

Analysis: Magnetic Resonance Targets Telomeres/Telomerase for Cancer Treatment?

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Abstract

This paper essentially poses the question, "Is it possible to calculate target-specific magnetic resonance energies to restore bio molecular order and inhibit telomerase production under conditions of increasing entropy, even as cells pass M2 checkpoints?" Indeed, a diversity of positive experimental outcomes has pointed to the possibility that non-ionizing radiation (NIR's) produce positive bio effects of predictable nature. The hypothetical construct is that Pico-Tesla range magnetic fields are physiologic and may possibly affect molecules and molecular assemblies through the piezoelectric effect; most especially due to the supposed quasi-crystalline semi conductive nature of various biological structures. The initial physical mechanism is hypothesized to be photon-phonon transductions, i.e., electro mechanical conversions, via resonance phenomena. Utilizing a novel particle-wave equation, $mc^2=BvLq$, specified magnetic flux densities have been calculated, formulating a possible system of dual resonance. The energy produced by the interaction of the organism and the magnetic field is set equal to the intrinsic energy of a molecule. The outcome of this methodology may possibly provide a new, non-invasive holistic paradigm for adjuvant treatment of cancer. In support of said hypothesis, various experimental outcomes are considered, wherein the given method was utilized to establish magnetic resonance protocols, based on molecular species known to be associated with pathophysiologic conditions including cancer. Thus, if successful, then a possible new adjunctive, non-invasive and non-significant risk approach for telomerase inhibition with conventional cancer therapy is proposed.

Keywords: Cancer; Magnetic resonance; Telomeres; Telomerase; Piezoelectric effect; Jacobson resonance

Introduction

There has been a collective and persistent view in the physical and engineering sciences that non-ionizing electromagnetic fields (EMF's) many orders of magnitude below kT are incapable of inducing biological effects [1]. Even if one accepts that ligand-receptor association alters conformational states of extruding portions of intra membranous proteins (IMP's) at cellular surfaces, and that communication of changes can be transmitted to the cytoplasm by non-linear vibrations of helical proteins (solitons), one may still be unable to account for restoration of biological order [2,3]. Nevertheless, microtrabecular reticulum receptors are associated with actin filaments and ATP molecules, therein contributing to activation of cyclase enzyme systems through piezoelectricity [4]. Yet, when one considers the hydrophobic nature of IMP's, and energy dissipation after each photonic interaction, current models are wanting in structuring and thermodynamical properties to maintain sufficient energy states necessary for amplification by a factor of 10^{12} (the vibrational frequencies of proteins). It is therefore proposed that single system perturbations may induce photon-phonon transductions to provide direct enhancement of molecular vibrational energies responsible for biological amplifications of weak triggers. In other words, there may be another kind of interaction occurring, not dependent upon the photonic interaction at the membrane surface. Classical electrodynamic theory does not yield a model for biomolecular resonance responsiveness to low intensity, extremely low frequency (ELF) electromagnetic fields, but quantum mechanics does [5]. It is noted that quantum mechanics yields statements relating to discontinuity of transitions from one total system to another. It does not yield a representation of any specific process. This affords to notion that quantum mechanics doesn't operate with single systems, but with totalities of systems. Taking into account this interpretation of quantum mechanics, it is evident why quantum mechanics does account for the notion that very weak

disturbing forces are capable of yielding changes of any magnitude to physical conditions of systems. Weak forces may provide interference to electromagnetic interactions, wherein exceedingly small changes to the statistical density within the ensemble of said systems occurs. Thus, infinitely small alterations of the single system e.g. non-ionizing electromagnetic wave interactions with living systems, may account for the profound amplifications within complex systems. Our analysis may provide a new method by which one may acquire a glimpse into the nature of critical alterations of the single system. In consideration of biological entropy, it is noted that the environment is critically important to maintain a reversible system, i.e., such that infinitesimal changes of extrinsic or intrinsic nature can make the difference in retaining homeostatic function and biological order [2,5-13].

Background

Non-Ionizing electromagnetic field effects in biological systems

Over the past 35 years, scientists have demonstrated that externally sourced, extremely weak, ELF EMF's many orders of magnitude below the potential within the pericellular fluid do indeed modulate actions of antibodies, hormones and neuromodulating molecules at cell surface receptor sites. Recorded sensitivities are as low as 10^{-7} volts per centimeter in the extremely low frequency spectrum [3,14,15].

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Examples of subtle electromagnetic field effects include: altered rate of cellular growth, [16] increases in ornithine decarboxylase, a growth related enzyme [17], suppression of T-lymphocyte cytotoxicity [18], changes in quantities of RNA transcripts and proteins [19-21], changes in cell surface properties [15] effects on translated developmental processes [22] and healing of ununited fractures and arthrodeses that failed with conventional therapy [23].

