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The Internet of Brain Things: Theoretical Basis for the Usage of Neuralink Chip

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***Abstract*—If the chip called Neuralink is realized and marketable then a wide spectrum of biomedical opportunities shall be available. One of them is the potential usage of chip as an implanted antenna. In fact, once the chip begins to be operative for biomedical purposes, then it might be a key piece in a prospective Internet of Brain Things (IBT). This Internet would have a direct impact in all those patients that have lost domain in the nervous system. Thus, the scenario by which these patients have been injected of artificial neurotransmitters (ANT), the trajectory of them can be modified through the electromagnetic fields emitted by Neuralink. Thus, it is expected to have effect in the recovery of motility, speak and memory of affected patients. This paper explores the physics grounds of IBT and how communication between neurons might be improved under the synergy of ANT and IBT.**

I. Introduction

Neural synapse constitutes a basic function of nervous system that contemplates the emission and reception of neurotransmitters between two neurons [1]. This neural communication is mainly governed by electrical pulses that allow to transmitter-gated ion channels on the post-synaptic membrane, the entry of information towards the completion of the signal processing along the neural network [2]. In other words, synapse can also be defined in a nutshell as the electric continuity of molecular communication between the presynaptic neuron and the membrane of postsynaptic receptor. Thus, it is expected that synapse might be behaving as a kind of micro electrical currents depending on the electrical basic unit such as neurotransmitters and whose existence can be explained in terms of input and output signals [3]. In this manner, if the neural function is critically damaged then the nervous system would reduce its efficiency drastically. This might be a potential cause of the apparition of well-known neurodegenrative diseases such as Alzheimer and Parkinson diseases among others (nowadays affecting to around 6 millions of people in USA [4]). On the other hand, brain and functions can also be seriously affected due to sequels of the so-called ischemic stroke by fails of heart, abnormal values of blood pressure, and diabetes among other diseases [6]. In fact, ischemic stroke in adults patients would cause the lost of axon functionalities so that synapse is damaged. Most of them might be eligible to continue either pharmacological or therapy based on permanent rehab. Nevertheless, with the apparition of artificial neurotransmitters (ANT) such as the ones made of sol-gel silicate films [7], one expects that ANT

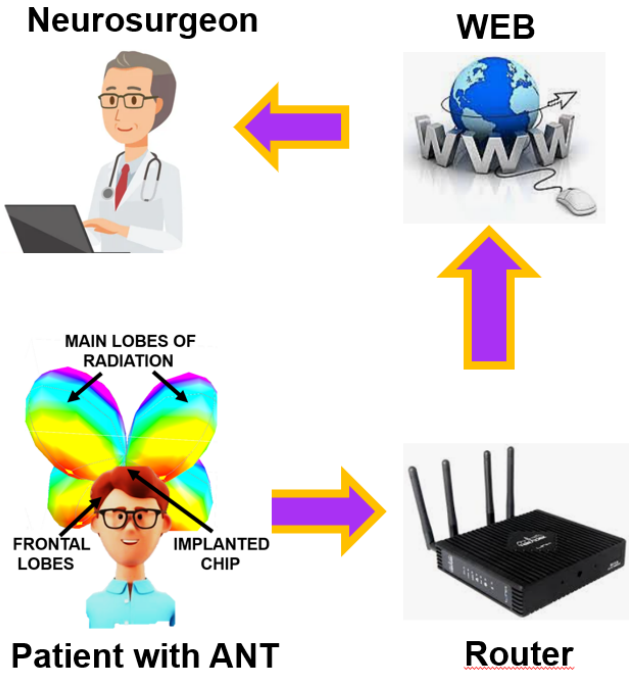

Fig. 1. The Internet of Brain Things in a nutshell: (i) An implated Neuralink chip [5] emits innoxious electromagnetic fields to lobes of brain affecting the spatial trajectory of neurotransmitters, (ii) the chip sends packets of data to wireless router, (iii) this data is uploaded to network, (iv) Neurosurgeon gets update information about emitted fields and status of patient motility. Doctor can modify to distance directivity and others electromagnetic properties of chip.

would improve the lost performance between adjacent neurons towards the completion of synapse. Indeed, with a net electric charge on their surface then the Coulomb forces as well as electromagnetic waves would affect them substantially [8]. It is noteworthy that one can take advantage of this in a prospective scenario of implantable powerful chips at the brain area such as for example the so-called Neuralink. In the scenario that chip (as reconfigurable) works also as near-field antenna [9], then electromagnetic fields affecting diverse lobes of brain (as seen down left-side Fig.1) and would change notably the trajectory of implanted ANT, so that one expect the apparition of a electrodynamics towards the completion of synapse.

