the intraabdominal (i.e., visceral) fat stores, a form of energy storage with more rapidly mobilized free fatty acids than subcutaneous fat depots.

Mitochondria play a key role in the allostasis of neurotransmission and depression. Dysfunctional mitochondria may promote oxidative stress and the inflammatory tone that contribute to the depressive state (Raison et al., 2009).

A link between mitochondrial function and social behaviors has started to emerge in recent years. The ability to generate whole-body physiological response to environmental and physiological challenges is critical to allostasis and was likely a diving force behind natural selection (Weiner, 1992). The integrated energy-demanding stress responses, which involve transcriptional regulation, the secretion of various stress hormones such as glucocorticoids and catecholamines, acute metabolic changes, inflammatory mediators, neural plasticity, and many others, determine an organisms' ability to thrive and adapt, or become ill in the face of stressful situations (McEwen, 2012).

Psychological or physical stress triggers neuroendocrine, inflammatory, metabolic, and transcriptional perturbations that ultimately predispose to disease via allostatic load/overload (Juster et al., 2010). The systemic neuroendocrine consequences of mitochondrial defects have implications for our understanding of the pathogenic mechanism underlying mitochondrial disease onset and progression. Stress influences the biology of multiple diseases including cancer growth and metastasis (Thaker et al., 2006, Cole et al., 2015); diabetes (Faulenbach et al., 2012); neurodegenerative disorders (Schon and Przedborski, 2011, Picard and McManus, 2016), as well as cellular aging (Epel et al., 2004) and age-related physical and cognitive decline (Juster et al., 2010). Thus, combining the notions that mitochondria regulate the activation of stress

responses and release of allostatic mediators, and that stress response mediators influence disease trajectory, an emerging possibility is that mitochondrial dysfunction – or mitochondrial allostatic load (MAL) – may contribute to translating stressful experiences into pathophysiological processes (Picard et al., 2014).

Animals need oxygen for the conversion of food into useful energy. The fundamental importance of oxygen has been understood for centuries, but how cells adapt to changes in levels of oxygen has long been unknown. Oxygen, with the formula O2, makes up about one fifth of Earth's atmosphere. Oxygen is essential for animal life: it is used by the mitochondria present in virtually all animal cells in order to convert food into useful energy.

Oxygen is obviously a critical substance for life and its delivery to all tissue as well as physiological processes dependent on oxygen need to be carefully regulated by the body. During evolution, mechanisms developed to ensure a sufficient supply of oxygen to tissues and cells. The carotid body, adjacent to large blood vessels on both sides of the neck, contains specialized cells (chemoreceptors) that sense the blood's oxygen levels, and in response send signals to the heart to increase blood flow and pressure. The carotid sinus is a baroreceptor that senses changes in systemic blood pressure and is located in the adventitia of the carotid bulb of the internal carotid artery. Due to its location the carotid sinus is an intimately related but distinct organ from the carotid body. Innervation: same as carotid body (Hering's nerve, aka carotid sinus nerve, a branch of CN IX). In addition, carotid massage triggers the carotid sinus pathway (increased pressure on carotid sinus due to massage → sends signal to decrease systemic BP; see below NAP's indirect vagal maneuvers).

Chemoreceptors, baroceptors, thermoreceptors, mechanoreceptors, osmoreceptors, nuclear receptors, are essential allostatic sensing components which monitor and respond in order to maintain internal viability amid changing conditions (Sterling & Eyer 1988; McEwen 1998a; McEwen 1998b; Schulkin 2003). The brain generally has priority when it comes to blood, oxygen, and nutrient delivery, and so if the brain is getting enough oxygen, the rest of the body probably is also. This is an immediate-response system.

There are also longer-term responses that react to chronic hypoxia (when tissues do not have enough oxygen to meet their needs). One response is to grow new blood vessels to deliver more blood to the tissue. The other is to release a hormone called erythropoietin (EPO), which is made in the kidneys. EPO increases the production of red blood cells, which contain the oxygen-carrying hemoglobin that delivers oxygen to tissues.
Biological systems are complex webs that exist in dynamic equilibrium states. It's important to remember this whenever we think about potential new interventions. Simplistic notions that if something is low we will just increase it and have a predictable benefit without downsides are often naïve (although sometimes things do work this way – we just have to be careful). We have to think of our interventions in the context of interacting with an existing equilibrium.

Oxygen sensing is also important not just for normal physiological functioning, but for development as well. This highlights the fact that develop itself is a feedback system. Genes are not blueprints telling cells exactly how to build a person, but rather a set of processes that will unfold to develop a person. Blood vessels, for example grow based only partly on a basic anatomical plan, but mainly in response to hypoxia. Wherever there is low oxygen, grow more blood vessels to deliver more oxygen until an optimal equilibrium is reached.

The concept of "allostatic load" focuses on the paradox that the same mediators that help the body and brain adapt can also cause pathophysiology when overused and dysregulated. This terminology is more inclusive of life events than "stress". "Homeostasis" represents the physiological state which the body maintains to keep us alive – that is, body temperature, pH, and blood oxygen levels are kept within a narrow physiological range. In order to maintain homeostasis, our body triggers hormone secretion and activates the autonomic and central nervous systems (we call these "mediators" like cortisol, adrenalin, the immune system, and metabolism) to help us adapt.

In research, "stress" is often used to denote molecular damage or dysfunction that occurs from a challenge, overuse, or even from toxins. But in a precise sense, any ever so slight perturbation to the system (such as standing up from the sitting position) induces a myriad of biological changes that aim to preserve homeostasis. So using the word "stress" does not really describe all of the underlying biology.

The term allostasis (homeostasis) refers to the active process by which the "mediators" of the neuroendocrine, autonomic, metabolic, and immune systems help us adapt, as long as they are turned on in a balanced way when we need them and then turned off again when the challenge is over (Sterling et al., 1988). In other words, allostasis is what allows certain physiological parameters (e.g., blood glucose) to remain constant through changes in other parameters (e.g., insulin) – stability through change. When allostatic mediators are not turned off, these same mediators can cause unhealthy changes in brain and body. This is also the case when the mediators are not produced in an orchestrated and balanced manner – for example, too much or too little cortisol or an elevated or too low blood pressure. When dysregulation of these systems continues over weeks and months, we call it allostatic load, which refers to the wear and tear on the body that results from the chronic overuse and imbalance of the "mediators" (McEwen, 1998, McEwen and Stellar, 1993). Allostatic load also includes the consequences of the health-damaging behaviors that often accompany a stressful lifestyle or are present in society, like and unhealthy diet, alcohol, smoking, inadequate sleep, lack of exercise, social isolation. Accumulation of belly fat is an example of allostatic load, as is the development of chronic hypertension.

K (+)-selective ion channels are critical determinants of membrane excitability in neuronal cells. Like many other cells in our body, neuronal cells have a propensity to maintain their homeostasis. Action potential firing is the most important function to maintain in brain neurons, as they are the elements of neural networks. If one element fires action potentials at an abnormally high rate, the entire network could become epileptic. Therefore, brain neurons adjust their intrinsic membrane excitability to maintain the firing rate within their own optimal operational range. When a neuron receives an enormous input, it will reduce the membrane excitability to prevent overshooting. When it is deprived of stimulus, the membrane

becomes more excitable to avoid total quiescence. The homeostatic regulation of intrinsic excitability provides stability to the neural network in the face of dynamic and plastic synaptic inputs. Neurons achieve this type of homeostatic regulation through a variety of ion channels, including K (+) channels. Under certain pathological conditions, these homeostatic mechanisms provide neuroprotection.

Moreover, stress is associated with changes in the functioning of immune cells.That is, there is a relatively large decrease in both lymphocyte proliferation and natural killer cell activity in individuals who have experienced stress. There seems to be some connection between the duration of the stress and the amount of immune change. For example, the longer the stress, the greater the decrease in the number of specific types of white blood cells.

Connections between negative psychological states (such as anxiety and depression) and immune system variables have also been explored. The results suggest that depressed and anxious mood states are associated with decreases in lymphocyte proliferation and natural killer cell activity, as well as changes in the numbers of white blood cells and the quantity of antibody circulating in the blood. It also appears that the body's ability to produce antibody to a specific substance is related to the level of anxiety that the individual is experiencing: with more anxiety the less antibody is produced after exposure to the potentially harmful susbtance.

How could stress or negative emotional states alter the immune system? Both physiological and behavioural mechanisms provide possible explanations. In the case of physiological mechanisms, stress is associated with the activation of several systems, including the hypothalamic-pituitary-adrenal axis and the sympathetic nervous system. The activation of these two pathways results in elevated blood levels of specific hormones, namely cortisol and the catecholamines (epinephrine and norepinephrine). Blood levels of these hormones are related to immune functioning. For example, acute increases in cortisol and epinephrine are related to decreases in the number of white blood cells in circulation. Lymphocyte proliferation and natural killer cell activity are also decreased when there are acute increases in cortisol and epinephrine.

Other hormones released under stress - such as growth hormone, prolactin, and the natural opiates (beta endorphin and enkephalin)- have been implicated in influencing the immune system. At a cellular level, these hormones become attached to receptors on white blood cells and so affect them. An alternative explanation involves the association of stress with specific behaviours that modulate the immune response. Stressed persons tend to sleep less, exercise less, have poorer diets, smoke more, and use alcohol and other drugs more often than non-stressed people. These behaviours have all been shown to affect the immune system.
The interpretation of these changes in the immune system due to stress is difficult. Even though decreased natural killer cell activity is evident in certain human diseases (such as cancer, chronic viral infection or autoimmune diseases), the direct health consequences of such a decrease have not been established. Nevertheless, it is clear that stress has an adverse effect on health, probably mediated- at least in part -by the body's immune system. It is hoped that future research will show how, by reducing stress, we can improve health.