The existence of the human brain's Pico-Tesla (PT) range magnetic field profile was first established by the seminal work of David Cohen [24]. Using the superconducting quantum interference detector (SQUID) he determined that alpha brain waves were associated with a magnetic field flux density of 5×10^{-9} Gauss (0.5 PT). He also discerned a DC magnetic field of 5×10^{-8} Gauss or 5 PT. The heart's maximum magnetic flux density was about 5×10^{-7} Gauss or 50 PT. The maximum magnetic field of the brain was found to be about a microgauss (100 PT) by Anninos and Tsagas [25]. Indeed, the differential between the magnetic profile of a normal brain vs. a pathologic brain was determined by Anninos [25] with study of Parkinson's disease. Considering the remarkable fact that one PicoTesla is fifty million times weaker than the Earth's steady magnetic field (at 0.5 Gauss or 5×10^{-5} Tesla), the collective view was initially that these extremely weak magnetic profiles represented noise emanating from stronger interactions within the body.

The prediction that Pico Tesla electromagnetic fields (PTEMF's) are physiologic was originally based on a particle-wave equation, $mc^2 = BvLq$ (Jacobson Resonance) [26-30]. Jacobson Resonance was established to enable calculation of target-specific magnetic fields to affect molecular species associated with pathophysiological conditions. Thus, the clinical work of Anninos et al [31] began with the notion that an externally applied physiologic magnetic field might renormalize the brain's magnetic profile in epileptic patients. Equipped with a SQUID, they obtained the initial magneto-encephalograph (MEG) profile providing the foci, frequency and intensity of the abnormal brain magnetic profile [32]. They used an array of small coils contained in a plastic flexible patch held about a patient's cranium for treating epilepsy, MS, and Parkinson's disease [31-33]. They observed positive outcomes; when entraining the brain with slightly stronger renormalizing physiologic PicoTesla magnetic fields. Jacobson and Yamashita [34] established clear mathematical correlations of $mc^2 = BvLq$ to other well-known resonance phenomena, including cyclotron resonance and Zeeman resonance. At the very least, the hypothesis of biological quantum gravity was founded [13,35]. Subsequent to the first tier of PTEMF research, NASA engineers at the John C. Stennis Space Center prototyped various biomedical devices to further evaluate the potential for holistic methods in PTEMF resonance therapy. These devices of Helmholtz configuration would also be utilized in basic scientific research employing the physico-mathematical construct of Jacobson Resonance.

Thus, PicoTesla magnetic resonance therapy was enabled by a capacity to calculate specified magnetic flux densities focused on relevant molecular species and/or electromagnetic domains. PicoTesla magnetic resonance energies have been demonstrated to affect human brain waves [25,31-33] enhance regeneration of peripheral nerve ultrastructure in mice while restoring fore-limb grip strength functionality [12,36] regulation of autonomic nervous system tone, e.g. increase parasympathetic innervation to the canine heart, and mechanisms of conduction, affecting the rate and rhythmicity [5,7,12] demonstration of decreased pain and improvement of range of motion in osteoarthritic knees for patients in a double

blind, randomized and placebo-controlled study [9] modulation of endogenous opioid action (enkephalin, endorphin) [11] affectation of benefits in Parkinson's disease subjects seen in a Phase II double-blind, randomized and placebo-controlled trial [5,37] demonstration of benefits in various case controlled studies for epilepsy and multiple sclerosis subjects [31,32,38] speeding wound-healing in sutured and non-sutured wounds [10] regulation of thoracic spinal neuronal potentials subsequent to administration of inflammatory chemicals to the rat heart (with stimulation of nociceptive afferent fibers) [11] and decreased viability and/or proliferation rate of human breast carcinoma cells in-vitro [5] reduction of pain and stiffness in fibromyalgia subjects in a double-blind randomized and placebo-controlled study [35] just to cite some of the studies that were conducted under rigorously controlled conditions. The hypothesis that PicoTesla magnetic fields are physiologic did gain impetus, as the novel particle-wave equation, $mc^2 = BvLq$, was ostensibly predictive. Still, the physical mechanism underpinning observed bioeffects in the aforementioned studies did not satisfy a collective view. The mechanism remains unknown.

The possible connection of biological piezoelectricity to cancer

Imagine having a quartz crystal sandwiched between metal plates, wherein the plates are connected to a source of alternating voltage having a particular frequency. The quartz crystal will vibrate mechanically at the same frequency. The semi-conductive crystal changes alternating electrical energy, i.e. electromagnetic oscillations, into vibrational mechanical energy. This is known as electromechanical transduction, or the piezoelectric effect. There are many biological structures now conceived to be piezoelectric in nature, such as bone, keratin, alpha and beta sheaths of proteins, microtubules, centrioles, and even genes [4,39-41]. When an atomic crystal lattice structure of a semiconductor is subjected to mechanical pressure or temperature alteration, charge displacement occurs on crystalline faces, resulting in potential differences across the faces. Conversely, when an electromotive force is applied between crystalline faces of semiconductors, the crystals become dimensionally deformed. Now, a biological analog can be extrapolated, in that the diameters of collagen fibers in tendons increase with the growth of a maturing animal. The collagen fibers reflect the tensions to which they are subjected. When a tendon is severed, it is known that DNA synthesis increases in the first day within the peritendinous sheath [8]. Additionally, neuromagnetic measurements of response to auditory stimuli (pure tones) have been used to ascertain the location of cortical activity. The evoked field source increases in depth beneath the scalp with increasing frequency of the tone [8]. Filamentous structures may be viewed in a new light, considering (1) current knowledge concerning photo and infra optical emissions of biological systems, (2) connections between ordered electron and protonic flows associated with semiconductor biostructures, (3) protein structure, e.g. filament shaped assemblies of globular proteins, e.g. especially alpha helices and beta sheaths and (4) H-bond reticulum [4].

