

Insufficiency of Cellular Energy (ICE) in Neurons: From Electrical Hyperactivity to Quiescence

Review Article

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Abstract

An alternative cellular energy (ACE) pathway exists which is different from cellular energy obtained from the metabolism of food. It occurs from the absorption of an external force termed KELEA (kinetic energy limiting electrostatic attraction). This force increases the dynamic quality of the body's fluids, resulting in enhanced biological activities. KELEA is reversibly attracted to separated unbound, electrical charges. It was previously proposed that the fluctuating electrical activity of the brain, as reflected in the electroencephalogram (EEG), provides the body with a mechanism for attracting KELEA from the environment for transfer to the body's fluids. An extension of this proposal is that individual cells with fluctuating electrical activity may also be providing a direct source of added energy to the cell. Consequently, the depolarization/repolarization process may actually provide a net energy gain to the cell. This concept is consistent with hyper-responsiveness to depolarization being an adaptation to early stages of energy insufficiency in neurons and neurosensory cells. This adaptation may fail with continuing insufficiency of cellular energy (ICE), if it impacts on the mechanisms required for repeated cellular activation. The resulting non-responsive cells can, nevertheless, remain viable. Moreover, the cells can potentially regain sustainable, fluctuating electrical capacity if their ACE pathway is enhanced. Similarly, moderately damaged, electrically hyperactive cells may lose their hyper-responsiveness through corrective normalization of their ACE pathway. Many neurological, psychiatric and other illnesses can be attributed to either increases or decreases of the fluctuating electrical activities of various grouping of cells. The premise of this paper is that many of these illnesses are indicative of an impaired ACE pathway in the abnormally functioning cells. Such illnesses are potentially rapidly and sustainably reversible through the input of additional KELEA and restoration of their normal ACE pathway.

Keywords: KELEA; Water; Insufficiency of cellular energy; Alternative cellular energy; Chronic fatigue syndrome; Autism; Alzheimer's disease; Epilepsy; EEG; Water; Energy based therapy; enerceutical™; Homeopathy; Neurology; Psychiatry; Membrane potential; Cellular electricity

Abbreviations: ACE: Alternative Cellular Energy; ICE: Insufficiency of Cellular Energy; KELEA: Kinetic Energy Limiting Electrostatic Attraction; CFS: Chronic Fatigue Syndrome; CAM: Complementary and Alternative Medicine; EEG: Electroencephalogram; HIV: Human Immunodeficiency Virus; ATP: Adenosine Triphosphate; ADP: Adenosine Diphosphate; mV: millivolt; Na⁺: Sodium Ion; K⁺: Potassium Ion

Introduction

Living cells are normally capable of performing complex, specialized functions, which are appropriate to the type, location and external influences acting on the particular cell. These specialized functions require cellular energy beyond the demands of purely survival metabolism. Cellular damage can occur, which although not leading to cell death, can restrict a cell's ability to undertake its more specialized activities. The term "ICE Cells" refers to cells that are functionally impaired due to an insufficiency of cellular energy (ICE) [1]. An early response to ICE would likely be increased efforts to obtain additional cellular energy. If this were not successful, the energy deficient cells could

simply dispense with their intended specialized activities and enter into a basic survival mode, until the required energy for more specialized functioning became available. This scenario may be particularly relevant to the functioning of cells that undergo fluctuating changes in the electrical potential across their external cell membrane.

Membrane Potential

An electrical potential exists between the outer and inner surfaces of the exterior membrane of all cells [2]. This occurs because the inner cell membrane has more negatively charged anions than positively charged cations, with a more even ratio occurring on the outside of the cell membrane. The resulting voltage differential varies somewhat with different cell types but is in the range of -70 millivolts (mV) for most cells, including neurons [3].

In addition to the difference in electrical charge, the relative concentrations of sodium (Na⁺) to potassium (K⁺) cations differ with Na⁺ being predominantly extracellular and K⁺ being

predominantly intracellular [4]. These differences are maintained by low intrinsic permeability of the cell membrane for these cations, especially Na^+ , and by a membrane transporter, which export three Na^+ cations from the cell in exchange for importing two K^+ cations into the cell [4]. The membrane transporter requires the conversion of adenosine triphosphate (ATP) to adenosine diphosphate (ADP) as the energy source to achieve the Na^+/K^+ exchange.