This fact would allow affected patients at the middle or long term improve aspects of motility (for example) avoiding

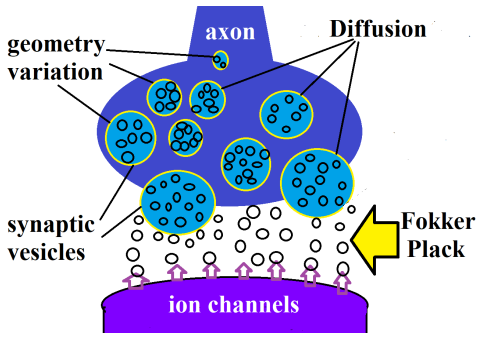

Fig. 2. Sketch of neural synapse as two-phase process: Initially would exist diffusion of vesicles that ends at the releasing of neurotransmitters. Finally, diffusion and drag together due to Fokker-Planck dynamics would be a possible cause to push neurotransmitters out axon.

extra time of therapy and excess of pharmacologies. Based at this, the present paper proposes a prospective Internet of Brain Things (IBT) whose central action is the alternative usage of chip Neuralink as antenna to create electrodynamics on the injected ANT to produce displacement of them to the ion channels. Clearly, this would depend on the accuracy of antenna and the net number of available ANT around the axon zone.

A sketch of IBT is displayed in Fig.1. Thus, one would expect that a important fraction of ANT improves the completion of synapse. In order to lay the theoretical foundations of IBT, this paper tries to provide another view to the process of neural synapse through a hybrid view that combines the Fokker-Planck equation and the classical electrodynamics [10]. To accomplish this, it is assumed the basic idea that vesicles as ell as neurotransmitters are under Coulomb interactions in the sense that them experience the well-known Coulomb forces. Consider for example Fig.2 that vesicles manifest electric interaction with the Ca^{2+} waves. While them have a volumetric charge density due to internal neurotransmitters, then the volumetric charge density of a vesicle containing neurotransmitters can be written as $\rho = \frac{dQ}{dV} \Rightarrow \int dQ = Q = \int \rho dV$ and the electrical current:

$$\frac{dQ}{dt} = \int \frac{d\rho}{dt} dV + \int \rho \frac{dV}{dt} = \int \frac{d\rho}{dt} dV + \int \rho \frac{d(A\ell)}{dt}, \quad (1)$$

thus whereas ℓ the traveled distance from a constant area A. In this manner the electric current that is assumed that emerges [15] inside the vesicles previous to the releasing of neurotransmitters can be written under the validity of a classical electrodynamics in the axon zone, as:

$$\frac{dQ}{dt} = \int \frac{d\rho}{dt} dV + \int \rho A \frac{d\ell}{dt} = \int \frac{d\rho}{dt} dV + \rho A v, \quad (2)$$

by which by knowing the neurotransmitters density the the created electric current can be calculated.

Indeed, one can see that the velocity v can be expressed as function of Coulomb force: $v = |(2\kappa/m) \int qQ/|R(s)|^2 ds|^{1/2}$ that is responsible in the generation of electric currents. Therefore a naive modeling of electric currents can be written as:

$$\frac{dQ}{dt} = \int \frac{d\rho}{dt} dV + \frac{2\kappa\rho A}{m} \left| \int \frac{qQ}{|R(s)|^2} ds \right|^{1/2}. \quad (3)$$

Eq.3 summarizes to some extent the scope of paper as to the theoretical basis of proposal of an IBT. Clearly, it is indicating that Coulomb forces might to generate complexities at the modeling of injected ANT. Because of this in second section, the process of synapse is described by the Fokker-Planck equation. Here, the diffusion and drag terms are examined. In third section, with the results of previous section, the equations corresponding to the electrical current, the spontaneous apparition of RC circuit at the synapse process is proposed and discussed. In fourth section, under this context is discussed the concrete role of Neuralink chip. Finally, the conclusion of paper is presented.