Piezoelectric mechanisms appear to be spontaneously present in physiology, as in specialized cells concerning reception of external stimuli (pressure, heat and sound). Such specialized cells may convert the types of energy they are sensitive to, into electrical energy of nervous impulses. Franco Bistolfi [4] points out that proteins containing more than one alpha helix have a nearly spherical polyhedral geometry, whereas such structures are quasi-crystalline, suggesting piezoelectric effects.

A most remarkable possible example of piezoelectricity, i.e., photon/phonon transductions, follows: One of the electrical responses

of the cochlea to sound is the cochlea microphonic, a potential fluctuation recorded between an electrode on or near the cochlea, and an indifferent electrode. This is generated by a deformation of the processes of hair cells, and is linearly proportional to the magnitude of the displacement of the basement membrane. Consequently, it reproduces the waveform of sound stimuli. The reproduction of the frequency and the magnitude of the sound is so accurate that if music is played into an animal's ear, a faithful rendition of the music can be produced by feeding the cochlea microphonic into an audio amplifier. The cochlea microphonic can be recorded from the auditory nerve close to the cochlea, but it is not a neural response. It can be obtained from a recently killed animal in which neural activity has ceased. It is construed to be produced by mechanical distortion of the hair cells exemplifying the piezoelectric effect in biological matter [4,8].

In Barbara McClintok's system [42] controlling elements of genes do not correspond to stable loci on chromosomes. They change position. Transposition is a property regulated by regulator genes and activator genes. Viral DNA can insert itself into host DNA and thereafter detach itself. Normal DNA of a cell can also rearrange itself. This regulatory capacity is subject to influence by the cell's environment. It has been conceived that information flow is not unidirectional, but flows to and from genes that vibrate in space as they communicate via photonic force carriers; and regulated by elements derived from the root of life itself [8]. Researchers have catalogued numerous chromosomal changes that appear in association with certain cancers. Connections have been elucidated between oncogenes, discrete transforming DNA sequences (350 base pairs to 1000 base pairs), translocations, deletions, and other carefully studied chromosomal alterations. An example refers to the MYC oncogene, moving from the normal position in chromosome 8 to a region in chromosome 14, where it is positioned near genes for immunoglobulins. Notably, genes continue to structurally change throughout the life cycle. This was demonstrated by Susumu Tonegawa [43] as he outlined the genetic principle of antibody diversity. Polypeptide trophic factor and oncogene research did converge when homology between some oncogenes and growth factors (or their receptors) were indeed shown through sequence analysis. Transformations of cells occur due to excessive synthesis or altered versions of polypeptide growth factors (or their receptors). Also, certain oncogene products can induce differentiation of recipient cells. This calls attention to the complex interplay between transforming and differentiative processes. The example of H-ras and V-src, whose expression in PC-12 cells results in mitotic arrest and neuronal differentiation, is comparable to inductions by NGF. Peptide hormone growth factors also share a common mechanism of action with viral oncogene protein products. Rita Levi Montalcini [44] maintained that whenever cell death of specific neuronal populations is connected to the availability of growth factors such as NGF, its exogenous supply or endogenous production may offer an approach to cancer [44,45].

It appears that cancer may no longer be considered more than 100 diseases, each characterized by a different tumor type heterogeneous on a cellular level. Instead, it appears that there may be a small number of atomic/molecular mechanisms manifesting essential common denominators in all tumor types. We note that the various biological structures of importance may possibly be piezoelectric in nature, susceptible targets for magnetic resonance therapy. It is conceivable that restoration of biological order may be possible, to therein induce quantum transpositional states such that the electromagnetic and biochemical immunological functions of the whole organism can renormalize itself [2,4,8,13,46-50].

