Synapse by QED Induced Radiation

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***Abstract*—Mainstream theory of neurons is based on chemical signaling by neurotransmitters (NTs) injected into the cleft by exocytosis. The NTs comprise submicron vesicles containing small molecules or neuropeptides that may be treated as biological nanoparticles (NP). But the NPs having diameters from 20-250 nm are generally larger than the 20-50 nm cleft, and therefore the NP vesicles are required to fuse with the presynaptic cell membrane prior to exocytosis. Chemical signaling is based on by the "lock and key" mechanism of olfaction whereby the postsynaptic receptors (lock) only accept the precise shape of the NT molecules (key). The chemical signal therefore begins on binding and continues until the NT molecule dissociates from the receptor. Enzymes may be required to make the dissociated NT molecules nonfunctional and endocytosis to remove them from the cleft prior to the next action potential. In contrast, QED induced signaling relies on the QM condition that the NPs lack the heat capacity to conserve absorbed thermal energy by an increase in temperature that instead is conserved by the emission of EM radiation. QED stands for quantum electrodynamics, QM for quantum mechanics, and EM for electromagnetic. QED signaling is therefore a burst of EM radiation, thereby terminating itself and avoiding problems with termination in chemical signaling: the unbinding of NT molecules from receptors, enzymes to make the remaining NT molecules in the cleft nonfunctional, and the removal of NT molecules from the cleft before the next action potential.**

Keywords – neuron, synapse, neurotransmitters, cleft, presynaptic, postsynaptic, quantum mechanics

I. INTRODUCTION

Classical biology [1] holds neurotransmitters (NTs) provide the chemical signal that sends action potentials throughout the nervous system providing rapid communication across the cleft between presynaptic and postsynaptic cells. NTs comprise vesicles containing a number of small molecules or neuropeptides. Small molecules include acetylcholine (ACh) made up of choline and acetate; whereas, neuropeptides are larger molecules that range from 3 to 36 amino acids in length. NTs are synthesized in the presynaptic cell or may be transported from the nucleus along axons. Upon activation by an action potential, the vesicles fuse with the cell membrane and empty the NT molecules into the cleft by exocytosis.

Vesicles of small molecules have diameters from 40 to 60 nm while those of neuropeptides are 90 to 250 nm. In chemical signaling, the NTs may therefore be considered biological nanoparticles (NPs). However, the neuronal cleft is only 20 to 50 nm wide, and therefore the NPs cannot empty their NT molecule cargo into the cleft without exocytosis. Delay in exocytosis is critical because chemical signaling cannot be initiated unless the NT molecules bind to the correct receptor on the postsynaptic cell.

Chemical signaling by binding of NT molecules to postsynaptic receptors is consistent with the shape theory [2,3] of olfaction where the odorant molecule in the manner of a "lock and key" fits into precisely matched receptors. However, the probability of this occurring even in olfaction is unlikely. In humans, the odorant molecule must promptly bind with a receptor over a few square centimeters of surface area in the nose. Even far less likely is chemical signaling in mating moths [4,5] where scent molecules from a female must bind to the receptor of a male at distances of hundreds of meters.

Certainly, the submicron cleft improves the probability of neuron synapse by chemical signaling over that by odorants in the nose and scents in mating moths. Nevertheless, it can be safely [6] concluded it is still unlikely NT molecules bind to postsynaptic receptors. Given that neurons do signal quite efficiently suggests a mechanism other than the "lock and key" is at play.

Signaling by chemical binding of NT molecules with receptors is proposed superseded by EM signaling from a burst of QED induced emission corresponding to the unique EM molecular spectra of the NT molecules. The EM signal emitted at the instant of exocytosis travels across the synaptic cleft allowing unique recognition by postsynaptic receptors. Chemical binding of NT molecules to postsynaptic receptors is not required.

What this means is that both exocytosis and endocytosis occur in a prompt Exo/Endo Cycle. Indeed, such a mechanism has been proposed [7] in pancreatic β cells linked to diabetes and metaphorically described as a "walk, kiss, pause … then run" process where vesicle fusion at the presynaptic cell membrane is a partly reversible process. But this is not a new idea. Over 30 years ago, experiments [8] showed after fusion and NT release the synaptic vesicles are reformed rapidly, i.e., the possibility that an individual vesicle may remain essentially intact during exocytosis without a full merger of the vesicle and presynaptic membranes. The Exo/Endo cycle in combination with QED induced EM signaling is proposed here as an alternative to mainstream theory based on chemical signaling.