Separate ion channels also exist for Na^+ , K^+ , hydrogen (H^+), calcium (Ca^{++}), magnesium (Mg^{++}), chloride (Cl^-) and other ions [5]. Induced changes in the intracellular concentrations of various ions have major biochemical effects on cells. Beyond these effects, however, opening of certain ion channels can have a direct effect on the electrical potential of the cell membrane; the most notable of which are the Na^+ channels on neurons. These are evolutionary conserved complexes of relatively large proteins, with considerable glycosylation especially with sialic acid [6,7]. The glycosylation prevents the molecules being brought back into the cell for degradation and re-synthesis. Ion channels can be shed from cells, although it has not been formally shown that the rate of shedding is increased with cellular hyperactivity.

Voltage Gated Ion Channels

The Na^+ ion channels of neurons, muscle cells and some other cell types are normally closed but will open in response to electrical, chemical and/or other modes of stimulation [3]. The extent of the channel opening can vary with regards to the preexisting membrane potential, strength of the stimulus and the presence of other modulating influences. For example, a small reduction in the normal -70 mV difference across the neural cell membrane, renders the Na^+ ion channels more responsive to further electrical or other stimulation. It can also lead to a minor opening of the channel allowing for a low level leakage of Na^+ into the cell. This leakage can be offset by increased activity of the Na^+/K^+ transporter pump. When the reduction in membrane potential reaches a threshold, however, the Na^+ ion channel fully opens, leading to an uncompensated rapid inflow of Na^+ into the cell. The negatively charged membrane potential becomes transiently positive (depolarization). The Na^+ ion membrane channel closes in response to the depolarization. The Na^+ ions, which flowed into the cell are exported via the Na^+/K^+ exchange transporter. Along with a transient, delayed opening of the K^+ channel, the cell reestablishes the preexisting electrically negative membrane potential (repolarization). There is a further delay before the Na^+ channel is again able to reopen in response to lowered voltage [3].

The depolarization of a section of the cell membrane can lead to the opening of the Na^+ ion channels on the adjacent cell membrane [8]. This sequential depolarization allows for a linear propagation of an electrical impulses along a neuron. For myelinated nerves, the electrically-driven depolarizations are limited to the non-myelinated sections of the cell membrane identified as nodes of Ranvier. This is more efficient than the uninterrupted, continuing depolarization process, which occurs along non-myelinated nerves. At neuronal cell junctions (synapses), the incoming depolarization causes the release of neurotransmitters, which chemically depolarize the connecting neurons allowing for the further propagation of the impulse along neural pathways [8].

The depolarization signaling process is also utilized by a range of sensory cells that are linked to neurons, which communicate the sensory information to the brain [9]. The voltage gated membrane channels on sensory cells are typically complexed with molecules that respond specially as receptors to environmental changes. Major classes of sensory cells include those responsive to light, sound, touch, taste, smell, vibration and heat [10].

The brain assigns meaning (interpretation) to sensory inputs, not all of which arise to the level of consciousness. The brain can respond to sensory input by transmitting nerve impulses to peripheral effector cells, including muscle, endocrine and immune cells [11]. The actual response of these effector cells is commonly triggered by the opening of their membrane ion channels. Many of the outgoing nerve pathways comprise the sympathetic and parasympathetic autonomic nervous systems. In addition, the brain can voluntarily, as well as involuntarily send impulses to many skeletal muscles for purposeful movements.

The brain also has so called higher levels of brain activity. These include the storage and recall of memories, self-awareness (consciousness), emotions, formation of images and their interpretations, intention, attentiveness, thoughts, reasoning, perceptions, predictions, etc. The brain also cycles between periods of being awake and periods at different levels of sleeping [11].