Anxiety is an aversive emotional state, in which the feeling of fear is disproportionate to the nature of the threat. In response to threatening situations, the feeling of the emotion that constitutes the subjective feature of anxiety is accompanied by emotional stress, which involves behavioral, expressive and physiological features, such as an avoidance of the source of the danger, assuming defensive postures and an increase in blood pressure, respectively. Anxiety is a normal emotional response to a threat or potential threat. When this emotion is inappropriate, extreme and persistent, it is classified as pathological. Anxiety is implicated in a number of psychiatric disorders, such as depression, panic attacks, phobias, generalized anxiety disorder, obsessive-compulsive disorder and post-traumatic stress disorder.

There's a fine line between stress and anxiety. Both are emotional responses, but stress is any demand placed on the brain or physical body, typically caused by an external trigger. The trigger can be short-term, such as a work deadline or a fight with a loved one or long-term, such as poverty, discrimination and chronic illness. People under stress experience mental and physical symptoms, such as irritability, anger, fatigue, muscle pain, digestive troubles and difficulty sleeping. Anxiety, on the other hand, is defined by persistent fear, excessive worries that don't go away even in the absence of a stressor. Anxiety leads to a nearly identical set of symptoms as stress: insomnia, difficulty concentrating, fatigue, muscle tension and irritability. It can be a reaction to stress, or it can occur in people who are unable to identify significant stressors in their life.

Kuloglu et al. recently established a link between oxidative stress and certain anxiety disorders (obsessive-compulsive disorder and panic disorder), demonstrating that other systems, such as oxidative metabolism, can affect the regulation of anxiety. These findings, which establish a link between oxidative stress and pathological anxiety, inspired a number of other recent studies focusing on the link between oxidative status and normal anxiety, and also on a possible causal relationship between cellular oxidative stress and emotional stress.

It is well known that low/moderate concentrations of reactive oxygen species (ROS) affect a great number of physiological functions. However, when ROS concentration exceeds the anti-oxidative capacity of an organism, animal cells enter a state termed oxidative stress, in which the excess ROS induces oxidative damage on cellular components. As a result, oxidative stress has been implicated in a large range of diseases, including cancer, diabetes, male infertility, autoimmune diseases, atherosclerosis and cardiovascular disorders. The brain is highly vulnerable to oxidative stress due to its high O2 consumption, its modest antioxidant defenses and its lipid-rich constitution. As a result, oxidative stress can alter neurotransmission, neuronal function and overall brain activity.

Oxidative stress describes a state when the body responds to various harmful stimuli and produces excessive amounts of reactive oxygen free radicals, known as reactive oxygen species (ROS), and reactive nitrogen radicals, known as reactive nitrogen species (RNS). Oxidative stress reflects an imbalance between the systemic manifestation of reactive oxygen species and a biological system's ability to readily detoxify the reactive intermediates or to repair the resulting damage. This leads to accumulation of ROS and RNS in the body or in cells, causing cell toxicity and eventually leading to

tissue damage. The damage to intracellular proteins, lipids, and DNA caused by oxidative stress products such as reactive free radicals and peroxides is an important factor in aging and is also involved in the pathogenesis of cancer.

Although excessive oxidative stress is considered harmful, reactive oxygen is beneficial for maintaining the normal physiological activity of the body and plays a role in defense, killing pathogens by regulating the immune system. The outcome of oxidative stress depends on the degree of damage in the balance between the oxidative and antioxidative responses of the body. Cells are capable of self-regulation of mild oxidative stress changes and restoring cell homeostasis. However, severe oxidative stress can lead to cell death, and it has been reported that although moderate oxidative stress can cause cell apoptosis, intense oxidative stress may lead to necrosis.

Traumatic brain injury (TBI) is a major healthcare concern, constituting a major cause of death and disability throughout the world. Among the factors leading to TBI outcome are biochemical cascades which occur in response to primary and secondary injury. These mechanisms generate oxidative stress, an imbalance between oxidant and antioxidant agents that can result in neural dysfunction and death. After TBI, an assembly of oxidative stress markers (carbonylated proteins, lipid peroxides, reactive oxygen and reactive nitrogen species) are produced in the brain, while antioxidant defense enzymes decrease (GSH, ratio GSH/GSSG, GPx, GR, GST, G-6PD, SOD, CAT). This imbalance is directly related to the pathogenesis of TBI. Therefore, the development of antioxidant strategies is of primary interest in ongoing efforts to optimize brain injury treatment.

Intracerebral hemorrhage (ICH) as a result of severe head trauma, is a serious medical condition. The pathological mechanisms of hematoma after ICH within brain parenchyma triggers a series of adverse events causing secondary brain injury (SBI) and severe neurological deficits. Oxidative stress plays an important role in SBI after ICH, which leads to irreversible disruption of the components of the neurovascular unit, constituting gray and white matter, and is followed by blood brain barrier disruption and deadly brain edema with massive brain cell death.

A variety of pathways can induce the generation of free radicals after ICH, of which there are two major pathways. First, blood cell decomposition products such as iron ions, heme, and thrombin can induce the production of free radicals. Experimental results show that divalent iron ions can interact with lipid and generate free radicals, leading to nerve damage. Second, inflammatory cells, such as microglia and neutrophils, can generate free radicals.
During the inflammatory response following ICH, neutrophils are stimulated and activated, resulting in outbreak of the respiratory chain, releasing large amounts of reactive oxygen species, nitric oxide, and so on, and the excessive consumption of superoxide dismutase (SOD) and the occurrence of lipid peroxidation.

Damage to nerve cells caused by free radicals manifests in a number of ways, with free radicals involved in pathological processes ranging from cell membrane damage to DNA interruption or even apoptosis. Cell damage caused by oxygen free radicals is due to the induction of lipid peroxidation. The lipid-rich brain tissue is particularly sensitive to oxygen free radicals that can enhance lipid peroxidation, cause membrane damage, and increase cell membrane permeability and calcium ion influx.

In the meantime, cross-linking and polymerization of membrane lipids will occur due to lipid peroxidation, which will indirectly inhibit the activities of membrane proteins such as calcium pumps, sodium pumps, and Na+/Ca2+ exchangers. This leads to a further increase in intracellular calcium concentration which subsequently stimulates mitochondrial calcium pumps to take in calcium. Calcium and phosphate in the mitochondria combine and form insoluble calcium phosphate, which causes interference in mitochondrial oxidative phosphorylation and leads to a decrease in ATP production.

Meanwhile, increased intracellular calcium ion concentration can activate phospholipase, promoting membrane phospholipid decomposition and causing damage to the structure of cell and organelle membranes. In summary, free radicals are the major killers of hemorrhagic brain tissue, with considerable recent research indicating that free radicals are closely related to brain injury and disorders caused by bleeding.

Due to a multiplicity of causes provoking traumatic brain injury (TBI), TBI is a highly heterogeneous pathology, characterized by high mortality and disability rates. TBI is an acute neurodegenerative event, potentially and unpredictably evolving into sub-chronic and chronic neurodegenerative events, with transient or permanent neurologic, cognitive, and motor deficits, for which no valid standardized therapies are available.

A vast body of literature demonstrates that TBI-induced oxidative/nitrosative stress is involved in the development of both acute and chronic neurodegenerative disorders. Cellular defenses against this phenomenon are largely dependent on low molecular weight antioxidants, most of which are consumed with diet or as nutraceutical supplements. The analysis of the very few clinical studies does not allow strong conclusions to be drawn on the real effectiveness of antioxidant administration to TBI patients.

The reduction of oxidative stress could be achieved in three levels: by lowering exposure to environmental pollutants with oxidizing properties, by increasing levels of endogenous and exogenous antioxidants, or by lowering the generation of oxidative stress by stabilizing mitochondrial energy production and efficiency. Endogenous oxidative stress could be influenced in two ways: by prevention of ROS formation or by quenching of ROS with antioxidants. However, the results of epidemiological studies where people were treated with synthetic antioxidants are inconclusive and contradictory. Recent evidence suggests that antioxidant supplements (although highly recommended by the pharmaceutical industry and taken by many individuals) do not offer sufficient protection against oxidative stress, oxidative damage or increase the lifespan. The key to the future success of decreasing oxidative-stress-induced damage should thus be the suppression of oxidative damage without disrupting the wellintegrated antioxidant defense network. Approach to neutralize free radicals with antioxidants should be changed into prevention of free radical formation (read below: biophoton emissions decrease from the body due to reduced free radicals in meditating subjects).

Traumatic brain injuries (TBI) carry numerous symptoms, side effects and comorbid conditions. A recent study showed that one

of the more common issues is dry eye disease (TBI related vision problems may cause eyes also to tear up more than usual), which is caused by a lack of lubricating tears in the eye. Ocular and other eye pain was also shown to be higher in those with a prior brain injury. Past research has generally supported the connection between traumatic brain injury and dry eye, with multiple studies linking the two conditions as part of larger problems related to vision.

This new data is also important because painful light sensitivity and photophobia—a common symptom of TBI—is also a prominent side effect of dry eye disease. Combined with the greater likelihood of headaches and PTSD as validated by this latest study, and this reinforces the increased risk faced by patients for long-term neurological problems. Therefore, preventative care and pain management are critical steps to helping patients with traumatic brain injuries who also manifest issues of dry eye.

While high ROS levels within the mitochondria lead to oxidative stress and potential organelle damage, ROS outside the mitochondria may be involved in inflammation, a primary mechanism of dry-eye disease. This inflammation can be the result of ROS exiting the mitochondria, or the generation of ROS in other cellular structures. Macrophages and other phagocytes involved in fighting infection use ROS as a weapon against foreign invaders, but control of these ROS is not always adequate. In particular, pro-inflammatory cytokines, such as IL-1β, can stimulate ROS to levels that can lead to oxidative tissue injury.