Sampling of studies demonstrating possible target-susceptibility to calculated magnetic resonance energies

$Mc^2=BvLq$ (Jacobson Resonance) is utilized to establish the magnetic flux density (B) of an exogenously sourced magnetic field to the whole organism, with (L) as its longest dimension. (Mc^2) represents the intrinsic, or rest energy of a target mass (m) which may be any atomic or molecular species, e.g. peptide hormone growth factor, enzyme, neurotransmitter, DNA sequence such as a telomere (TTAGGG), or any immunogenic mass. (BvLq) represents an electromagnetic interaction energy (wave energy), whereby the body of length (L) containing the mass (m) interacts with the magnetic field (B) to establish a system of dual resonance, i.e., $mc^2=BvLq$. It is speculated that a coherent excitation is produced in the target mass (m) via a photon-phonon transduction, i.e. the piezoelectric effect (v) is any inertial velocity such as Earth's orbital velocity, because Newton's second law of motion doesn't distinguish between celestial and terrestrial; velocity; and (q) represents a unit of electrical charge, or a single ab-coulomb in the CGS system; established by defining electromotive force as energy per unit charge. A more detailed description of the physico-mathematical model is accessible in the literature; including the derivation, rationale for variables m, B, v, L and constants c and q; as well as the correlations to other known resonance phenomena: cyclotron resonance and Zeeman resonance. (c) is the velocity of light, and also the velocity of the force carrier (photon) for a magnetic field, moving independent of the inertial frame of reference or source.

A sample calculation follows to show how the theory is applied to biological systems. Once the magnetic flux density (B) is derived from $mc^2=BvLq$, the derived (B) field is inserted into the cyclotron resonance equation, $f=qB/2\pi m$, to establish the associated frequency [2,5,8,13,34,35].

Neuroelectromagnetics

Study sample in parkinson's disease and calculation

Nerve growth factor (NGF) exhibits trophic influences on many populations; promoting survivability, synaptic transmission regulation and plasticity at adult synapses in various regions of the central nervous system; and homeostatic regulation of intrinsic neuronal excitability. Nerve Growth Factors contains anti-apoptosis inducing segment which prevents cell death. Selecting NGF as a target mass, we consider the following:

- (1) NGF is 26,500 Daltons, or 4.425×10^{-20} gram
- (2) $9 \times 10^{20} \text{ cm}^2 \text{ sec}^{-2} = c^2$
- (3) (L) is the average height of a human, or 177 cm.
- (4) Earth orbital velocity is $3 \times 10^6 \text{ cm sec}^{-1}$ (V)
- (5) (q) is one ab-coulomb (a single unit charge by definition)

The CGS system of physical units is selected, while in the MKS (SI) system force is established between moving charges, whereas in the CGS system force is determined between stationary charges. Thus, we desire:

$$Mc^2=BvLq$$

$$(4.425 \times 10^{-20} \text{ gm}) (9 \times 10^{20} \text{ cm}^2 \text{ sec}^{-2}) = (7.5 \times 10^{-8} \text{ Gauss}) (3 \times 10^6 \text{ cm sec}^{-1}) (177 \text{ cm}) (\text{ab-coulomb})$$

Then, we notate that (q) is normalized in CGS. Consequently, when converting from CGS to the MKS system, $mc^2 = BvLq$ becomes

$mc^2 = BvL$ (10q), because one ab-coulomb is equal to 10 coulombs. Therefore, when using the MKS expression, $f=qB/2\pi m$, we use $f=10 qB/2\pi m$, and we note:

$$f=2.1 \text{ Hz}$$

(q) is the electron charge and (m) is the electron mass.

Normalization allows the process of introducing a numerical factor into an equation and is utilized in quantum mechanics. Additionally, the signal, 7.5 Pico-Tesla @2.1 Hz, has been successfully used in the treatment of Parkinson's disease. (PD) improving the quality of life for patients. [5,37]

A clinical pilot study in PD with subjects experiencing motor fluctuations was conducted to determine safety and tolerability with fine tuning of treatment parameters; to ascertain the appropriate PicoTesla Electromagnetic Field (PTEMF) protocol. Thirteen subjects (9 women, 4 men) with an average age of 62 +/- 13 years and PD duration of 11.7 +/- 7.4 years completed the study. Treatment sessions lasting 1 hour and 20 minutes were performed 3x/week for a minimum of 5 weeks. No study related adverse effects were noted, and all subjects demonstrated improvement in multiple areas of evaluation in this pilot study.

Anecdotal improvements were reported by multiple subjects in the following areas: decreased fatigue, improvement in the quality and quantity of sleep, enhanced sense of smell, increase in the "on" time, and ability to return and/or increase participation level in activities, e.g. golf and driving [5]. Subsequently, a single center, double blind, randomized, placebo controlled trial utilizing PTEMF as an adjuvant to standard medical therapy in PD patients with motor fluctuations was performed in 12 subjects (6 per group). 24 sessions of 1.5 hours of total body PTEMF were administered over 8 weeks. Standardized motor and non-motor assessments were performed prior to treatment, at endpoint, and monthly for 3 months. The treatment group demonstrated significant improvement over placebo after 8 weeks of therapy.

Importantly, statistically significant improvement on several scales persisted up to 2 months post treatment. No treatment related adverse events were reported. Concluding, PTEMF may improve motor and non-motor features of Parkinson's disease beyond that achieved with standardized medical therapy. The effects are long lasting. Larger double-blinded, placebo-controlled studies are indicated to confirm and continue to investigate the potential benefit of this unique, noninvasive and promising therapy [37].