II. Diffusion and Drag of Neurotransmitters

As sketched in Fig.2, the entire process of synapse might be modeled in up to steps: A first one that is governed by diffusion processes involving the dynamics of synaptic vesicles and that ends just at the boundary of emitter neuron when neurotransmitters are released [11][12][13]. Intuitively one can directly apply the well-known diffusion equation [14]. Secondly, the neurotransmitters are released from vesicles and due electric repulsion are expelled forcefully out to the synaptic cleft. In this zone, it is demanded to employ the Fokker-Planck equation so that either drag and diffusion are constituting the entire dynamics of neurotransmitters previous their arrival to ion channels. Because all this, Fokker-Planck equation for one dimension in an approximate manner to treat the synapse problem can be written down as:

$$\frac{d\rho}{dt} = D\frac{d^2\rho}{dx^2} - G\frac{d\rho}{dx}, \quad (4)$$

with D and G constants. Thus, it is argued that Eq.4 encloses a kind of electric circuit. Therefore, the starting point in this debate is the volumetric integration in both sides:

$$\int dV \frac{d\rho}{dt} = \int dV D\frac{d^2\rho}{dx^2} - \int dV G\frac{d\rho}{dx}. \quad (5)$$

In order to get a compact equation, the integrations term to term are given in an explicit manner below:

A. The Term: $\int dV \frac{d\rho}{dt}$

The variable ρ is referred to a kind of volumetric density of neurotransmitters having each one an electric charge due to ions basically [15][16][17]. In this manner the solution of this term is done in a straightforward manner yielding in the electric current created by the neurotransmitters:

$$\int dV \frac{d\rho}{dt} = \frac{d}{dt} \int \rho dV = \frac{dq}{dt}. \quad (6)$$

B. The Term: $\int dV D\frac{d^2\rho}{dx^2}$

It is actually a diffusive term, so that a special attention should be done basically because it contains a second derivative in space. Actually one can take advantage of this in the sense that it can be seen as a double operation of gradient operator (clearly in one dimension): $d^2/dx^2 = \nabla \cdot \nabla = \nabla^2$. To exploit this operators, ρ the volumetric charge density can be written as its simplest definition $q/V = q/A \times \ell$. So far, these operators are acting on the space inside the axon. Also, one can associate a cylindric geometry that encloses the neurotransmitters, so that from above $A = \pi r^2$. With this one gets below:

$$\int dV D\frac{d^2\rho}{dx^2} = D\nabla\nabla\int \rho dV = D\nabla\nabla\int \frac{q}{\ell\pi r^2}dV \quad (7)$$

$$= \frac{4\epsilon_V}{r^2}D\nabla\nabla\int \frac{q}{4\pi\epsilon_V\ell}dV = \frac{4\epsilon_V D}{r^2}\nabla\nabla\int \frac{q}{4\pi\epsilon_V\ell}dV. \quad (8)$$

Thus one can see that $\Phi(\ell) = \frac{q}{4\pi\epsilon_V\ell}$ the electric potential. When $\ell = x$, then the first operator ∇ enter to integration: $\int \nabla\Phi(x)dV = -\int \mathbf{E}dV$. Subsequently the second operator: $-\int \nabla\mathbf{E}dV = -q/\epsilon_A$ in according to divergence theorem one finds for this diffusive term:

$$\int dV D\frac{d^2\rho}{dx^2} = -\frac{4q\epsilon_V D}{\epsilon_A r^2}. \quad (9)$$

According to the electrodynamics laws, both ϵ_V and ϵ_A corresponding to the electric permittivities of vesicles and axon, respectively.

C. The Drag Term: $\int dVG\frac{d\rho}{dx}$

For this term, it is assumed that the neurotransmitters are in transit through the cleft synaptic to next neuron arriving to ion channels. Nevertheless, again one can take advantage of the presence of gradient operator. In this manner one gets below (by following an analogue procedure done above for the diffusive term):

$$\int dVG\frac{d\rho}{dx} = G\frac{d}{dx}\int \rho dV = \frac{G4\epsilon_I}{R^2}\frac{d}{dx}\int \frac{q}{4\pi\epsilon_I x}dV \quad (10)$$