Tears are a clear salty liquid secreted by the lacrimal glands (tear gland) found in the eyes of all land mammals (except for goats and rabbits). Their functions include lubricating the eyes (basal tears), removing irritants (reflex tears), transfering oxygen and nutrients to the cornea, and aiding the immune system. When blinked, the eyelids help spread tears evenly over the surface of the eye. When closed, the eyelids help trap the moisture against the surface of the eye. Small glands at the edge of the upper and lower eyelids secrete an oily substance that contributes to the tear film and keeps tears from evaporating. Tears keep the surface of the eye moist. Without such moisture, the normally transparent cornea can become dried, injured, infected, and opaque. Tears also trap and sweep away small particles that enter the eye. Moreover, tears are rich in antibodies that help prevent infection. The eyelids and tears protect the eye while allowing clear access to light rays entering the eye.

Weakening of the immune system as a result of organic stress also contributes to eye problems (eg. dry eye, conjunctivitis, uveitis, retinitis etc). The eye, similar to other tissues, is subject to both immune protection and attack. However, in many ways the eye is a unique tissue immunologically, in part due to a complex structure. The external surface of the eye is a mucosal tissue that is exposed to the environment, and is subject to constant challenges both in terms of microorganisms as well as a hostile external environment. Yet, normally the delicate ocular surface remains clear and healthy. Protection of the ocular surface and antibacterial activity is the role of substances present in the tear film, mucins, and antibacterial substances produced by immune cells and ocular surface structural cells.

Perturbation of immune homeostasis by environmental stress and/or infectious agents can lead to immune-mediated damage to the ocular surface that threatens vision. The eye and the brain are prototypical tissues manifesting immune privilege (IP) in which immune responses to foreign antigens, particularly alloantigens are suppressed, and even completely inhibited. As a result, IP functions normally as a homeostatic mechanism preserving normal function in tissues, particularly those with highly specialized function and limited capacity for renewal such as the eye and brain. IP mechanisms such as blood–ocular barriers, intraocular immune modulators, induction of T regulatory cells, lack of lymphatics, and other properties maintain tissue integrity.

When someone suffers a head trauma, sometimes there is damage to the optic nerve that is responsible for passing information between the eyes and the brain. When the optic nerve is injured, there are tears and swelling in the affected area that causes the nerve cells to die. This type of injury is called traumatic optic neuropathy, or TON, and results in irreversible vision loss. At this point, there is no effective treatment for TON and the mechanisms of the optic nerve cell death have been largely unclear. Research from The University of Texas Medical Branch in Galveston has shed new light on what causes the permanent vision loss sometimes seen in the wake of a head injury. The findings are detained in The American Journal of Pathology.

Wenbo Zhang, UTMB associate professor in the department of ophthalmology & visual sciences, and his team found that inflammation brought on by white blood cells play a role in head trauma-induced vision loss. Limiting inflammation could decrease nerve damage and preserve cell function, researchers discovered. Inflammation is part of the body's defense system against injury and infection and is an important component of wound healing. White blood cells travel to injured areas to help repair the damaged tissue, causing inflammation in the process. Excessive or uncontrolled inflammation can actually make injuries worse and contribute to disease in a couple of different ways -- by activating cell death processes, clogging and rupturing blood vessels and producing toxic molecules like free radicals (see below: after practice of meditation, biophoton emissions from the body decrease; this could be due to reduced free radicals in meditating subjects).

"Our data clearly showed that one of the protein receptors on white blood cells called CXCR3 brings white blood cells to the optic nerve in response to production of its binding partner CXCL10 by damaged nerve tissue," said Zhang. "When we deleted CXCR3 or gave mice a drug that blocks the receptors following optic nerve damage, we observed fewer white blood cells on the scene by real-time noninvasive imaging, nerve damage was decreased and nerve cell function was preserved compared with mice that did not receive any intervention following injury".

Traumatic Optic Neuropathy (TON) is a condition in which acute injury to the optic nerve from direct or indirect trauma results in vision loss, which can be severe and irreversible. The severity of optic nerve damage may range from simple contusion to complete avulsion of the optic nerve.

Avulsion of the optic nerve is a rare complication after ocular trauma but carries a poor prognosis. Traumatic optic nerve damage can

occur via direct and indirect mechanisms in different parts of the optic nerve. Due to the absence of connective tissue between the optic nerve fibers in the lamina cribrosa region and the absence of myelin sheathing around the nerve fibers, the optic nerve head is a relatively weak structure. Since Hippocrates first noted visual impairment after facial trauma, numerous reports have described TON, focusing on its causes and methods of treatment.

The most common cause of TON is indirect injury to the optic nerve, which is thought to be the result of transmitted shock from an orbital impact to the intracanalicular portion of optic nerve. Direct TON can result from penetrating injury or from bony fragments in the optic canal or orbit piercing the optic nerve. Orbital hemorrhage (orbital compartment syndrome) and optic nerve sheath hematoma can also cause TON by direct compression. There are no known risk factors for TON. The most common mechanisms of injury were motor vehicle accident, bike accident, fall and assault.

The exact pathology of indirect TON is not well understood. The optic nerve dura is continuous with the orbital periosteum, leaving the optic nerve susceptible to transmission of force from blunt head trauma, particularly that affecting the superior orbital rim. Indirect TON has been hypothesized to result from shearing injury to the intracanalicular portion of optic nerve, which can cause axonal injury or disturb the blood supply of the optic nerve. It has also been suggested that the optic nerve may swell in the optic canal after trauma resulting in increased luminal pressure and secondary ischemic injury. Direct TON is presumed to be the result of tissue disruption secondary to foreign body or bony fragments impacting on the optic nerve.

The diagnosis of TON is made clinically based on history and ophthalmic signs. Like other optic neuropathies, patients with TON may have decreased central visual acuity, decreased color vision, an afferent pupillary defect and/or visual field deficits. It is important to remember that albeit rare, TON can be bilateral, so an afferent pupillary defect may not be seen in patients with bilateral injury and vision loss. The optic nerve head will appear normal initially, but optic atrophy can be seen 3-6 weeks after the initial traumatic event.

A history consistent with TON would be vision loss after blunt or penetrating trauma that could not be explained by slit lamp or dilated fundus findings. Often these patients complain of acute unilateral decrease in vision, color vision deficits, or visual field deficits. The history and subjective complaints may be delayed due to the impact of and treatment for other concomitant head injuries or other systemic comorbidities.

Beginning around 1970, researchers began seriously to study the visual brain. One of the chief discoveries is that it is composed of many different visual areas that surround the primary visual cortex (V1). V1 acts as a post office, distributing different signals to different destinations; it is just the first, vital stage in an elaborate mechanism designed to extract essential information from the visual world.

What we now call the visual brain is therefore V1 in combination with the specialized visual areas with which it connects either directly or indirectly. Parallel systems are devoted to processing different attributes of the visual world simultaneously, each system consisting of the specialized cells in V1 plus the specialized areas to which these cells project. In other words, vision is modular.

Researchers have long debated why a strategy has evolved to process the different attributes of the visual world in parallel. The most plausible explanation is that we need to discount certain kinds of information in order to acquire knowledge about different attributes. With color, it is the precise wavelength composition of the light reflected from a surface that has to be discounted; with size, the precise viewing distance must be ignored; and with form, the viewing angle must become irrelevant.

Recent evidence has shown that the processing systems are also perceptual systems: activity in each can result in a percept independent of the other systems. Each processing-perceptual system has a slightly different processing duration, and reaches its perceptual end-point at a slightly different time from the others. There is a perceptual asynchrony in vision, as color is seen before form, which is seen before motion. Color is processed ahead of motion by a time difference in the order of 60-100 ms. This means that visual perception is also modular. The visual brain is characterized by a set of parallel processing perceptual systems, and a temporal hierarchy in visual perception.

The human eye and brain together translate light into color. Light receptors within the eye transmit messages to the brain, which produces the familiar sensations of color. In a biological sense, the human eye can distinguish exactly three colors - red, green, and blue. Those who say the eye can see more than three colors are actually giving the answer to the question: “How many colors can the human brain interpret?”. Newton observed that color is not inherent in objects. Rather, the surface of an object reflects some colors and absorbs all the others. We perceive only the reflected colors. Thus, red is not “in” an apple. The surface of the apple is reflecting the wavelengths we see as red and absorbing all the rest.

An object appears white when it reflects all wavelengths and black when it absorbs them all. Red, green and blue are the additive primary colors of the color spectrum. Combining balanced amounts of red, green and blue lights also produces pure white. By varying the amount of red, green and blue light, all of the colors in the visible spectrum can be produced. Considered to be part of the brain itself, the retina is covered by millions of light-sensitive cells (photoreceptors), some shaped like rods and some like cones. These receptors process the light into nerve impulses and pass them along to the cortex of the brain via the optic nerve. So light enters the eye through the pupil and is focused onto the retina. All the wavelengths hit all the cells, but the red cones respond to red light, the blue to blue light, and the green to green light. These signals get sent to the brain and the brain interprets what color you must be looking at depending which cones were responding and how much the cones were responding.

So let’s say you look at yellow light. The yellow light stimulates the red and green cones. The brain senses this stimulation and thinks: “My red and green cones are responding. That happens when I look at yellow light. Therefore, I must be looking at yellow light”. So the brain gives you the illusion of seeing yellow even though you do not actually see the yellow light. You are actually “seeing” a combination of red and green and interpreting yellow.