The signs and symptoms of PD result from a widespread neuronal degeneration, resulting in a downstream cortical and subcortical dysfunction. We may postulate that beneficial effects of PTEMF result from piezoelectric effects. Synaptic activity in neurons at various levels of dysfunctional cortical-basal ganglionic loops in PD may be favorably influenced. Further research will be needed to clarify the mechanism of action.

Cardioelectromagnetics

Specific study sample and calculation

Vasostatin-1 was chosen as the target molecule for anti-arrhythmogenic studies for its inhibitory effects on the cardiac autonomic nervous system (CANS), particularly the adrenergic component. Vasostatin-1 is a recently recognized cardio regulatory peptide with diverse actions, including anti-adrenergic and anti-

inflammatory effects. It was shown that vasostatin-1 injection into the major atrial ganglionated plexi (GP) suppressed atrial fibrillation (AF) inducibility and inhibited the activity of the intrinsic CANS. These results indicated that vasostatin-1 mimicked the anti-arrhythmic effects of low-level vagal nerve stimulation (LL-VNS), supporting the notion that vasostatin-1 may be one of the critical neuromodulators mediating the effects of LL-VNS on AF susceptibility and intrinsic CANS inhibition. Studies were done on adult dogs. Therefore, we consider:

1) Molecular weight of VS-1=7000 Dalton or 1.169×10^{-20} gram.

2) L= 100 cm (length of an adult dog)

3) $C^2= 9 \times 10^{20} \text{ cm}^2/\text{sec}^2$

4) V= Earth orbital velocity = $3 \times 10^6 \text{ cm/sec}$

5) q = one ab-coulomb, by definition. Thus, $mc^2=BvLq$:

$$(1.169 \times 10^{-20} \text{ gm}) (9 \times 10^{20} \text{ cm}^2 \text{ sec}^{-2}) = (3.4 \times 10^{-8} \text{ Gauss}) (3 \times 10^6 \text{ cm sec}^{-1}) (100 \text{ cm}) (1 \text{ ab-Coulomb})$$

Then, $f=10qB/2\pi m$ yields: 0.952 Hz

The use of non-ionizing PicoTesla electromagnetic fields to suppress atrial fibrillation was studied by Yu [12] at the University of Oklahoma Heart Rhythm Institute. It was considered that extremely low-level EMF's were proposed to cause significant changes in neural networks, and it was decided that investigation of the effects of PTEMF's on arrhythmias would manifest positive outcomes. The question was asked, "Can PTEMF's suppress atrial fibrillation?" In seventeen dogs anaesthetized with pentobarbital, bi-lateral thoracotomies permitted the placement of multi electrode catheters in both atria and at all pulmonary veins. Atrial fibrillation, (AF) was induced by rapid atrial pacing (RAP) or programmed atrial extra stimulation. At baseline and at the end of every hour of RAP during sinus rhythm, programed atrial stimulation afforded the effective refractory period (ERP) and the width of the window of vulnerability (WOV). WOVI is a measure of atrial fibrillation inducibility. Microelectrodes were inserted into anterior right ganglionated plexi (ARGP) and recorded neural firing. Helmholtz coils were utilized, powered by an HP generator that produced the PTEMF signal parameters shown in the aforementioned calculation, i.e. 0.034 micro Gauss at 0.952 Hertz. The study sample was divided into two groups. Group 1, (n=7) included application of PTEMF to both cervical vagal trunks. Group 2, (n=10) included application of PTEMF across the chest so that the heart was located in the center between the coils. In Group 1, PTEMF induced a progressive increase in atrial fibrillation threshold at all pulmonary veins and atrial sites ($p < 0.05$).

Group 2 initially revealed an atrial ERP which was progressively shortened; and ERP dispersion and WOVI progressively increased ($p < 0.05$) compared to baseline values during three hours of RAP. Then, the next three hours included combined application of RAP plus PTEMF, which increased ERP and decreased the WOVI until the end of the third hour. The amplitude and frequency of neural activity recorded from the ARGP were significantly suppressed by PTEMF in Groups 1 and 2 [5,7,12, 51-53]

Preliminary Analysis of Cancer

Fissionary processes in nature may appear spontaneously as a consequence of quantum asymmetries, and it does appear possible to draw comparisons between the splitting of a uranium nucleus and normal cellular mitosis, from a quantum phenomenological

perspective. However, when a uranium nucleus splits, two lighter elements are produced with differing atomic masses, but when a cell undergoes normal mitotic division, two identical daughter cells are the product. Thus, the question is posed, "How can a biological system that undoubtedly exhibits highly non-linear, non-equilibrium quantum states (ostensible asymmetrical patterns) be able to essentially reproduce cellular products so perfectly symmetrical and identical?" Perhaps this conundrum may be at least partially explained via the global analytical purview. The human body is a veritable universe of electromagnetic interactivity, comprised of trillions of atoms that are incessantly communicating through the long range electromagnetic force carried by photons. Thus, it appears possible to consider the human body as an intrinsically regulated global system, the micro components of which oscillate about a steady state system, incessantly in flux to maintain equilibrium and homeostatic functionality. Indeed, reversible processes from a thermodynamic point of view keep the whole system viable. Yet, reversibility is an idealization, and quantum state entropy always increases in small volumes of space, with energetic states continuing to produce microscopic scarring, oxidative damage, desiccation and aging. This leads to consideration of the ongoing shortening of telomeres, our biological genetic clocks.