$$= \frac{G4\epsilon_I}{R^2}\int \frac{d}{dx}\Phi(x)dV = -\frac{G4\epsilon_I}{R^2}\int EdV, \quad (11)$$

with ϵ_I the permittivity at the region of cleft. Because the lack of a vector product, instead one has an integration of scalar. In virtue to this one can take advantage of volume differential dV that can be written as zdA with z a kind of height related to a cylinder but under the assumption that this geometry is applied inside the cleft area. Thus one can apply directly the Gauss's law yielding:

$$-\frac{G4\epsilon_I}{R^2}\int EdV = -\frac{G4\epsilon_I z}{R^2}\int EdA = -\frac{G4\epsilon_I zq}{\epsilon_N R^2}. \quad (12)$$

In this way the resulting Eq.6, Eq.9 and Eq.12 are inserted in Eq.4 so that one gets:

$$\frac{dq}{dt} = \frac{4q\epsilon_V D}{\epsilon_A r^2} - \frac{qG4\epsilon_I z}{\epsilon_N R^2} = -q\left(\frac{4\epsilon_V D}{\epsilon_A r^2} - \frac{G4\epsilon_I z}{\epsilon_N R^2}\right), \quad (13)$$

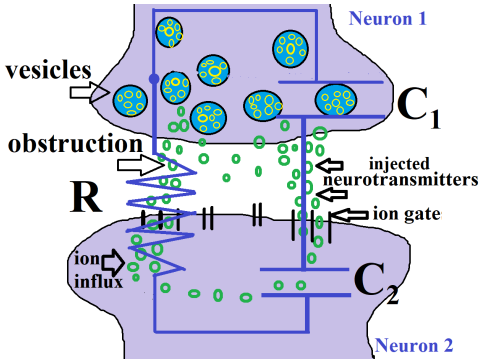

Fig. 3. A possible scenario of a RC circuit created at the process of neural synapse. To note the resistances as a consequence of bunching of neurotransmitters and the creation of obstructions at the synaptic cleft zone [18][19][20].

with ϵ_N the permittivity at the zone of next neuron. The solution of Eq.13 is done in a straightforward manner yielding:

$$q(t) = q_0 \mathrm{Exp}\left[-\left(\frac{4\epsilon_V D}{\epsilon_A r^2} - \frac{G4\epsilon_I z}{\epsilon_N R^2}\right)(t - t_0)\right]. \quad (14)$$

In this manner, the reader can see that the term in brackets has a direct value inside electrodynamics, so that one has below:

$$\frac{1}{R_{\mathrm{EQ}}C_{\mathrm{EQ}}} = \frac{4\epsilon_V D}{\epsilon_A r^2} - \frac{G4\epsilon_I z}{\epsilon_N R^2} \quad (15)$$

that is the time constant of a RC circuit, with R_{EQ} and C_{EQ} the respective equivalences of resistors and capacitors.

III. RC Circuits in Neural Synapse

From above Eq.15 it can again be written as:

$$\frac{4\epsilon_V D}{\epsilon_A r^2} - \frac{G4\epsilon_I z}{\epsilon_N R^2} = \frac{4\epsilon_V D\epsilon_N R^2 - 4G\epsilon_I z\epsilon_A r^2}{\epsilon_N R^2\epsilon_A r^2}. \quad (16)$$

It is noteworthy that one can assume $G < 0$ in order to get that $G \times z \Rightarrow \gamma$ then one can improve the physics meaning of Eq.16 to employ the well-known rules of series capacitors. Thus one has below:

$$\frac{1}{R_{\mathrm{EQ}}C_{\mathrm{EQ}}} = \frac{4\epsilon_V D\epsilon_N R^2 + 4\gamma\epsilon_I\epsilon_A r^2}{\epsilon_N R^2\epsilon_A r^2}. \quad (17)$$

A particular case is that of $\epsilon_I = \epsilon_V \approx \epsilon$ expressing the fact by which the permittivity is conserved from at the vesicles until to reach the synaptic cleft. In addition: $D \approx \gamma$ then one gets below:

$$\frac{1}{R_{\mathrm{EQ}}C_{\mathrm{EQ}}} = 4\gamma\epsilon \times \frac{\epsilon_A r^2 + \epsilon_N R^2}{\epsilon_N R^2\epsilon_A r^2}. \quad (18)$$