The varying degrees with which your cones respond are responsible for different shades and hues. This is where the millions of colors come from, as opposed to the three different colors you can actually see. You see three colors, but thanks to the magic that is the human brain, you can experience millions of colors. The color produced by light is a kind of an energy. This energy affects both the functions of our body as well as our mind and emotions. Although the eye is the organ of sight, studies conducted suggest that in reality the brain perceives the image.

There is a connection between our brain and our actions. Depending on how the brain is stimulated, a person can be rendered happy, angry, sad or anxious. The central nervous system is the main control center of human actions. According to studies, each stimulus received by the nerve cells first affects the brain stem then the entire nervous system. Human beings are subjected to many stimuli, including sight during the day. These stimuli can be small or very large in number (Mahnke, 1993). Colors affect the bodily functions, mind and emotions with the energy produced by light. Studies conducted have demonstrated the benefits of colors where the development of brain, creativity, productivity and learning are concerned.

Being subject to excessive stimuli can cause changes in breathing pattern, pulse, blood pressure and muscle tension. On the other hand, too little stimuli can lead to anxiousness, sleeplessness, excessive emotional reaction, loss of concentration and nervousness. For example, a completely white environment leads to lack of stimulus and this, contrary to expectations, does not cause a balanced or neutral effect.

Many factors can contribute to sensory overload after brain injury. First, the brain is devoting most of its energy towards repairing itself. Scientists have found that the injured brain is more active than a healthy brain, with many brain regions working overtime to compensate for lost function. Therefore, the brain has no residual strength to process or organize information from the senses. This means that even a little stimulation can cause an overload. Similarly, attention and concentration skills are limited after brain injury. So if more than one thing is happening at once, the brain will get easily overwhelmed. Finally, the stress, pain, and fatigue naturally experience after TBI can intensify the senses and put the patient on edge. Things that never noticed before can now bother and lead to sensory overload.

Some examples of sensations that might trigger sensory overload include:

- Background noises that cannot be ignored
- Itchy clothing
- Bright lights
- Large crowds

Overstimulation will cause adverse cognitive effects, which is why it is so important to learn how to cope with sensory overload. Sensory overload activates the body's defense mechanism, otherwise known as the "fight or flight" response. As a result, each person will experience overstimulation a little differently, depending on whether their instinct is to fight back or run away. Some people will become aggressive and display other behavioral problems such as screaming and violent behavior. Others will simply shut down emotionally and will not respond. Some might start crying or even vomiting.

Sensory deprivation refers to the lack of sensory stimulation, either by natural causes in cases of blindness or deafness, or in experimental settings. Experimental studies of sensory deprivation in animal models consider the role that sensory experience plays in development, reorganization, and plasticity of brain and behavior by withholding or restricting that experience. Sensory deprivation studies in all three major modalities (visual, auditory, and somatosensory) demonstrate the importance of sensory experience for normal brain development.

The activity-dependent reorganization of the brain (and in particular the cerebral cortex) follows rules postulated by Hebb for associative learning. In their most general form, these rules state that synaptic connections are strengthened in which the pre- and postsynaptic neurons are active together. Short-term sessions of sensory deprivation are described as relaxing and conducive to meditation; however, extended or forced sensory deprivation can result in extreme anxiety & stress, hallucinations, bizarre thoughts, and depression.

Meditation is a practice where an individual uses a technique, or focusing the mind on a particular object, thought, or activity – to train attention and awareness, and achieve a mentally clear and emotionally calm and stable state. Meditation is a mind and body practice that has a long history of use for increasing calmness and physical relaxation, improving psychological balance, coping with illness, and enhancing overall health and well-being. Mind and body practices focus on the interactions among the brain, mind, body, and behavior.

There are many types of meditation, but most have four elements in common: a quiet location with as few distractions as possible; a specific, comfortable posture (sitting, lying down, walking, or in other positions); a focus of attention (a specially chosen word or set of words, an object, or the sensations of the breath); and an open attitude (letting distractions come and go naturally without judging them).

Attention is the behavioral and cognitive process of selectively concentrating on a discrete aspect of information, whether considered subjective or objective, while ignoring other perceivable information. It is a state of arousal. As William James (1890) wrote, "[Attention] is the taking possession by the mind, in clear and vivid form, of one out of what seem several simultaneously possible objects or trains of thought. Focalization, concentration, of consciousness are of its essence".

Attention remains a crucial area of investigation within education, psychology, neuroscience, cognitive neuroscience, and neuropsychology. Areas of active investigation involve determining the source of the sensory cues and signals that generate attention, the effects of these sensory cues and signals on the tuning properties of sensory neurons, and the relationship between attention and other behavioral and cognitive processes. Prior to the founding of psychology as a scientific discipline, attention was studied in the field of philosophy. Thus, many of the discoveries in the field of attention were made by philosophers.

In the twentieth century, the pioneering research of Lev Vygotsky and Alexander Luria led to the three-part model of neuropsychology defining the working brain as being represented by three co-active

processes listed as Attention, Memory, and Activation. Attention is identified as one of the three major co-active processes of the working brain. Attention may be differentiated into "overt" versus "covert" orienting. Overt orienting is the act of selectively attending to an item or location over others by moving the eyes to point in that direction. Overt orienting can be directly observed in the form of eye movements. Covert orienting is the act of mentally shifting one's focus without moving one's eyes. Simply, it is changes in attention that are not attributable to overt eye movements. Orienting attention is vital and can be controlled through external (exogenous) or internal (endogenous) processes.

Exogenous (from Greek exo, meaning "outside", and genein, meaning "to produce") orienting is frequently described as being under control of a stimulus. Endogenous (from Greek endo, meaning "within" or "internally") orienting is the intentional allocation of attentional resources to a predetermined location or space. Simply stated, endogenous orienting occurs when attention is oriented according to an observer's goals or desires. Attention is best described as the sustained focus of cognitive resources on information while filtering or ignoring extraneous information.

Attention is a very basic function that often is a precursor to all other neurological/cognitive functions. Focused attention is the ability to respond discretely to specific visual, auditory or tactile stimuli. In many cases attention produces changes in the EEG. Many animals, including humans, produce gamma waves (40–60 Hz) when focusing attention on a particular object or activity.

A gamma wave is a pattern of neural oscillation in humans with a frequency between 25 and 140 Hz, the 40-Hz point being of particular interest. Gamma rhythms are correlated with large scale brain network activity and cognitive phenomena such as working memory, attention, and perceptual grouping, and can be increased in amplitude via meditation or neurostimulation. Altered gamma activity has been observed in many mood and cognitive disorders such as Alzheimer's disease, epilepsy, and schizophrenia.

The suggested mechanism is that gamma waves relate to neural consciousness via the mechanism for conscious attention: The proposed answer lies in a wave that, originating in the thalamus, sweeps the brain from front to back, 40 times per second, drawing different neuronal circuits into synch with the precept [sic], and thereby bringing the precept [sic] into the attentional foreground. If the thalamus is damaged even a little bit, this wave stops, conscious awarenesses do not form, and the patient slips into profound coma. Thus the claim is that when all these neuronal clusters oscillate together during these transient periods of synchronized firing, they help bring up memories and associations from the visual percept to other notions. This brings a distributed matrix of cognitive processes together to generate a coherent, concerted cognitive act, such as perception.

This has led to theories that gamma waves are associated with solving the binding problem. Gamma waves are observed as neural synchrony from visual cues in both conscious and subliminal stimuli. This research also sheds light on how neural synchrony may explain stochastic resonance in the nervous system. High-amplitude gamma wave synchrony can be self-induced via meditation. Long-term practitioners of meditation such as Tibetan Buddhist monks exhibit both increased gamma-band activity at baseline as well as significant increases in gamma synchrony during meditation, as determined by scalp EEG. fMRI on the same monks revealed greater activation of right insular cortex and caudate nucleus during meditation.

The neurobiological mechanisms of gamma synchrony induction are thus highly plastic (neuroplasticity). This evidence may support the hypothesis that one's sense of consciousness, stress management ability, and focus, often said to be enhanced after meditation, are all underpinned by gamma activity. At the 2005 annual meeting of the Society for Neuroscience, the current Dalai Lama commented that if neuroscience could propose a way to induce the psychological and biological benefits of meditation without intensive practice, he "would be an enthusiastic volunteer."

In Science, something is called "a problem" when there is no plausible model for its substrate. So we have the mind–body problem (Chalmers 1996), but not the color problem, although there is a great deal of ongoing color research. There is continuing progress in understanding the neural substrate for coordination in the brain, but there is still an air of mystery about "The Binding Problem".

Any coherent distributed system needs a way of assimilating information, so at a basic level some kind of binding is unavoidable. We start by considering the abstract computational problem and coordinated action in social systems as well as the traditional neural binding problem (NBP). Any large parallel system will have a lot of information that cannot be fully accessible at every node. The brain, with its billions of neurons, is one example, but the problem is inherent. Any distributed system should ideally make decisions/actions based on all available information, but this is combinatorially impossible—the system architecture needs to privilege certain combinations. The brain has the additional constraint that almost all connections are local. Most of the work on the NBP has been focused on the visual system. The purpose of combining information is to make good decisions and actions.

"The binding problem is, basically, the problem of how the unity of conscious perception is brought about by the distributed activities of the central nervous system" (Revonsuo and Newman (1999)). In its most general form it arises whenever information from distinct populations of neurons must be combined. Somehow the activity of specialised sets of neurons dealing with different aspects of perception are combined to form a unified perceptual experience. The binding problem also occurs in each modality of perception and different versions of the problem have been described in language production, visual perception, auditory perception, and other mental processes.