While one might predict that without additional cellular changes, signals from damaged DNA associated with telomere shortening (telomere uncapping) could provide tumor suppression, since such damage is irreparable in the absence of telomerase. Replicative senescence, as well as oncogene-induced senescence, can stop cells from proliferating. Yet, when alterations of specific cell-cycle regulatory pathways, e.g. p53 inactivation and p16/pRB pathway inactivation, cells may continue to divide even with critically shortened telomeres; still expressing activated DNA damage pathways. When bypass of senescence occurs (M1), there may occur an extension of cellular lifespan. Indeed, there might occur progressive reduction of telomere length. When telomeres become extremely short that end-end chromosome fusions occur, producing chromosome-breakage-fusion bridge cycles, the M2 or crisis stage is reached. This leads to extensive chromosome alterations, the hallmark of cancer. It is speculated that such transpositional states are based in quantum entropic environmental conditions, thus leading to transitional alterations of cell-cycle checkpoints, thereby encouraging cells to divide to meet energy exigencies. Therefore, terminally shortened telomeres might not always prevent continuing cellular divisions in a rarity of cells, enabled to bypass critical stages. This rarity of cell type manifests telomeric stability, with reactivation in telomerase production. Yet, the fundamental mechanisms underlying the bypass of senescent checkpoint M1 and continuation to the crisis point M2, may be based on quantum conditioned states ultimately determining biochemical changes. Actively dividing cancer cells could survive and continue to replicate. Recapitulating, human telomeres are repetitive non-coding structures at the ends of chromosomes that are bound by a series of single and double-strand DNA - binding proteins. Telomeres shorten with every cellular division based upon incomplete lagging strand synthesis, but much less understood are end processing events and oxidative damage. Quantum state interferences are produced by a host of environmental stressors [5,6,54-60].

Consideration is then given to the idea that an electromagnetic globally regulated equilibrating immune response provides autoimmune encouragement for a rarity of cells to divide while avoiding crisis stages, in spite of exhibiting terminally shortened telomeres. This idea is provided for contrasting normal mitosis vs. neoplasia, i.e., the distinction between self and non-self might be immunologically

mischaracterized. The basis is derived from a global electromagnetic miscommunication. The mechanism for telomeric mechanical error and electromagnetic immune function may well be the piezoelectric effect, because the binding protein may be piezoelectric in nature.

It is hypothesized that electromagnetic fields may stimulate up regulation or inhibition of telomerase when telomere length diminishes too greatly in specific tissues, while functionality has diminished. Telomerase is hypothesized to be a defensive mechanism of our body, because telomerase increases telomere length. When telomeres become too shortened, telomerase is naturally up-regulated to increase telomere length, but in crisis stages, M1 -M2, entropic states are too great for the defense mechanisms to overcome anarchy of cells when they would normally die. Thus, telomerase produces increased lengths of telomeres in actively dividing cancer cells, hence keeping the cancer cells surviving and dividing. As actively mitotic cells increase energies from fissions/ fusions of subatomic particles, cancer cells increase energy. Therefore, the cancerous chain reaction proceeds. The cancer cells have superseded the normal cells while using the natural defense mechanism of normal cells to survive and multiply. The level of chaos/ quantum entropy from fissions/fusions overcomes the natural defense mechanism based in telomerase production. In addition, if piezoelectric magnetic resonance profiles can provide electroporation of tumors, PTEMF therapy might prove efficacious as an adjuvant modality to kill cancer stem cells, while utilizing conventional chemotherapy and/or immunotherapy.

Specific Target Mass Magnetic Resonance Profiles for Research

We may ask whether magnetic resonance energies could maintain the structural/functional integrity of telomeres and/or telomerase. Telomeres are basically biological clocks that decrease in length with age, and once a critically shortened telomeric length is attained, cellular senescence may become activated. When sufficient numbers of cells undergo senescence in a tissue, a decline of function will occur that would contribute to aging or cancer. Since telomeres shorten due to incomplete lagging strand synthesis, and when conformational states of proteins and/or DNA are altered, this information may be transmitted to the rest of the DNA via piezoelectric communications. Single or double strand DNA- binding protein and/or DNA may be targetable. We shall consider the primary structure of a telomeric unit and multiples thereof as our target masses.

