NEURON SYNAPSE BY QUANTUM MECHANICS

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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.

KEY WORDS
Neuron, signaling, synapse, neurotransmitters, cleft, presynaptic, postsynaptic, quantum mechanics, quantum electrodynamics

1. 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 until NT molecules bind to the receptors.

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 [1] 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.

2. 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.

3. 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.

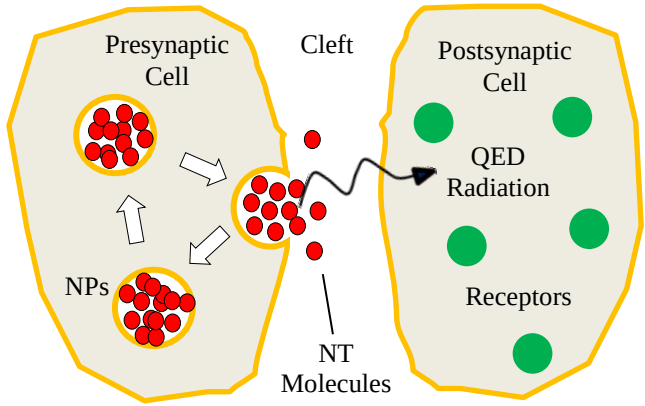

Fig.1 Exo/Endo Cycle - QED Induced Synapse

Biological NPs by QM 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. 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:

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

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,

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

The number N of QED photons / burst,

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

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. 2.

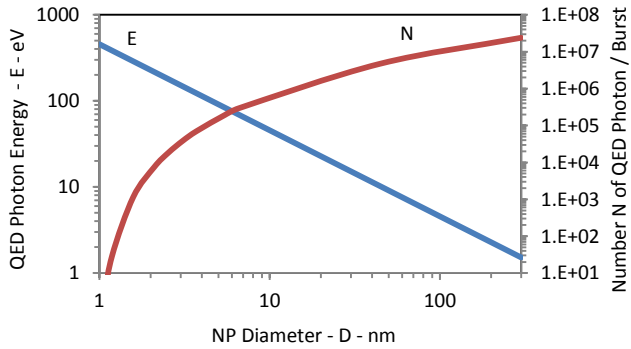

Fig. 2 Planck Energy and Number of QED Photons in Burst

The burst of QED radiation described in Fig. 2 shows vesicles with small molecules characterized as 40-60 nm NPs produce an EM signal comprising 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 signal of a burst of 0.60 MeV for an average 2×10^5 QED photons at 3 eV.

EM signaling is similar to the QED radiation induced in the natural fragmentation [10] of epithelial tissue. Regardless of whether the NPs are inanimate or biological, the DNA damage is of concern because if not repaired correctly by the DNA may lead to cancer. Indeed, reactive oxidative species (ROS) produced from metal and metal oxide NPs by QED radiation is consistent with decades of experiments that show NPs unequivocally produce the ROS that cause DNA damage.

4. SUMMARY AND CONCLUSIONS

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. EM signaling by QED emission occurs at the instant the NT molecules 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. Chemical signaling by NT molecules is mainstream theory, but may be superseded by QED radiation because of the obvious argument that the latter avoids many problems associated with binding and unbinding of NT molecules to postsynaptic receptors.

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