In the case of visual perception, the brains of humans and other animals process different aspects of perception by separating information about those aspects and processing them in distinct regions of the brain. For example, different areas in the visual cortex specialise in processing the different aspects of colour, motion, and shape. This type of modular coding yields ambiguity in many instances. For example, when humans view a scene containing a red circle and a green square, some neurons signal the presence of red, others signal the presence of green, still others the circle shape and square shape. Here, the binding problem is the issue of how the brain represents the pairing of color and shape. Specifically, are the circles red or green? The binding problem is also an issue in

memory. How do we remember the associations among different elements of an event? How does the brain create and maintain those associations? Both the hippocampus and prefrontal cortex seem to be important for memory binding.

It is important to recognize that the brain is a neural system that evolved to run a physical body in a social environment. It is constantly trying to find a best fit between the agent's goals and noisy perceptual input and is subject to all manner of illusions (Feldman 2006).

A phosphene is the phenomenon of seeing light without light actually entering the eye. The word phosphene comes from the Greek words phos (light) and phainein (to show). Phosphenes can be directly induced by mechanical, electrical, or magnetic stimulation of the retina or visual cortex, or by random firing of cells in the visual system. Phosphenes have also been reported by meditators. Phosphenes have been known since antiquity, and described by the Greeks. Most people see splashes of colors and flashes of light on a not-quite-jet-black background when their eyes are closed.

Human visual system — eyes and brains — don't shut off when denied light. Let's start with the almost-black background. The color black is often referred to as the absence of light, but when it comes to the human visual system, eigengrau is the color perceived in the absence of light. Eigengrau is a German term that roughly translates to 'intrinsic gray' or 'own gray.' When deprived of light — as in when eyes are closed, or when in darkness with eyes open — we are unable to perceive true blackness, and rather, perceive eigengrau. This is because light provides the contrast necessary to perceive darker-ness. For instance, the black ink of text might appear darker than eigengrau because the whiteness of the page provides the contrast the eyes need to understand black. But eigengrau is not a static color. It can change shades of gray, and it can be interrupted by phosphenes.

You can think of the visual system, when the eyes are closed, like a recording camera with the lens cap on. The camera is still fully functional. It's still recording and storing away minutes and hours of data — it's just not very interesting data. In the same way, retinas remain fully functional even with eyes closed. The retina is the layer of light-sensitive cells at the back of the eyeball; it records stimuli and transmits impulses through the optic nerve to the brain, which compiles them into a visual image. This colorful light show happening inside the eyelids, these strange blob, is not ordinary light — this light comes from inside the eyes. In the same way that fireflies and deep-sea creatures can glow, cells within our eyes emit biophotons, or biologically produced light particles.

"We see biophotonic light inside our eyes in the same way we see photons from external light," said István Bókkon, a Hungarian neuroscientist who works at the Vision Research Institute in Lowell, Massachusetts. Biophotons exist in the eyes because atoms constantly emit and absorb tiny particles of light, or photons. This photon exchange is just a part of normal cellular function. Eyes can't tell the difference between photons from outside light and the biophotons emitted by its own atoms. Either way, the optic nerve simply relays these light signals to the brain, which must then decide if it accurately represents the real world around, or if it's just a phosphene.

Eyes actually produce far more biophotons than we end up seeing as phosphenes. Inside the retina, millions of tiny cells called rods and cones collect light and convert it into electrical signals. These signals travel through the optic nerve to a part of the brain called the visual cortex. Here, the brain reconstructs an image using the information received from the eyes. When a reconstructed image looks abstract, the brain is quick to label the image as unreal, optical illusion imaginery or a phosphene. But that information doesn't always come from the retinas. According to Bókkon, phosphenes can originate in various other parts of the visual system, too.

Research has shown that direct electric and magnetic stimulation of the brain can trigger phosphenes, and Bókkon hopes to soon be able to prove that biophotons are responsible for these phosphenes as well. Depending on where a phosphene originates, it can take on a variety of shapes, patterns and colors. Different atoms and molecules emit photons of different wavelengths, which is why we see different colors. A phosphene with an orderly geometric pattern like a checkerboard may have originated in a section of the retina where millions of light-collecting cells are arranged in a similarly organized pattern. Researchers have also found that different areas of the brain's visual cortex create certain specific shapes of phosphenes.

In the 1950s, the German researcher Max Knoll at the Technische Universität in Munich came up with a classification scheme for phosphene shapes. He studied phosphenes in over a thousand volunteers and came up with 15 categories, including triangles, stars, spirals, spots and amorphous blobs. He discovered that by prodding different areas of the visual cortex with an electrode device, he was consistently able to induce the same kinds of phosphenes. The most common phosphenes are pressure phosphenes that occur when physical pressure is placed on the retina. Pressure phosphenes can last for a few seconds, like after rubbing your eyes, sneezing, coughing, or straining, or they may last longer, as with a retinal vitreous detachment. Pressure phosphenes can also occur due to issues in the eye, like an infection, tumor, inflammation, a blood vessel abnormality, traumatic brain injury, thyroid disease etc.

A disease like multiple sclerosis (MS) can affect the pressure in the eye and it can also cause phosphenes due to inadequate function of the optic nerve. Medication can also alter the function of the retina in ways that produce phosphenes and can make you see lights that aren't there; persons who endure long periods without visual stimulation (the prisoner's cinema), or those who ingest psychedelic drugs.

Drug and alcohol abuse can produce a variety of ocular and neuro-ophthalmic side effects. Novel, so-called "designer," drugs of abuse can lead to unusual ocular disorders. Legal substances, when used in manners for which they have not been prescribed, can also have devastating ophthalmic consequences. Effects of these substances on the visual system can range from mild keratopathy, to severe vision loss from endophthalmitis or occipital lesions. Abnormalities of the tear film can induce severe visual dysfunction due to the creation of an irregular refracting surface.

Smoking has been associated with an increased incidence of dry eye. The Beaver Dam Eye Study found that dry eye symptoms were related to a history of heavy alcohol consumption. The proposed mechanisms for the altered tear function were that ethanol increased tear osmolarity, acted as a solvent, and could disturb cytokine

production. In this manner, ethanol could exacerbate the signs and symptoms of dry eye syndrome.

A comparative case-control study between men who drank heavily (≥4 drinks per day) and non-drinkers showed that heavy ethanol ingestion was associated with a decreased tear break up time, lower Schirmer I test, as well as altered conjunctival impression cytology in comparison to non-drinkers. Several factors as drugs, alcohol, stress, psychotic conditions can induce phosphenes.

Rapid heart rate, fast breathing, and a sudden, overwhelming feeling of panic — anxiety can cause these physical and mental changes. Some people report other changes when their anxiety is high, namely, floaters or flashes of light that have them seeing stars. The concept that anxiety or other strong emotions could cause changes in what a person sees isn't a new concept.

The only people who never see phosphenes are people who have been blind since birth. But people who lose their vision due to illnesses or injuries usually don't lose all visual functions. Because phosphenes can originate in different parts of the visual system, "theoretically, all blind people who could previously see can retain the ability to see phosphenes," explained Bókkon. Researchers have also been studying ways to trigger phosphenes in blind patients to try and figure out a way to potentially restore their vision. If scientists can use technology to make the blind see phosphenes, perhaps they can use similar technology make them see real images.

Why do electrons emit photons?

Electrons carry electric charge. Electric charge is the source of the electromagnetic field. So electrons interact with the electromagnetic field. In a quantum field theory, this interaction between the electronic field and the electromagnetic field comes in set chunks, set units at any given frequency/energy. Therfore, whenever an electron interacts with the electromagnetic field, this interaction is in the form of emitting or absorbing such a unit, or quantum, of electromagnetic field energy. That quantum is known as the photon. So it really is the electric charge. If there was no electric charge, the electromagnetic field would exist just by itself (as an idea), without interacting with anything else. No photons would be emitted or absorbed.

Does light ever become mass? Yes, light can become mass (phosphenes are light particles inside the eye, which can be considered as minimal in action photons in respect to the maximum action of photons entering the eye from the external enviroment. However, truth is that every reverse processing is neccessarily a quasi-static one, and the real path is the one being minimal in action. If we're lucky to find it, Hamilton's principle of least action just says we should be happy in this regard).

Here is the simplest example. Take an object. Measure its mass. Now shine a powerfull light onto that object, allowing it to absorb some of that light. As a consequence, the object warms up a little (its constituent particles wiggle a little faster, having a little more kinetic energy). Now measure the object's mass again. You will find that it's ever so slightly more, as the mass associated with that thermal energy is now added to its mass (eg. biophotons -> phosphenes -> retina-optic nerve-thalamus-visual cortex V1 activation).

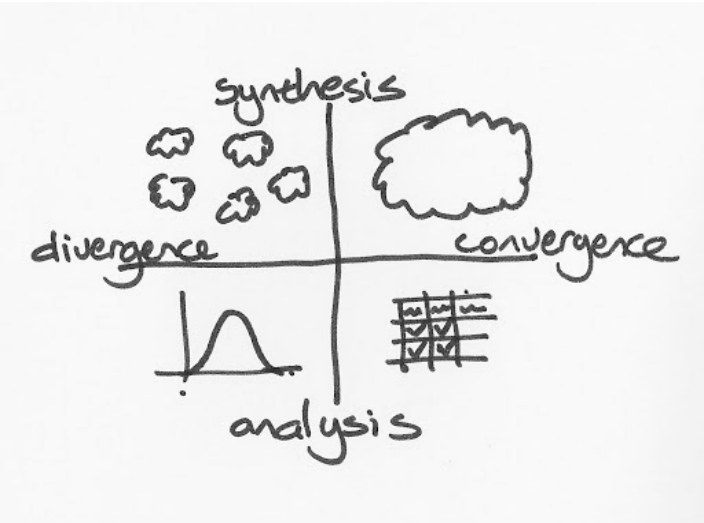
This measurment may not be realizable in practice (the mass difference is really, really tiny) but other, more complex measurments can be, so we know that light does become mass. And as an extreme astrophysical example, there are supergiant stars, in which a considerable fraction of their total mass is in the form of a "photon gas", which is trapped radiation (that is, light or electromagnetic radiation at other wavelengths).