Now one can divide the fraction over a random distance δ so that one recognize a pair of capacitances in series:

$$\frac{1}{R_{\mathrm{EQ}}C_{\mathrm{EQ}}} = 4\gamma\epsilon \times \frac{\epsilon_A\frac{r^2}{\delta} + \epsilon_N\frac{R^2}{\delta}}{\epsilon_N\frac{R^2}{\delta}\epsilon_A\frac{r^2}{\delta}} \quad (19)$$

$$= \frac{1}{R_{\mathrm{EQ}}C_{\mathrm{EQ}}} = 4\gamma\epsilon \times \left[\frac{1}{\epsilon_N\frac{\pi R^2}{\delta}} + \frac{1}{\epsilon_A\frac{\pi r^2}{\delta}}\right]. \quad (20)$$

One can finally see that:

$$R_{\mathrm{EQ}} = \frac{1}{4\gamma\epsilon} \tag{21}$$

$$C_{\mathrm{EQ}} = \frac{1}{\left[\frac{1}{\epsilon_N \frac{\pi R^2}{\delta}} + \frac{1}{\epsilon_A \frac{\pi r^2}{\delta}}\right]} = \frac{1}{\left[\frac{1}{C_2} + \frac{1}{C_1}\right]}. \tag{22}$$

Therefore, Fig.4 resumes the electrical equations written above. In fact, while the Ca^{2+} waves travel along the dendrite so that it is expected that them exert repulsion forces on the vesicles [21][22][23]. These repulsive forces would generate the diffusion of vesicles that in turn might be a physical reason by which one or various vesicles after of adhesion have the role to store or release neurotransmitter for a time. The capacitor is therefore $(\epsilon_A \pi r^2/\delta)$ with ϵ_A the permittivity in the axon belonging to neuron 1 (in accordance to Fig.4) where is formed that capacitor. This formula responds to a one of circular plates. In effect, nature can minimize energy so that a circular geometry as one that constitutes the optimal case for the releasing neurotransmitters. It should be noted that $r >$ the radius of vesicle as well as $\delta >$ radius of neurotransmitter. In this way, this first capacitor emerges as consequence of diffusive processes of neurotransmitters. Subsequently, the neurotransmitters have reached the synaptic cleft due to the voltage regulated calcium channels that allow them (neurotransmitters) to active the ion receptors in the postsynaptic neuron to produce an imminent influx of neurotransmitters going through the neuron 2.

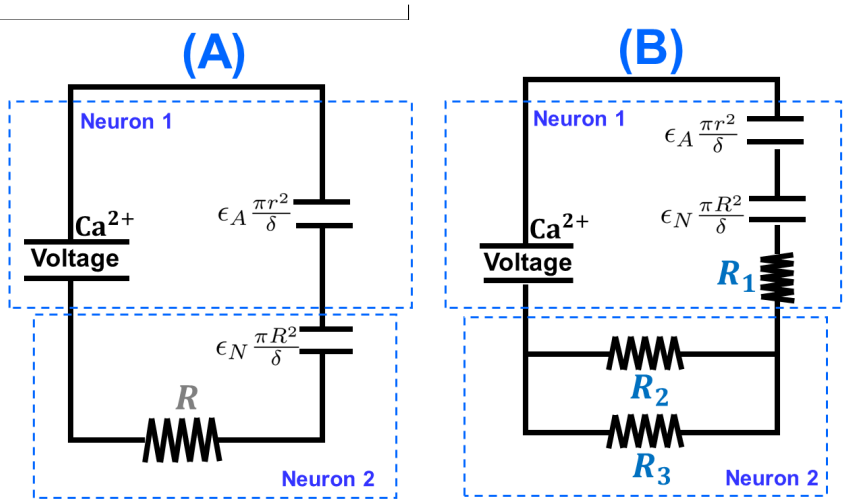

Fig. 4. Evolution of RC circuit at the neural synapse. In left-side: up to 2 capacitors in each neuron involved at the synapse. Right-side: Apparition of various resistances due to the generation of new paths for neurotransmittters passing to next neuron and producing bunching and obstructions.