In the Exo/Endo Cycle, the NPs during endocytosis acquire the thermal kT energy of the presynaptic cell. Here k is Boltzmann's constant and T is absolute temperature. But isolation at the instant of exocytosis leaves the NPs with thermal kT energy not allowed by QM. Since QM also requires the heat capacity of the NPs to vanish, the kT energy cannot be conserved by an increase in temperature. Instead, conservation proceeds by the NPs emitting a burst of QED radiation acquired in the presynaptic cell. The QED photons have Planck energies beyond the UV that excite the NT molecules to emit a burst of QED radiation given by their EM spectra, thereby providing a unique signal for recognition by the postsynaptic receptors.

Since the EM signal given by the burst of QED radiation terminates itself, long standing problems with terminating chemical signaling are avoided, i.e., how to unbind NT molecules from postsynaptic receptors, the need for enzymes to chemically render the NT molecules remaining in the cleft nonfunctional, and the removal of NT molecules from the cleft before the next action potential.

Conversely, the NT molecules essentially remain in the presynaptic cell. Even if some NT molecules enter the cleft, they are promptly returned to the presynaptic cell by endocytosis. The Exo/Endo Cycle recycles NT molecules, and therefore the burst of QED induced radiation may be repeated for successive action potentials with the same NT molecules without burdening the supply of NPs from the axon that is limited to NP speeds < 400 mm / day.

QED induced radiation applies not only to biological processes, but also to diverse areas [9] of physics. In astronomy, QED radiation allows the light from distant galaxies to be redshift in cosmic dust instead of by Hubble's interpretation that the galaxy is moving away from us, thereby negating an expanding Universe. Charge In flow electrification is induced by nanoparticle impurities in the liquid. Human olfaction is enhanced by the emission of microwave spectra of the odorant molecule upon colliding with epithelial surface in the nose. Cancer is enhanced from DNA damage by NPs, etc.

II. PURPOSE

To show nerve cells signal across the synaptic cleft by the QED induced burst of EM radiation corresponding to the EM spectrum of the NT molecules.

III. THEORY

The Expo/Endo Cycle with QED induced signaling across the cleft between the presynaptic and postsynaptic cells is depicted in Fig. 1. Vesicles containing NT molecules approach and fuse with the presynaptic cell membrane by exocytosis. Isolated NT molecules entering the cleft emit QED radiation to signal the postsynaptic receptors. NT molecules remaining in the cleft promptly return to the presynaptic cell by endocytosis and are recycled into NPs in preparation for the next action potential.

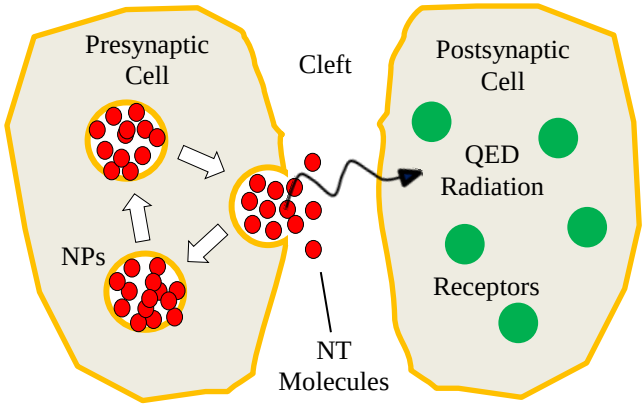

Figure 1 Exo/Endo Cycle - QED Induced Synapse

QM and Classical Oscillators. QM differs from classical physics by the heat capacity of the atom. The average Planck energy <E> of the QM oscillator is,

$$< E > = \frac{hc/\lambda}{\exp\left(\frac{hc}{\lambda kT}\right) - 1} \qquad (1)$$

where, h and k are Planck's and Boltzmann's constants, c is the speed of light, T is absolute temperature, and λ is wavelength. At 300 K, the QM dispersion of average Planck energy with wavelength is shown in Fig. 2