In the tangible domain, two subtle energy carriers come to mind: biophotons and bioelectrons. Biophotons are photons (light particles) that are generated within the body, and these could be measured as they emanate from the skin. Similarly, bioelectrons are available from within the body; these are measured in instruments such as electro-photonic imaging. Biophotons, however, are light particles that are generated within the body and are constantly radiated from the body surface. These spontaneous emissions are thought to be associated with generation of free radicals due to energy metabolic processes. Since these dynamic metabolic processes are common to most living systems, it is likely all living beings give rise to biophotons. Further, these light emissions are extremely weak and hence cannot be observed by the naked eye. Detections of biophotons need special photon counters which are sensitive to pick up even a single photon in the environment.

It is known further, that after practice of meditation, biophoton emissions from the body decrease (an energy leak is when we're expending energy in ways that cause an energetic deficit); this could be due to reduced free radicals in meditating subjects. Communication and control are two required activities within and between cells to maintain homeostasis. Normally, it is thought that both these functions are achieved through biochemical and neurological means. The coherent light source is now thought to be another arm through which both control and communication are achieved. This may be true especially in long-range communications in the body.

It is known that oxidative stress is harbinger of many metabolic syndrome disorders. This also seems to contribute to aging and related degeneration in the body. Hence, measuring metabolic syndrome through a consistent method is of importance. It is likely that biophoton emission is a fundamental process and its measurement could portend a stable method; however, measurement method itself is expensive and complex.

It is possible that the photon measurements could be substituted or complemented with electron availability in the biosystems. This could become an easy and noninvasive method of measuring oxidative process in the body. While the problems of aging and oxidative stress are inherent in any living system, it is also possible to reverse these processes through practice of meditation. Work is also emerging in this area; if lifestyle confers these problems obviously changing lifestyle and reversing and mitigating these degenerative processes is also possible.



Cogito, ergo sum

Abstract is something that exists in thought or as an idea but not having a physical or concrete existence. Stress is abstract. It is a complex ocillational circuit that creates an uncertain (foggy) wave patterning of complex reverbatory circuits of neuronal influence. It is a mind state first that inevitably will be translated into a body state afterwards. So in order to deal with stress effectively, you have to come with a comatable strong idea too.

Recent studies have shown clearly that abstract reasoning activates logic.

Logic exists in thought or as an idea but not having a physical or concrete existence either. It is also an ocillational circuit that creates instead a certain (geometry) wave patterning of complex reverbatory circuits of neuronal influence (antithesis). It is a mind state first that inevitably thereupon will be translated into a body state.

Logic and stress are two dimetrically opposed variables. They form a strong polarity of influence according to the vectoring. If the direction is pointed towards logic, then we talk about coherence, cohesion, geometry, tensional integrity, biotensegrity, golden ration, beauty, eurythmia, order, normal functioning, health. If the orientation it is pointed towards stress, then we talk about disorder, disarythmia, deformation, deterioration, decay, disease.

Case report

This case report is about a near-death traumatic brain injury (TBI) with complete loss of vision, mental disorders, history of post-traumatic epilepsy, frequent headaches, migraines, vertigos, posture & balance problems, ocular inflammatory conditions (dry eye syndrome), painful light sensitivity, photophobia, and use of dark glasses to compensate these weaknesses (if the blindness is caused by damage to the optic nerve, eventhough the eye itself is intact, there will be no pupillary response), heavy alcoholism combined with anti-epileptic drugs use, smoking addiction, poor, unhealthy diet, overweight, extended belly, sleep/circadian rhythm disorders, nightmares, fatigue.

According to the patient's testimonials, her ordeal had started in April 1998 when she had a horrible – near-death motorcycle accident, in which she lost control of her motorcycle on a wet, slippery road surface and shattered her head on the protective street bars. From the massive blow and with the absence of a helmet, her bare head suffered severe cranium damage. As a result of the impact, her right eye popped out from the eye socket, her nose – teeth – lips, and tongue were broken and torn. She was transferred to the hospital and had undergone immediate tracheostomy. All the emergency doctors expected her to die due to the severe TBI and the intracerebral hemorrhage (ICH). With surgeries and medication, they tried to manage and reduce the internal bleeding, the edema, the formation of blood clots, the inflammation, and they tried a complete reconstruction of her head fractures with implants and cement.
In the end, she survived by a miracle. However, she suffered many losses (physical and mental). She was in the intensive care unit (ICU) for months, she suffered from seizures, and she spent many years in the mental institute under neuropsychiatric care and medication. When she finally returned home, she was a complete wreck.

As the years were passing by, so did her hope to see again. The severe compression of the optic nerve from the intracerebral hemorrhage (ICH), the edema, and the hematoma formation, was one parameter. The oxidative stress responses, and the neuroinflammation, was the second. As a result, she ended up an alcoholic in the Greek blind community.

In September 2014, a friend of hers paid a visit to her house and informed her about my Salutogenic, stress coping, health-ease oriented mind-body intervention, neuronal stimulation research; an independent system with neutral behavior.

In the absence of real stressors and without the need to engage in physically demanding behavioral responses such as running away or fighting, stress hormones can dysregulate metabolism. In humans, individuals with higher circulating levels of cortisol under resting (non-stressed) conditions also have higher levels of glucose and triglycerides, and a higher score reflecting insulin resistance and a pre-diabetic state (Phillips et al., 1998).
Chronic glucocorticoid administration can also induce physical inactivity and depressive-like behavior (Karatsoreos et al., 2010, Gourley and Taylor, 2009).

With her consent, I started working daily, performing thorough and careful brief sessions of my research mind-body intervention on the temporal and occipital parts of her head while lying relaxed, face upward (supine) with eyes closed.

My mind-body's intervention (MBI) logical, transpersonal meditation-deep relaxation-stress coping- indirect vagal maneuvers (mastoids & occipital artery; temporomandibular joint & trigeminal-vagus nerve-cilliary ganglion connection), algorithmic model of transdermal (sensory) neuronal stimulation:

Skin (senses) -> haptic (relating to the sense of touch, in particular relating to the perception and manipulation of objects using the senses of touch and proprioception), quasi-static, isotropic mechanical pressures -> kinetic-elastic rythmical oscillations (slow vibrational response of skin's elastic connective tissue/ fascia) -> mechanoreceptors -> free nerve endings -> tonic property

-> connection to proprioception & recording of the slow stimuli (intrinsic excitability and synaptic plasticity work hand in hand to form engrams; synaptic strength and neuronal connectivity are critical for memory formation; the excitability of neurons, is the ability to generate a large, rapid change of membrane voltage in response to a very small stimulus) -> reticular formation -> locus coeruleous homeostatic control center; principal site for brain synthesis of norepinephrine (focuses attention; eg. tactile stimuli); involved with physiological responses to stress and panic; part of the reticular activating system -> scanning attentiveness (tonic mode) -> hypothalamus-thalamic relay nuclei-amygdala-hippocampus neuronal network connection -> relation to meditation (mind/abstract) -> gamma waves -> stress reduction, homeostatic regulation of intrinsic excitability, relaxation, mood elevation, increased life expectancy of the mind and, by extension, the body (mind-body). After practice of meditation, biophoton emissions from the body surface (skin) decrease (an energy leak is when we're expending energy in ways that cause an energetic deficit); this could be due to reduced free radicals in meditating subjects. Thus, energy saved from biophtons can be a potential element to trigger phosphenes in blind patients.

Classical conditioning (also known as Pavlovian conditioning) is learning through association and was discovered by Pavlov, a Russian physiologist. In simple terms, two stimuli are linked together to produce a new learned response in a person or animal (eg. small + slow external stimuli -> NAP's user friendly, neutral, abstract model of transdermal (sensory) neuronal stimulation algorithmic response). John Watson proposed that the process of classical conditioning (based on Pavlov's observations) was able to explain all aspects of human psychology. Everything from speech to emotional responses was simply patterns of stimulus and response.

How do neurons decide where to fire (which connected neuron to fire at)?

A neuron has several branches at its distal end, and those knobby tips at the end terminate on different postsynaptic neurons. The neuron cannot decide which of those other neurons to send a signal to. A signal traveling down its axon will always branch out and go equally to all neuron this one is connected to. The decision as to which of those will fire is made by those receiving neurons, not by the sending one. The receiving neurons exhibit tiny (~0.5 mV) membrane voltage changes called postsynaptic potentials (PSPs) in response to the incoming signal.

Postsynaptic potentials can be either excitatory (EPSPs) or inhibitory (IPSPs). That depends on what kind of neurotransmitter is released by the presynaptic neuron pictured, and what kind of receptors the postsynaptic neurons have. From a typical resting potential of –70 mV to a typical firing threshold of –55 mV requires at least 30 EPSPs to happen quickly (each coming before the previous one decays very much) for the receiving cell to fire. However, a receiving (postsynaptic) neuron gets input from many presynaptic ones. Each neuron in the brain, typically receive input from about 40,000 presynaptic neurons. Some of those may be telling it to fire (with EPSPs) and some of them may be telling it not to fire (with IPSPs). The postsynaptic neuron more or less integrates the algebraic sum of these positive and negative changes in voltage to "decide" whether to fire or not. The EPSPs have to outnumber the IPSPs by enough to reach firing threshold, or else the postsynaptic neuron will not fire. So, it's not the sending (transmitting) cell that determines which of its target cells respond; it stimulates all of those equally. It's the receiving neurons that make that decision, and they can all decide differently even though they're all getting identical input from any one transmitting cell.