The brain has electrical activity, which appears to go well beyond the types of neural networking expected of a highly complex computer. There is an abundance of spontaneous synchronized electrical discharges occurring throughout the brain. This activity is partially measurable in the electroencephalogram (EEG), using electrodes placed onto the scalp. The majority of the brains' measurable electrical discharges have repetitive frequencies, which are typically classified into alpha, beta, gamma and delta waves [12]. Broad correlations can be drawn between the EEG frequencies and the mental state. Other electrical characteristics relate to the amplitudes of the different wave forms, which can vary over time, and to the symmetry of spontaneous wave forms over the left and right sided brain hemispheres.

Causes of Brain Damage

Damage to the brain and to sensory organs can be caused by trauma, inadequate blood, oxygen or nutrient delivery, developmental and genetic disorders, infections, autoimmunity, toxins, neoplasia and aging [13]. Several of these factors can act in concert with one another. Infections can be viral, bacterial, fungal, parasitic or caused by prions. The virus infections can be acute or more chronic and persistent.

Although still not widely acknowledged, persistent brain infections with stealth adapted viruses may play a major contributing role to such common illnesses as the chronic fatigue syndrome (CFS), autism, Alzheimer's disease, bipolar illness and schizophrenia [14-21]. These viruses differ from the conventional cytopathic (cell damaging) viruses from which they are derived, by not being able to evoke an inflammatory response; the normal hallmark of an infectious process. They fail to do so because of the loss or mutation of the relatively few virus genes normally targeted by the cellular immune system [22].

Clinical experience derived from discussions with CFS patients and with parents of autistic children have provided two important insights regarding the underlying disease process. The first is that the severity of the illness in individual patients can fluctuate markedly, even within short periods of time. This indicates that viable cells must be remaining within the damaged areas of the brain, even though the cells may generally fail to function normally. It is not meant to imply that some neuronal cells do not actually die from stealth adapted virus infections. Indeed, severely damaged brain cells have been observed histologically in stealth adapted virus infected patients [16,18] and in virus inoculated cats [23]. The second major insight is that many of the symptoms in CFS patients and in autistic children are indicative of hyperactivity (irritability) of their brain. This again argues for the continuing presence of viable, but dysfunctional neurons, rather than an illness primarily occurring because of cell death. The same consideration can be applied to other illnesses of the brain, which may seemingly be mistakenly labeled as being primarily neurodegenerative, rather than dysfunctional.

Hyperactivity of the Damaged Brain: Is it a Lowered Threshold of Depolarization

Many of the symptoms occurring in patients with neurological and psychiatric illnesses can be attributed to increased activities of groupings of neuronal cells and/or of the sensory and effector cells that interact with neuronal cells. An obvious example is grand mal epilepsy in which depolarizing impulses spread throughout the cerebral cortex [24]. Delusions and hallucinations are also examples of aberrant impulses generated within the brain [25]. So too are several types of muscle tremors and fasciculation. Heightened sensory inputs can manifest as various pain syndromes [26], multiple chemical sensitivity [27], adverse reactions to electromagnetic fields [28], photophobia [29], hyperacusis [30], pruritus [31], paresthesia [32], etc. Inappropriate nerve impulses emanating from damaged neurons can adversely trigger the autonomic nervous system [33]; cause bowel [34] and bladder [35] irritability. Affect endocrine glands [36]; and alter immune responses, potentially promoting allergies [37] and autoimmunity [38].

The efficient internal functioning of the brain relies upon the orderly and meaningful transmission of neural impulses. Heightened responsiveness of neural networks can obscure meaningful signals and can also lead to inappropriate cross-signaling of depolarization events between normally separated neural networks. At a minimum, this can impede clarity of thoughts, actions, memory recall, sensory inputs and peripheral reflex responses.

The most probable explanation for hyper-responsiveness of illness-affected, excitable cells is a reduction of the baseline differential voltage across the cell membrane. This change commonly occurs in damaged, excitable cells. In nerve cells this is accompanied by a low level leakage of Na⁺ into the cell and increased activity of the ATP dependent Na⁺/K⁺ transporter.

Hyperactivity of excitable cells can clearly be disruptive to normal body functioning and would seem at first to be counter-productive, especially in terms of cellular energy. As will be reasoned below, there may be a beneficial, adaptive aspect to the

increased electrical fluctuations of the neuronal cell membrane potential as an energy source for the cell.