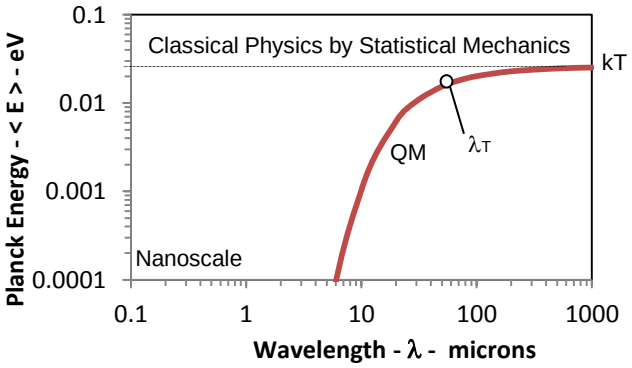

Figure 2 Classical and QM Oscillators at 300 K

In Fig. 2, the thermal wavelength λ_T = hc/kT separates classical physics from QM. Classical physics by statistical mechanics allows the atom to have heat capacity (constant kT energy) from the macroscale ($\lambda > \lambda_T$) to the nanoscale (λ < 1 micron) thereby allowing atoms under EM confinement at the nanoscale to have heat capacity. In contrast, QM allows the atom to have kT energy only at the macroscale ($\lambda > \lambda_T$) and forbids atoms at the nanoscale (λ < 1 micron) to have heat capacity.

TIR Confinement Biological NPs lack specific heat and cannot conserve absorbed EM energy by an increase in temperature. Instead, conservation may only proceed by the QED induced frequency up-conversion of the absorbed EM energy to the TIR confinement frequency of the NP. TIR stands for total internal reflection. Since NPs have high surface to volume ratios, absorbed EM energy of any form is confined by TIR almost entirely in the NP surface. The TIR confinement is momentary and occurs only upon absorption of EM energy, and therefore, the TIR confinement effectively sustains itself.

Unlike metal and metal oxide NPs, biological NPs fragment into individual NT molecules upon exocytosis. At least initially, the TIR confinement may be considered that of the NPs. Subsequently, the QED radiation induced in the NPs excites the NT molecules to emit their EM spectra. Otherwise, QED induces individual NT molecules to emit their EM spectra. Either way, the signal given by the NT molecular spectra is emitted for recognition by the postsynaptic receptors.

NT molecules emit absorbed thermal kT energy by their EM spectra. But NPs as a continuum emit QED radiation depending on their diameter D and refractive index n. The QED photon energy E and frequency f are:

$$E = hf \qquad f = \frac{c}{\lambda} \qquad \lambda = 2nD \tag{2}$$

NP Thermal Energy The NPs acquire the thermal kT energy at body temperature from the presynaptic cell. The total energy U is dependent on the diameter D and number N_A of atoms having a cubical spacing Δ is,

$$U = \frac{3}{2} kT\, N_A = \frac{3}{12} \pi kT \left(\frac{D}{\Delta}\right)^3 \tag{3}$$

The number N of QED photons / burst,

$$N = \frac{U}{E} \tag{4}$$

For NT molecules having cubic spacing of Δ = 0.25 nm and refractive index n = 1.36, the Planck energy E and number N of QED photons are shown in Fig. 3.

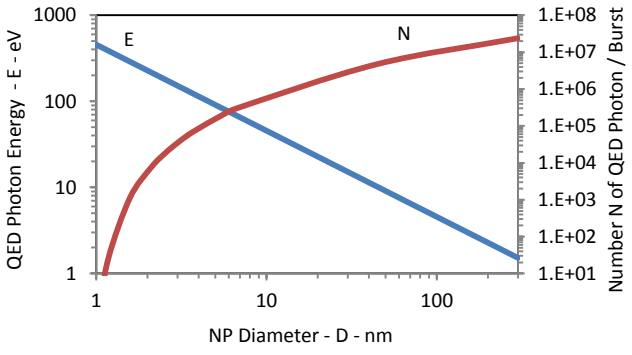

Figure 3 Planck Energy and Number of Photons in Burst

The burst of QED radiation described in Fig. 3 shows vesicles with small molecules characterized as 40-60 nm NPs produce an average 1.8×10^4 – 9 eV QED photons, for a total burst energy of 0.16 MeV; whereas, the vesicles of neuropeptides having 90-250 nm NPs produce a burst of 0.60 MeV for an average 2×10^5 QED photons at 3 eV.

IV. Discussion

A. *Biophotons* Biological systems may signal other systems and interact with the environment by biophotons [10] depending on the type and complexity of the biological system and the nature of the information being communicated. Chemical signalling by NT molecules is mainstream theory, but may be compared to biophotons and QED radiation because of the obvious argument that the latter avoid many problems associated with binding and unbinding of NT molecules to postsynaptic receptors.

Biophotons as a source of EM radiation from biological systems are described in the literature as Ultra-weak Photon Emission (UPE). In chemiluminescence assays, reactive oxidative species (ROS) are inferred [11] from the absorption of externally supplied UV light. However, the mechanism by which UPE having Planck energies beyond the UV necessary to create ROS by itself independent of external sources is not identified.