In cases of severe traumatic brain injury (TBI), the allostatic load of the nervous system is extremely high when post-traumatic epilepsy is present. The addition of other neuropsychological burdens, external environmental factors, and lifestyle errors make it even worse. That means the brain is under a tremendous firing load, a neuronal siege. To balance this high excitation, you have to come with an opposed behavior. To become neutral, abstract, to bring the counterweight factor into play; simplicity and subtraction over accumulative bio-complexity.
If 360 degrees considered as the ultimate maximum of a circle, then 0 is the highest minimum of it. But actually, 0 and 360 are the same. The numerical difference between them only denotes a beginning and an end. So imagine having this mathematical paradigm in mind, a traumatized brain with post-traumatic epilepsy history is working at 360. To be able to influence this super-high status of function and get a chance from the neurons that decide to welcome your incoming information, this could only happen if the one has the same power degree as the dominant ones.

Small + slow is the logical combination that exhibits powerful offset properties towards this highly firing status.
To battle against something big and have an equal chance to prevail, you have to come with something big too; a pattern of stimulus and response in a complete diametrically reverse condition; a David to a Goliath or a turtle to a rabbit, for example. These vivid opposing examples show clearly that properties of size (David versus Goliath) and speed (turtle versus rabbit) do matter. Meaning, the abstract quality of the antithesis is the other side of the same coin.

David and turtle win in the end because of the working axis difference. They flip the coin upside down since they are moving on the vertical and not on the horizontal axis as Goliath and the rabbit. Both David and the turtle seem to appear in a completely different dimension of action regarding their opponents. However, this neglected parameter of the dimensional spectrum from Goliath and the rabbit is the main reason for their defeat (Alexandros Senarelis-Sinaris).

The same goes for complete darkness, vacuum, or void. In the grand absence of light or matter, a single electron, and its tiny electric charge, which is the source of the electromagnetic field, create from this quantum interaction the photon -a mathematical abstraction-, that as a wave or/and as a matter can illuminate this recondite condition with an amount of energy undoubtedly small. And this because it can fit in and fill in with its duality every possible lacuna. Every single hole, pit, missing gap, or invisible detail that creates loose ends; unexplained, unsettled, unresolved, and unstable situations (Alexandros Senarelis-Sinaris).

Electrons circle the nucleus in fixed orbits. There's a huge amount of theory around electron orbitals, but to understand light there is just one key fact to understand: An electron has a natural orbit that it occupies, but if you energize an atom, you can move its electrons

to higher orbitals. A photon is produced whenever an electron in a higher-than-normal orbit falls back to its normal orbit. During the fall from high energy to normal energy, the electron emits a photon -a packet of energy- with very specific characteristics. The photon has a frequency, or color, that exactly matches the distance the electron falls.

And what happens to a mega fall, or a massive suppression? Perhaps a big packet of energy concetration. A durable core of condensed and trapped potential, buried deep though under the debris created by a dramatic twist of events (Alexandros Senarelis-Sinaris).

Blindness from seeing is as 0 to 360, or 360 to 0. As noted above, whichever way you see it is the same. The only thing that changes is the numerical polarity, the perception of the beginning and the end. But the bizarre thing is that even though they are extremities, so far apart, geometrically they appear like Siamese, one being. And the question is on which verge they dangle to change, what is the hidden variable that switches them from one side to the other?

The answer is simple and complicated at the same time (simplexity).

0 and 360, fast and slow, phasic and tonic, big and small, the story of little David and giant Goliath, the myth of Asopus with the rabbit and the turtle, darkness and light, least and maximum action, high orbit and fall, they are all in essence abstract. Brain dependent. Thus, abstract plus brain equal to abstract reasoning. Abstract reasoning has a coherent scientific link with logic. Logic, therefore, is the hidden variable. The needed loop through which things flow, circulate, shift, interchange, and rotate. If it is missing, the end is never going to get connected with the beginning, and vise versa. Meaning, nothing is going to change, to grow, to develop, to evolve, and be understood.

After all, the neurons themselves decide. The neurons come to a resolution as a result of an intrinsic consideration, a reflection of the mind. And logic as a high neurobiological mechanism seems to be the influential catalyst in this decision-making process of internal dynamics (Alexandros Senarelis-Sinaris).

Logic is the true pathfinder, the trustworthy explorer. The impetus that with vehemence will search and scan for all the possible nexuses that can have firmly cocooned and sealed the contingent of inversion (Alexandros Senarelis-Sinaris).

Logic is intuitive illuminated energy. It dwells in high brain regions (neocortex), but it also dives into emotions (limbic system) and primordial biological functions (brain stem). With every other needed descent, with every required level reduction, logic behaves like an electron. Therefore, during this evolutionary neurodevelopment fall from high to low, a photon -a packet of energy- is emitted, having specific characteristics: a frequency, or color, that exactly matches the distance the electron falls. As a result, if the space that logic covers stretch far, analogous will be the energy produced from this space-time trajectory (Alexandros Senarelis-Sinaris).

Life emerges when biological structures are animated by energy. Energy is defined as a fundamental entity of nature that is transferred between parts of a system in the production of physical change within the system and usually regarded as the capacity for doing work (Merriam-Webster, 2017). Without energy, there is no life – molecules alone do not interact in meaningful ways, and complex structures do not assemble nor replicate.

Energy is life, and life is energy. Energy is abstract. It can become everything.

So for the eyes that they cannot see, light is the form of energy that gives them life. Inevitably, I see logic as light. And I regard that it reflects, refracts, diffracts, diffuses, and lights up everything when present. From the perspective of Lagrangian mathematics, logic can act as a multiplier. When the energy reserves are low, the insertion of it in the equation will reverse the biological shortage; and this by falling from high cortical orbits to deeper and deeper structures of the matrix to produce packets of energy, photons of light, life itself in every other cycle of interaction with mechanisms, behaviors, and their biological effects (Alexandros Senarelis-Sinaris).

In May 2015, seventeen years of a complete absence of light, and all the visual anatomic structures considered collapsed from the neuronal quiescence, unexpected changes to her had emerged regarding the eyesight. Neuropathological research has demonstrated signs of neuroinflammation up to 18 years' post-injury in patients with moderate/severe TBI, and these findings were related to white-matter degeneration. The spectrum of her vision consisted of phosphenes (various black & white geometric patterns-flashes-stars-colors & colorful geometric patterns-amorphous black & white or colored shapes), and floaters that she described as small flies. However, this unexpected phenomenon was a shock to her and her social circle. Everybody started laughing out of awkwardness and mocking her bizarre, "magical" and illusory visual experiences.

Due to phosphenes' abstract neural character, I have received criticism for fooling a blind person with unreal, imaginary information (phosphenes). I have received criticism for taking advantage of her need to see again. I have received criticism for taking advantage of her physical and mental weaknesses. As a result, all my actions were suddenly considered illegal. Ignorance and superstition are risky. Bias against or in favor is perilous. But simple thinking (and not careful thinking; reflection) in a complex world is a recipe for disaster.

If God listened to the prayers of men, all men would quickly have perished: for they are forever praying for evil against one another. - Epicurus

Orbiting around this black hole of social prejudice, I continued my research.

As the visual information was increasing and getting established in every repetitive session, she started to experience more complex and advanced phenomena: like the fuzzy shape with the depth perception of different objects using visual scanning, visual attention & visual memory, partial active/fast motions of waving hands, people walking and passing fast in front of her, cars & bikes, her blurry reflection on the mirror, the distinction between day and night, and the perception of light from electric bulbs.

This new status for her was painful and strenuous in the beginning.

Along the way, she reported that she was able to handle more easily the incoming information. Her mind-eye connection was getting stronger with every other single visual process. So to check upon the

liability of the information given, I told her to inform the neurologist and do all the necessary actions for the Visual Evoked Potentials (VEP) test. Instead, she went to the central clinic of ophthalmology in Athens.
She learned that her severe TBI case with post-traumatic epilepsy history required two neurologists to perform this demanding test in the controlled area of a hospital, and not in a clinic from an ophthalmologist. So she went to her ophthalmologist, who was working in a well-known and highly equipped private hospital in Athens, seeking for his medical advice and guidance (consultation). He claimed that the hospital didn't have the specific equipment to perform this test, even though this hospital's official website advertised in detail the neurology department, with the high tech appliances for every medical procedure needed. So he told her instead, to go to a private diagnostic ophthalmological center where he was an associate, even though he knew in detail the neurological gravity of her case.

The electrophysiological test reported: inability to focus, conductivity disorder, and unsatisfactory recording performance of the left eye; signed by an ophthalmologist, from a private diagnostic ophthalmological center, instead of (a) neurologist(s) from a public hospital. Additionally, there was a significant discrepancy of information between her technical description of the test and the known standard procedure of it: placing area, number, and patterning of the electrodes on the the scalp using a custom-made hat (10-20, 10-10, 10-5). No physical discomfort reported after this strenuous test. My next move/option was to ask for an EEG (electroencephalogram).

The EEG results signed by a neurologist, documented response to light stimuli while opening the eye, plus other neurological findings regarding her brain's function, and absence of hyperexcitable indications. She also confirmed that she was able to see the light stimuli while opening her eye during the neurological procedure.

The same test, according to her testimonials, was performed by a group of Russian ophthalmologists in 2012, where they diagnosed the complete absence of eye-brain reaction to intense light stimuli. The only thing that she was feeling back then was the torture of the burning sensation on her face and inside her head, due to the absence of pupil reaction, and due to the insane amount of light entering her eye (maximum action), meaning 100% disability.