IV. Canceling of Electrical Currents and Role of Chip Neuralink

Although synapse can be seen as a process of deterministic nature, one would expect that the exit of neurotransmitters is done in a random manner. Also, abundance and subsequent avalanche would create a bunching of neurotransmitters that would constitute a kind of electrical resistance along the synaptic cleft with a permittivity ϵ as seen in Eq.21. Thus, it is claimed that in some space of synaptic cleft is produced a kind of obstruction due to a probabilistic accumulation of neurotransmitters. It is noteworthy that these obstructions might appear also in regions inside the receptor neuron (or neuron 2 as established in Fig.4). Therefore one can speculate that bunching of neurotransmitters at the cleft as well as cancellation of current, might be a potential cause for the apparition of neurodegenerative diseases such as Parkinson, Ataxia, Alzheimer, etc, that are generated by the lack of neural connectivity. Clearly one can see the critical role of Ca^{2+} ions waves to push out the vesicles of neurotransmitters initially. From this one can adjudicate to these ions as a kind of voltage source by following the analogue of a RC as derived above. The human intervention in the regulation of Ca^{2+} and the dynamics of neurotransmitters would establish a key factor to restore the lost functionalities of synaptic processes in patients with neuralgic-like diseases.

A. Potential Task of Neuralink

One can wonder about the mechanism that would avoid neurotransmitters accumulation. In a first instance it might be needed to inject to patient artificial neurotransmitters (ANT) so that them can be moved through electric fields from an external antenna. In this manner, the chip Neuralink might do this job in order to guarantee the homogeneous exit of neurotransmitters from a neuron to another. In Fig.5 a prospective scenario of action by the mode-antenna of chip Neuralink. While injected neurotransmitters are charged electrically compounds on their

surface, then them are sensitive to moderate dose of radiation provided by the electromagnetic fields of antenna. This, guided by the field lines, anti-bunching effects would allow a fluid traffic of them between two neurons independent of the presence of Ca^{2+}. Whereas in (A) is to some extent displaying an initial electric configuration with a single resistance at the next neuron due to accumulation and bunching of neurotransmitters, one can note that at neuron-1 no any bunching is sketched because the expected anti-bunching effect of antenna Nerualink and repulsive forces produced by the ANT. In (B) is a critic case when a RC has evolved and creating various resistances that if perceived as sources of bunching. Apparition of resistances makes the natural neurotransmitters do not obey the Fokker-Planck equation canceling the synapse. Again, the presence of ANT would clean the synaptic cleft by accumulation and random bunching.

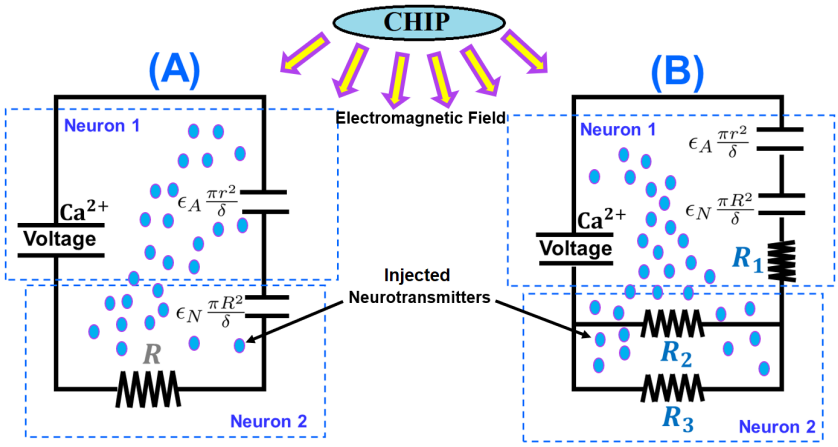

Fig. 5. Working as an antenna, the chip Neuralink emits electrical fields that would move out the neurotransmitters from a neuron to another one ANT have been injected. The lines of field would act as anti-bunching mechanism to provide traffic in both inside and outside of neurons.