Hypoactive Brain

Clearly, if the extent of cell damage exceeds a certain limit then cells can no longer undergo depolarization. A limiting factor is that the high molecular weight, voltage-gated Na⁺ channel is heavily glycosylated, such that it cannot be recycled. Cells that lose their excitability can, nevertheless, remain viable, although if sufficiently damaged they can obviously die. The clinical manifestations of the loss of excitability of neuronal cells correspond to their primary function and location. Prominent examples include the deficits of Alzheimer's and Parkinson's diseases, amyotrophic lateral sclerosis, multiple sclerosis and the negative symptoms of schizophrenia. Loss of function of sensory cells can occur either because of a failure to synthesize the Na⁺ gated channel or the sensory-receptor molecules that interact with the Na⁺ channel. Lack of action potential by sensory cells is the underlying cause of illnesses, such as neurosensory deafness, some cases of blindness, loss of touch and of pain, etc.

Cellular Energy Pathways

The metabolism of food is used by cells to generate ATP from ADP plus phosphate [39]. The resulting ATP is the major source of chemical energy for maintaining cell viability, including the cell's negative membrane potential. Chemical energy also allows for the continuing synthesis of cellular components and for various specialized cellular activities. It is estimated that the brain utilizes approximately 20% of all of the ATP generated by the body from glucose metabolism [40].

It is now clear that food metabolism is not the only source of cellular energy. The body can also acquire energy through the alternative cellular energy (ACE) pathway [41-43]. This pathway is driven by the body's absorption of an external force called KELEA (kinetic energy limiting electrostatic attraction). In Nature, KELEA is seemingly reversibly attracted to separated electrical charges with the proposed fundamental purpose of preventing fusion and possible annihilation of electrostatically attracted opposite electrical charges. The brain is continually undergoing membrane electrical discharge and recharging as reflected in the EEG. It was, therefore, postulated with some supporting observations, that the brain's fluctuating electrical activity, as well as that of muscles, may provide an antenna for attracting KELEA into the body [44]. KELEA is presumably being continually released from the electrically excitable brain and muscle cells to activate the body's fluids. The release of KELEA from separated ions is thought to occur via several mechanisms, including the transient electrostatic bonding of oppositely charged ions. Within the body's fluids, the released KELEA results in the loosening of intermolecular hydrogen bonding [45]. This can facilitate many biological reactions and enhance the flow and penetration of fluids throughout the body. If sufficiently activated, the separated electrical charges on the body's fluids may directly attract KELEA from the environment and possibly via resonance, lead to further activation of the fluid. Under some circumstances, the body can also acquire KELEA through the production of electrostatic mineral binding materials termed ACE pigments [46]. These materials were initially identified in cultured cells

infected with stealth adapted viruses [47]. ACE pigments are also observed in brain biopsies of stealth adapted virus infected patients [16,48] and in tissues of stealth adapted virus infected cats [23]. When present in dried perspiration, ACE pigments have been misidentified as parasites because of their electrostatic properties [49].

Electrically Active Cell as an Antenna for KELEA for their Own Energy Needs

The premise of the present paper is that increased depolarization/repolarization of individual excitable cells may also be in response to ICE. This concept is germane to the above described hyperactivity of damaged neurons and neurosensory cells occurring in various neurological and psychiatric disorders. Moreover, the concept is consistent with examples of rapid recovery, or at least marked improvements in patients receiving energy based therapies. The extent of cellular damage can extend beyond the capacity of cells to respond with increased KELEA attracting electrical activity and/or production of ACE pigments. As suggested above, a possible limitation of continued electrical activity may be the need to replace membrane ion channels and/or their associated receptor molecules. This could occur, for example, if there is increased shedding of ion channels with excitation. Such cells would then become quiescent and, at least for a time, become refractory to further stimulus-mediated ion channel opening. Interestingly, CFS and other patients describe debilitating post-exertional and post-stress associated malaise [50,51]. This could be accounted for by the need to replenish shed ion channels or their associated molecules. Temporarily or persistently unresponsive cells would essentially be precluded by ICE from performing their intended specialized functions and would enter into a survival mode. While cell death could result, the important principle is that recovery from both the hyper- and unresponsive states is potentially possible by providing the damaged cells with additional KELEA to enhance the cells' ACE pathway.