From Coral to Coal (the blind shall lead the blind)

In the end, after two endless years of enormous physical, mental, and emotional effort, I had to disengage myself from this falsity. The decision was due to biased, warped, untrue, incorrect, and offensive behavior from family members, relatives, close friends, other sufferers from the blind community, health care professionals, and many more relevant and irrelevant skeptics concerning mind-body interventions. Their attitude was also censorious, coming forcefully against me and my research's directive principle and teleological purpose, which is basically to bring the abstract parameters of logic & truth into play. How primitive, retrospective, pathetic, discouraging, and depressing.

And the logical question that comes into my mind is: who is blind after all?
The one who is not able to see, or the ones who lack thought clarity?

"Clarity of thought"- That's not saying "logical thinking," although that's important.

One's thoughts alone are often not a solution to a problem. It is also possible that your thoughts contain faulty assumptions or the ability to lead you in a circle creating faulty reasoning. It is almost impossible to determine if you are in this process alone, but you may be able to change your thinking at any time if you are willing though to do so.

As a vivid antithesis to the above information, the Integrative Medicine and Health Research Program at Mayo Clinic studies the effectiveness and feasibility of using mind-body techniques to reduce anxiety before and during medical procedures, improve quality of life for patients and caregivers, and help people manage stress. Mind-body techniques can also complement conventional therapies for conditions such as major depression, sexual distress, spinal cord injuries, or cancer.

Mind-body techniques focus on strengthening a harmonious connection between a patient's mind and body to maintain or improve health. Techniques include meditation, deep breathing, stress management and resiliency training, and music therapy.

Since 2008, authors documenting research conducted on behalf of the NCCIH have used terms "mind and body practices" and "mind-body medicine" interchangeably with mind-body interventions to denote therapies, as well as physical and mental rehabilitative practices, which "focus on the relationships between the brain, mind, body, and behavior, and their effect on health and disease. The center has also stated that "mind and body practices include a large and diverse group of procedures or techniques administered.

The United States National Center for Complementary and Integrative Health (NCCIH) defines mind-body interventions as activities that purposefully affect mental and physical fitness. The NCCIH does not consider mind-body interventions as within the purview of complementary and alternative medicine when there is sufficient scientific evidence for the benefit of such practices along with their professional application in conventional medicine.

All mind-body interventions focus on the interaction between the brain, body, and behavior and are practiced with intention to use the mind to alter physical function and promote overall health and well-being. Research shows that mind-body practices can have a positive effect on almost every system in our body (MD Anderson's patient tip).

Western mind-body interventions (MBI) was popularized in the early 20th century but dates back to Ancient Greece. The Greek values of strength and beauty in combination with Greek mythology led to activities intended to promote confidence. Proponents of mind-body intervention (MBI) techniques suggest that a rationale for mind-body training is that the mind follows the body and the body follows the mind. The body-mind connection can be attributed to hormones

and chemicals released during movement, although the mind-body connection is dominated by the brain and is considered to be more of a neurological mechanism. My MBI is a western, user-friendly mind-body, neuro-based intervention with neutral, abstract behavior and salutogenic bearings.

With the deepest respect to the human imperfect, my mind-body intervention (MBI) operates non-aggressively towards the cryptoalgorithm of any form of problem and the noisy, foggy, obfuscating dynamics of its potential complications. With the physiological tactic of its centrifugal approach, moves peripherally and in between (neutral behavior) the event horizons of the healthy bio-available and any pathology, disease, illness, syndrome, injury etc, which is basically the "abstract" area of the emerging stress before the true values-1, such as biophotons-light-energy-electricity-3D holographic & elctromagnetic fields, inevitably become false values-0 (nihilism).

With the introduction and the usage of valuable and complex information (informational medicine) of undoubtedly rigorous sciences such as mathematics, physics, biology and philosophy, Natural Anamorphosis Process (NAP) as a regulator/buffer aims for the least action path (Hamilltonian physics) and with the controlled repetition (enrgams) of its intentional systems & agents, intends to maintain and enhance the physio-logical mechanisms of homeostasis and, by extension, through the parasympathetic system which returns the body to homeostasis (vagus nerve-interoception), the stored energy levels of healthy bioavailability, since they form the absolute natural intrinsic self-repair mechanism of existence & smoothness (Navier-Stokes equation).

References

1. Traumatic brain injury - Symptoms and causes - Mayo Clinic
2. Traumatic Brain Injury and Posttraumatic Stress Disorder: Conceptual, Diagnostic, and Therapeutic Considerations in the Context of Co-Occurrence - the journal of Neuropsychiatry and Clinical Neurosciences
3. An energetic view of stress: Focus on mitochondria - Martin Picard, Bruce S Mc Ewend, Elissa S Epel, Carmen Sandi, Frontiers in Neuroendocrinology, Elsevier/ScienceDirect
4. Nobel Prize in Physiology or Medicine 2019 - Science Based Medicine
5. Carotid Body and Carotid Sinus -- General Information - Iowa Head & Neck Protocols
6. Neuroinflammation in the Evolution of Secondary Injury, Repair, and Chronic Neurodegeneration after Traumatic Brain Injury - NCBI
7. An integrated perspective linking physiological and psychological consequences of mild traumatic brain injury - Journal of Neurology
8. Stress and Traumatic Brain Injury: A Behavioral, Proteomics, and Histological Study - frontiers in Neurology
9. Brain Injury &
Neurobehavioral Disorders - May Institute
10. Use integrative approach to stress-induced dry eye - Healio
11. Stress and the immune system - World Health Organization
12. What's the difference between stress and anxiety? - American Psychological Association
13. Stress and Anxiety - Healthline
14. Oxidative stress and anxiety - NCBI
15. Intracerebral Hemorrhage, Oxidative Stress, and Antioxidant Therapy - Oxidative Medicine & Cellular Longevity, Xiaochun Duan, Zunjia Wen, Haitao Shen, Meifen Shen, and Gang Chen
16. Oxidative stress in traumatic brain injury - PubMed
17. Antioxidant Therapies in Traumatic Brain Injury - NCBI
18. Strategies for Reducing or Preventing the Generation of Oxidative Stress - Oxidative medicine and Cellular Longevity
19. Traumatic Brain Injury and Dry Eye - TheraSpecs
20. Oxidative Stress Reduction for Dry Eye - Review of Ophthalmology, Mark B. Abelson, MD, CM, FRCSC, FARVO, George Ousler and Linda Stein, Andover, Mass
21. Tears - Wikipedia
22. Protective Features of the Eyes - by James Garrity, MD, Mayo Clinic College of Medicine
23. Immunology of the Eye—Inside and Out - NCBI
24. Why there is sometimes permanent vision loss after head injury - University of Texas Medical Branch at Galveston, ScienceDaily
25. Traumatic Optic Neuropathy - EyeWiki
26. Traumatic Optic Neuropathy - NCBI
27. Optic Nerve Avulsion and Retinal Detachment After Penetrating Ocular Trauma: Case Report - NCBI
28. Seeing color | Causes of Color - Webexhibits
29. Color Think Tank - How Do We See Color? - Pantone
30. How many colors can the human eye distinguish? - Quora
31. Good news–bad news: the Yin and Yang of immune privilege in the eye, John V. Forrester and Heping Xu - frontiers in Immunology
32. The Effect of Color | Psychology and Color - Renk Etkisi
33. 33. Best Ways to Avoid Brain Injury Sensory Overload - FlintRehab
34. Sensory Deprivation - an overview | ScienceDirect Topics
35. The Power of Shutting Down Your Senses: How to Boost Your Creativity and Have a Clear Mind - Belle Beth Cooper
36. Meditation - Wikipedia
37. Meditation: In Depth | NCCIH
38. Attention & Gamma waves - Wikipedia
39. The neural binding problem(s) - NCBI
40. Binding problem | Psychology Wiki | Fandom
41. Phosphenes - Wikipedia
42. Why We See Swirling Colors When Our Eyes Are Closed - by Liesl Goecker
43. Why do we see colors with our eyes closed? - by Hanneke Weitering
44. Why You Sometimes See Stars and Flashes of Light - Troy Bedinghaus, OD
45. Ocular manifestations of drug and alcohol abuse - NCBI
46. Eye Flashes and Anxiety: Is There a Connection? - Healthline
47. Why do electrons emit photons? - Quora, Viktor T. Toth
48. Does light become mass? - Quora, Viktor T. Toth
49. Biophotons as Subtle Energy Carriers - NCBI
50. Natural Anamorphosis Process - Alexandros Senarelis Sinaris, Journal of Clinical Immunology & Research
51. Classical conditioning - Simply Psychology, by Saul McLeod
52. How do neurons decide where to fire (which connected neuron to fire at)? - Quora, Ken Saladin, professor emeritus, human anatomy & physiology, author of "Anatomy & Physiology", Ph.D. from Parasitology, Florida State University, former professor of histology (microscopic anatomy)
53. Producing a Photon | HowStuffWorks - Science, by William

Harris & Craig Freudenrich, Ph.D
54. To Acu or not to Puncture? - Alexandros Senarelis Sinaris, Journal of Clinical & Experimental Immunology
55. Neuronal Excitability - an overview | ScienceDirect Topics
56. Homeostatic regulation of neuronal excitability by K (+) channels in normal and diseased brains - PubMed, Hiroaki Misonou
57. Memory engrams: Recalling the past and imagining the future - Sheena A. Josselyn, Susumu Tonegawa
58. How do you build clarity of thought? - Quora, Lynn Wright
59. Integrative Medicine & Health Research Program - Mayo Clinic
60. Mind-body interventions - Wikipedia