In contrast, QED radiation [12] beyond the UV is capable of producing ROS from the photolysis of water molecules. The UV emission is caused by natural or manmade metal and metal oxide NPs in the biological sample that absorb thermal energy from colliding water molecules. Strictly, QED radiation cannot be considered biophotons because the NPs are inanimate.

Conversely, the UV produced in the natural fragmentation [12] of epithelial tissue is a source of biophotons. Regardless of whether the NPs are inanimate or biological, the DNA damage by ROS is of concern because if not repaired correctly by the DNA may lead to cancer. Indeed, ROS produced from metal and metal oxide NPs is consistent with decades of experiments that show NPs unequivocally produce the ROS and cause DNA damage.

B. *Source of EM Signaling* Over 50 years ago, biological systems were postulated [13] to signal each other by UPE from coherent vibrations of electrically polar structures. Most protein molecules are polar structures, and therefore if vibration excited emit radiation. Metabolic energy of guanosine triphosphate (GTP) and adenosine triphosphate (ATP) was postulated to be the source of protein vibrations in EM signaling.

Specifically, microtubules (MTs) have been proposed as the source of biological energy. MTs fulfill requirements [13] for generation of biophotons in that MTs are composed of energy rich GTP tubulin while displaying dynamic instability between growth by tubulin polymerization and

shrinking MT depolymerization. Claims [14] that the free energy for GTP hydrolysis is stored in the MT lattice assume polymerization occurs without the lowering of the activation energy by the thermal energy absorbed by the MTs from water molecule collisions. Certainly, high temperatures or pressure cannot be the source of polymerization. Only QED induced radiation beyond the UV may polymerize GTP tubulin.

In mitochondrion, ATP production [15] in the citric acid cycle has 40% efficiency with the remainder of the energy usually dissipated as heat. Hence, the ATP energy from mitochondria represents the most significant source of energy for excitation of MT vibrations. However, the ATP energy must somehow be converted to vibrating force.

Consistent with EM radiation [13] from mechanical vibrations of polar MTs, various energy sources [15] are reviewed, a summary of which is as follows.

(1) Energy released from hydrolysis of GTP in dynamic instability of MTs is estimated to be 7.1 kJ/mol giving a corresponding power input to the MT lattice of about 1.8 $x10^{-14}$ W/mm of MT length or about $3x10^{-18}$ W for the total MT network.
(2) Energy transferred from moving of motor proteins and their interaction with MTs amounts to the hydrolysis of a single ATP molecule for each step of motor protein is estimated to be < 10^{-15} W.
(3) Energy released from mitochondria as "wasted" energy in the course of citric acid cycle may be the most significant energy source for excitations of MT vibrations is estimated to be about 10^{-14} W.

Except for moving motor proteins where momentum is directly transferred to the MTs in the axial direction, the energy from GTP and ATP hydrolysis do not participate in axial MT vibration. Even so, the axial MT vibration from motor proteins is limited to the GHz range and is not capable of polymerizing GTP or ATP. Moreover, UV and VIS photons are not created by vibrating polar MTs or protein molecules in the GHz range.

In contrast, QED radiation beyond the UV produced from the thermal energy absorbed in MTs by water molecule collisions was proposed [16] as the source of polymerization during MT assembly into cell spindles during hydrolysis. The thermal energy absorbed by MTs is significant, i.e., the 25 nm MTs absorb about 1.7 nW/nm that is many orders of magnitude greater than the energy of GTP hydrolysis [15] of $1.4x10^{-14}$ W/mm or $1.4x10^{-20}$ W/nm.

QED radiation emitted from the MTs at 18 eV is in the PHz range and far beyond EM radiation at GHz microwave frequencies associated with vibrating polar MTs. QED radiation at UV or higher frequencies grows MTs by photochemical induced polymerization. Hence, GTP or ATP hydrolysis by an unphysical mechanism to induce axial MT vibration is not required. MT shrinking occurs as the QED radiation activates cleaving proteins in the surroundings that bind to the MTs.

D. *Hydrolysis Review* Significant differences between GTP and ATP hydrolysis [15] and QED induced radiation [16] needs to be resolved. Perhaps, Density Functional Theory (DFT) commonly used in estimating reductions of ATP activation energy in hydrolysis may need review.