Methods of Activating the ACE Pathway

Complementary and Alternative Medicine (CAM) practices have provided significant benefits to patients with neurological and psychiatric illnesses. Some of the devices used by CAM practitioners emulate the fluctuating electrical activity of the brain with rapid on-off electrical switching. Examples include the Violet Ray of Edgar Cayce; Beam Ray of Royal Raymond Rife; Multi-wave Oscillator of Georges Lakhovsky; pulse electromagnetic field generator of Panos Papas, etc. [43]. The therapeutic benefits of these and similar devices can now be explained as delivering KELEA to the body's fluids. Energy fluctuating devices can also be used for KELEA activation of drinking water. Moreover, the therapeutic uses of numerous natural remedies are consistent with their capacity to attract KELEA into water. Such compounds, termed *enerceuticals*[™], include humic and fulvic acids, zeolites, magnesium oxide, moringa oleifera, cocoa, Brown's gas, various herbal remedies etc., [43]. The compounds can be subsequently removed from the activated water following the activation process by zero residue filtration. Moreover, if water is sufficiently activated, its separated electrical charges can directly attract

KELEA and transmit the energy to added water. This principle explains the effectiveness of certain homeopathic remedies [52]. An important goal of future studies is to better understand the intrinsic capacity of the body to effectively directly attract KELEA from the environment.

Clinical Effectiveness of ACE Pathway Therapy

The field of CAM is replete with anecdotal reports of reversal of the progression of neurodegenerative illnesses. Some of the claims are dubious with strong commercial bias and an unwillingness to provide proprietary details for confirmatory studies. Other studies have been more convincing in documenting favorable clinical outcomes [53,54]. Still, it has proven difficult to attribute the effect to specific interventions, since it is generally agreed that the patient's optimism can be a deciding factor (placebo effect). An understanding of the ACE pathway can be helpful in developing protocols, which are essentially designed to achieve sustainable cellular energy for optimal brain activity. Indeed, dramatic restorations of sensory functions, including hearing, eyesight and taste, have occurred with energy-based devices [1].

Successful ACE based therapies have previously been reported in the therapy of herpes simplex virus (HSV), herpes zoster virus (HZV), human papillomavirus (HPV) and human immunodeficiency virus (HIV) infections [55-57]. Ultraviolet (UV) light illumination of neutral red dye dissolved in KELEA activated fluid was effective in the therapy of children with autism (an illness caused by stealth adapted virus) [58] and in several CFS patients. Other published studies include the ACE pathway based therapy of cancer [59,60] and the treatment of children with tropical diarrhea [61]. The therapeutic benefits seen with these illnesses are likely to be as easily achieved using drinkable KELEA activated water, with regular water as the control. Similar controlled studies need to be conducted in patients with major neurological and psychiatric illnesses.

The minimal costs and lack of toxicity of ACE pathway based therapies argue for their universal use throughout life in the prevention of illnesses, including the possible delay of aging senescence. Results from ACE pathway based animal and agricultural studies should help contribute to the optimization of human studies.

Conclusion

It is proposed that electrically excitable cells may derive cellular energy for their own needs as well as contributing to the energy needs of the body by using the fluctuating electrical charges of membrane depolarization/repolarization to attract an environmental force termed KELEA (kinetic energy limiting electrostatic attraction). An initial adaptation to an insufficiency of cellular energy (ICE) can be an increased rate of depolarization by electrically excitable cells. With further ICE, the cell may become electrically quiescent and adopt an essentially survival mode without engaging in specialized cellular activities. These reversible phases of illness precede irreversible neurodegeneration, characterized by death of neuronal cells. Therapy of the early phases of illness should comprise efforts at enhancing the alternative cellular energy (ACE) pathway. The

consuming of KELEA activated water can potentially achieve self-sustainable cure of severe neurological and psychiatric illnesses. Moreover, this approach may directly assist in the cellular suppression of brain infections with stealth adapted viruses.

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