Nevertheless, the questions become: Can we piezoelectrically stabilize targeted telomeres and/or binding protein (while enhancing biological order) to then supersede the requirement of actively dividing cancer cells for telomerase up-regulation? Then, if the foregoing is possible, will tumors then stop growing secondary to telomerase inhibition? And, if the piezoelectric magnetic resonance energies provide electroporation of tumors, will chemotherapy and/or immunotherapy then prove more efficacious in killing cancer stem cells? Finally, if a holistic approach is taken, i.e. full body immersion in the magnetic field; will it then be possible to inhibit the growth of metastatic lesions? [13,56]

We note the telomeric prime:

(TTAGGG) Whereas, T=258.164; A=267.176; G=299.176; and TTAGGG=1,681.032 Daltons (Da). Thus, the molecular mass of the prime telomere unit is 1,681.032 DaDa or 1.67×10^{-24} gram. Considering $Mc^2=BvLq$:

$$(1,681.032 \text{ Da}) (1.67 \times 10^{-24} \text{ gm}) (9 \times 10^{20} \text{ cm}^2 \text{ sec}^{-2}) = mc^2$$

$$= (B) (3 \times 10^6 \text{ cm sec}^{-1}) (177 \text{ cm}) (1 \text{ ab-coul}) = BvLq$$

$$B = 4.7581 \times 10^{-9} \text{ Gauss (0.47581 PT)}$$

Now, $f = 10 \text{ qB}/2\pi m$, and we desire:

$$\text{And, } f = 0.133 \text{ Hz}$$

Therefore, the essential signal parameters for the telomere prime unit are:

$$4.7581 \times 10^{-9} \text{ Gauss @ } 0.133 \text{ Hz}$$

We must consider the foregoing as the prime set of signals, because the domain walls of the essential target represent magnetic interfaces of energy domains. Integral multiples of the prime may then be utilized within the context of a master protocol to specifically influence tissues of concern. For example, the protocol used successfully to diminish the viability of human mammary carcinoma cells included integral multiples of the prime telomere signals starting from about 15 PicoTesla incrementally decreasing to about 3 PicoTesla. Included within the context of this master protocol were interleukins and interferons as well as TNF. Most interesting is the fact that integral multiples of prime telomere signals not only include signal sets that may target cytokines but also specific tissues such as tendon (15PT @ 4.2Hz), brain (7.5PT @ 2.1Hz) and heart (3.4 PT @ 0.952Hz) [5,6]. Considering the flux density of PTEMF's used to treat Parkinson's disease, we note that the B value was about 7.5 PT, whereas the B value utilized to affect cardiac rate and rhythm was about 3.4 PT in experimental studies at the University of Oklahoma Health Sciences Center [2,5,12,37]. We also note that telomerase associated proteins such as NOP10, NHP2, GARI and Dyskerin reveal molecular masses analogous to telomere prime integral multiples, cytokines and tissue specific magnetic resonance energies including the brain and the heart.

In the absence of neoplasia, our essential hypothesis is that tissue specific PTEMF's (empirically determined) may affect telomeres of specific lengths (and specific molecular masses), to therein provide up-regulation of telomerase; with distinct relationships to other critical molecules. Table 1 shows integral multiples of the prime telomere unit (TTAGGG), associated flux densities, frequencies of magnetic resonance energies, and the molecular mass analogs.

Therefore, the essential signal parameters for the telomere prime unit are:

$$4.7581 \times 10^{-9} \text{ Gauss @ } 0.133 \text{ Hz, equivalent to the measured B value and f value of alpha brain waves.}$$

Indeed, we executed studies at Mississippi State University and screened PicoTesla range magnetic field schedules [2,5,13,63] Calculations using the Jacobson Resonance equation were based on multiples of the telomere prime and molecules considered to be associated with human mammary carcinoma cell populations (HTB-126 and MCF-7). Multi well tissue culture plates were employed. Two protocols utilizing Table I signal sets were discerned to compromise the viability and/or proliferation rate of HTB-126/MCF-7 cell types relative to untreated reference controls. Over the course of replicate studies (n=7) these PicoTesla range protocols were seen to consistently inhibit the viability and/or proliferation rate from 31% to 35% compared to untreated reference controls. We also identified membrane-associated complexes that were expressed at elevated or decreased levels in the MCF-7 populations. Several mRNA sequences were noted (n=3) that were expressed at higher levels (n=1) or uniquely expressed (n=2) in MCF-7 populations. Importantly, these cells were exposed to magnetic resonance wave energies for only thirty minutes

each time in five treatment sessions, compared to exposure times of 56 minutes, twice weekly, for 8.5 weeks as was executed for nerve regeneration studies at Fairleigh Dickinson University. The nerve study was a positive *in vivo* study in mice demonstrating restoration of radial nerve ultrastructure [2-5]. Our experience informs that outcome measures are directly related to exposure time- in addition to conditions of resonance determined by accuracy of flux densities and frequencies. Consequently, replicate cancer cell studies using increased exposure times are no doubt indicated.