The collision power Q_C of water molecules of mass *m* transferred to MTs having diameter D and length L is,

$$Q_C = \frac{\pi}{\sqrt{3}} pPDL\sqrt{\frac{kT}{m}} \quad (5)$$

where, *p* is the unit probability of full kT energy transfer for inelastic collisions and P is ambient pressure. The mass m = MW/N_{Avag} where MW = 18 and N_{Avag} is Avagadro's number. For D = 25 nm, the absorbed thermal power Q_C / L = 1.7 nW/nm as shown in Fig. 4.

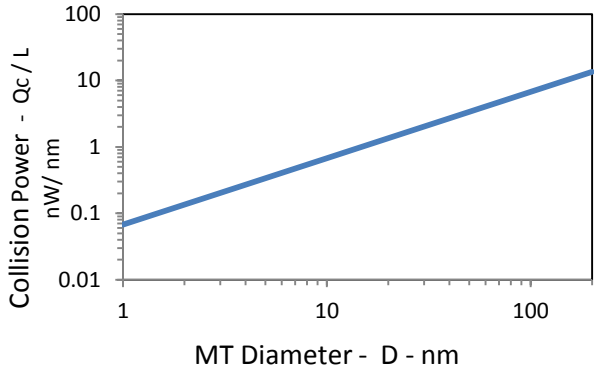

Figure 4 Collision Power and MT Diameter

Of interest is the effect of QED radiation in reducing the ATP activation energy in hydrolysis of 200-400 kJ/mol or 2-4 eV. QED induces [16] the 25 nm MTs to create 18 eV photons in conserving absorbed thermal energy from water molecule collisions, thereby exceeding the 2-4 eV necessary for ATP activation without enzymes. QED is therefore consistent with DFT in that hydrolysis reduces the ATP activation energy, i.e., for ATP to ADP + Pi, DFT gives 31kJ/mol. Perhaps, DFT should include QED radiation in calculations of ATP hydrolysis instead of simply attributing reductions in ATP activation energy to enzymes alone.

C. *Nano-Voltmeter* Classical biology [1] considers the E-field in the cell to be dominated by the cell membrane. Typically, a cell membrane potential of -150 mV is used which for a 5 nm membrane thickness gives an EM field of about $3x10^7$ V/m. The EM field is estimated to extend only 1-10 nm beyond the membrane.

Recently, a nano-voltmeter was developed [17] to measure EM fields within the cell. The nano-voltmeter is a 30 nm diameter NP encapsulating a voltage-sensitive dye [18] having a fluorescence spectrum that shifts in response to changing EM fields.

For the mitochondrion, the nano-voltmeter [16] showed the EM fields extending out much farther (microns) into the cytosol. Since EM field penetration is far more than predicted by electrostatics, the cell membrane is not the sole source of EM field in the cell. What this means is cell membranes of other organelles, or regions of the cell interior, or the MTs in the cytoskeleton itself are electrically active, but could not be measured with previous techniques, because of their submicron size.

Specifically, the cell spindle comprised of many MTs may be viewed as a source of UV and higher photons that may have confused the nano-voltmeter measurement of EM fields that did not include the high-energy QED radiation produced by the MTs in the calibration. The sensitivity of the nano-voltmeter to UV or higher radiation is required to support this conjecture.

V. SUMMARY AND CONC LUSIONS

1. QED induced radiation in EM signaling across the cleft between presynaptic and postsynaptic cells is shown to offer a reasonable alternative to chemical signaling in that problems with binding NT molecules to receptors, unbinding of NT molecules from receptors and their removal form the cleft, and making the NT molecule non-functional after synapse are avoided.

2. Signaling by EM radiation from vibration of proteins powered by GTP and ATP energy from mitochondria may be superseded by QED radiation from NPs and NT molecules in a prompt Endo/Exo Cycle. QED emission occurs at the instant the NT molecules begin enter the cleft. The NT molecules over the Exo/Endo Cycle essentially remain in the presynaptic cell during QED emission. Any NT molecules remaining in the cleft are promptly returned to the presynaptic cell by endocytosis.

3. DFT calculations in estimates of activation energies in GTP and ATP hydrolysis should consider the thermal energy absorbed from collisions of water molecules.

4. Nano-voltmeter measurements that suggest high EM fields exist far into the cytosol are likely to be an artifact of QED radiation beyond the UV affecting the calibration based on VIS photons. Data on nano-voltmeter sensitivity to UV or higher QED photons is required to confirm this conjecture.

5. This paper can at best only hope to be a preliminary attempt to present QED radiation as an energy source in living systems thereby supplementing mainstream theory of synapse by chemical signaling. Comments are solicited.

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