Collective interpretation of experimental findings revealed an ability of a multi-amplitude, multi-frequency, PicoTesla range magnetic protocol to induce alterations in viability/proliferation rate and expression profiles of: (a) cytosol-soluble as well as membrane associated protein fractions; and (b) transcription of mRNA sequences compared to non-exposed controls. In this context, these changes appeared to be of different patterns when experimental samples were hastily processed following MCF-7 exposure to the final protocol. In contrast, different and somewhat more subtle differences were appreciated when an intentional delay was implemented between the final exposure and the sample preparation. This observation implied that maximum alterations in protein expression and mRNA transcription may occur during or shortly after exposures. Additionally, there was also a relative difference in the affect excited by individual flux density/frequency approaches contained within "Master" multi-amplitude,

S.No	$\beta(\mu\text{G})$	f(Hz)	Mass(kDa)
1	0.004758100	0.133174371	1.681032000
2	0.009516200	0.266348742	3.362064000
3	0.014274300	0.399523113	5.043096000
4	0.019032400	0.532697484	6.724128000
5	0.023790500	0.665871855	8.405160000
6	0.028548600	0.799046226	10.086192000
7	0.033306700	0.932220597	11.767224000
8	0.038064800	1.065394968	13.448256000
9	0.042822900	1.198569339	15.129288000
10	0.047581000	1.331743710	16.810320000
11	0.052339100	1.464918081	18.491352000
12	0.057097200	1.598092452	20.172384000
13	0.061855300	1.731266823	21.853416000
14	0.066613400	1.864441194	23.534448000
15	0.071371500	1.997615565	25.215480000
16	0.076129600	2.130789936	26.896512000
17	0.080887700	2.263964307	28.577544000
18	0.085645800	2.397138678	30.258576000
19	0.090403900	2.530313049	31.939608000
20	0.095162000	2.663487420	33.620640000
21	0.099920100	2.796661791	35.301672000
22	0.104678200	2.929836162	36.982704000
23	0.109436300	3.063010533	38.663736000
24	0.114194400	3.196184904	40.344768000
25	0.118952500	3.329359275	42.025800000
26	0.123710600	3.462533646	43.706832000
27	0.128468700	3.595708017	45.387864000
28	0.133226800	3.728882388	47.068896000
29	0.137984900	3.862056759	48.749928000
30	0.142743000	3.995231130	50.430960000
31	0.147501100	4.128405501	52.111992000
32	0.152259200	4.261579872	53.793024000

Table 1: Multiples of the telomere prime (L=177 cm).

multi-frequency PicoTesla schedules. Ultimately, these laboratory findings should serve as an experimental foundation for future research devoted to delineate (a) time frames that non-ionizing PicoTesla range magnetic fields exert a biological affect, (b) the duration of PicoTesla magnetic field induced molecular/genetic alterations, (c) identity of PTEMF range magnetic fields that selectively produce specific biological affects in bio-systems, and (d) identify additional molecular/genetic “targets” that PicoTesla magnetic fields might interact with in a manner that creates a biological affect.

Suggested Pathways for Research

1. Targeting a particular molecular species does not necessarily predict the physiologic outcome. Therefore, physiologic mechanisms of action must be exhaustively explored. Other masses analogous to those targeted may also be affected, thus altering anticipated outcomes.
2. Particular tissues maintain susceptibility to discrete magnetic profiles within a broad physiologic range. Thus, study of individual cell types is indicated.
3. Utilizing the SQUID, magnetic flux density and frequency profiles should be exhaustively measured for normal vs. pathologic tissues and organs.
4. Single dose response analysis and multiple sequencing of calculated signal parameters are indicated; as well as evaluation of effects utilizing exposure time differentials.
5. Epidemiological studies are indicated for subjects chronically exposed to ambient EMF sources. SQUID measurements are needed to compare normal vs. pathologic profiles of tissues and organs. These studies may have predictive value for predisposition to cancer, and other pathophysiological states.
6. It has appeared quite remarkable that multiples of the telomeric prime have been shown to be tissue specific. The common denominators/mechanisms for the many forms of cancer should be analyzed.
7. The approach described herein may soften dense primary tumor, such that the effect of chemotherapy may be enhanced; to therein kill more undifferentiated cancer stem cells that may not be producing telomerase. This notion is open for research.

Conclusion

While magnetic resonance therapy is most assuredly still in its infancy, there have been a diversity and multiplicity of studies indicating (1) non-ionizing, low intensity and extremely low frequency EMFs remarkably affect biological systems, both positively and perhaps negatively and (2) the need for ongoing research: theoretical, experimental and clinical. The urge to consolidate premises and to penetrate the variety of the manifest, to unify conceptual frameworks and view the underpinning workings of an undifferentiated unity lying beneath the plane of the obvious, is the highest passion of the human consciousness.

Einstein defined science as, “That which covers the greatest number of empirical facts by logical deduction from the smallest number of hypotheses or axioms” [63,64].

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