B. Efficiency of Chip Neuralink

One can wonder how all above has a final straightforward impact on the improvement of neural system. Thus one can write down the efficiency of method in the sense that once the chip emits electromagnetic EM fields there is two very clear situations: (i) In one hand the artificial neurotransmitters move out or (ii) Stay static due to screening effect or cancellation of arrived fields to axon zone. Thus it is feasible to define n_M the number of ANT under moving and n_E the ones that are unaffected to EM radiation. Therefore, the efficiency of can be written as $\mathcal{E} = \frac{n_M}{n_M+n_E}$. On the other hand, the net quantity of ANT injected into blood quantified as a density is written $\rho(t)$=$Q(t)/V(t)$ so that $Q(t) = \rho(t)V(t)$. With $q(t)$ the electric charge of natural neurotransmitters, then the Coulomb-like repulsion is done by:

$$F = \frac{Q(t)\rho(t)V(t)}{r^2}. \tag{23}$$

Thus, $\mathcal{E}$ can be written under this definition as:

$$\mathcal{E} = \frac{\frac{Q(t)\rho(t)V(t)}{r^2}}{\frac{Q(t)\rho(t)V(t)}{r^2} + \frac{q(t)\rho(t)V(t)}{r^2}} = \frac{Q(t)\rho(t)}{Q(t)\rho(t) + q(t)\rho(t)} \tag{24}$$

whose simplification in the following manner:

$$\mathcal{E} = \frac{1}{1+\frac{q(t)}{Q(t)}} = \frac{1}{1+\frac{q(t)}{Q(t)} + \frac{1}{2!}\left(\frac{q(t)}{Q(t)}\right)^2 + ...} \tag{25}$$

Under the assumption that $Q(t) \gg q(t)$ then there is an excess of injected ANT over the static ones located in the axon zone of affected neuron. From Eq.16 and Eq.25 one has that:

$$\mathcal{E} = \frac{1}{1+\frac{q(t)}{Q(t)}} = \frac{1}{\text{Exp}\left[\frac{q(t)}{Q(t)}\right]} = \text{Exp}\left[-\frac{q(t)}{Q(t)}\right]$$
$$= \text{Exp}\left[-\frac{q_0 \text{Exp}\left[-\left(\frac{4\epsilon_V D}{\omega\epsilon_A r^2} - \frac{G4\epsilon_I z}{\omega\epsilon_N R^2}\right)\omega(t-t_0)\right]}{Q(t)}\right], \tag{26}$$

being this a phenomenological expression of the efficiency. Also: $Q(t)$=$\rho(t)4\pi\epsilon_N r^3/\epsilon_N$. The why is opted by ϵ_N is because ANT might be made of a similar material of the natural ones. It should be noted that it was inserted ω that is perceived as the frequency of EM wave arriving to neurons. Thus one has from Eq.26:

$$\mathcal{E} = \text{Exp}\left[-\frac{q_0\epsilon_N \text{Exp}\left[-\left(\frac{4\epsilon_V D}{\omega\epsilon_A r^2} - \frac{G4\epsilon_I z}{\omega\epsilon_N R^2}\right)\omega(t-t_0)\right]}{\rho(t)4\pi\epsilon_N r^3}\right]. \tag{27}$$

The emitted electric field would have a radial component that is responsible of ANT moving inside the axon. Thus the net electric field is given by $\mathbf{E}(r)$=$-\nabla\Phi(r)$, and subsequently one

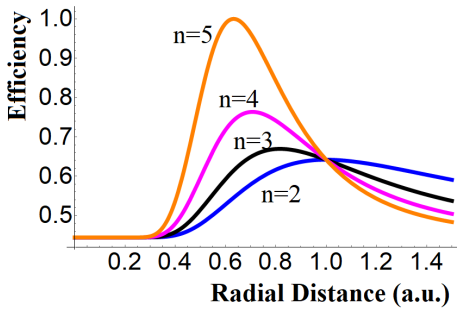

Fig. 6. Distributions of efficiencies as function of radial coordinate for up to 4 values of n. The distributions exhibit a peaked shape when n increases. Thus one can claim that a Gaussian-like form can be recognized.

arrives to:

$$\mathcal{E}(r,t) = \text{Exp}\left[-\frac{q_0\epsilon_N|\mathbf{E}(r)|e^{\left[-\left(\frac{4\epsilon_V D}{\omega\epsilon_A r^2}-\frac{G4\epsilon_I z}{\omega\epsilon_N R^2}\right)\omega(t-t_0)\right]}}{\rho(t)r}\right] \quad (28)$$

$$= \text{Exp}\left\{|\mathbf{G}(r,t)|e^{\left[-\left(\frac{4\epsilon_V D}{\omega\epsilon_A r^2}-\frac{G4\epsilon_I z}{\omega\epsilon_N R^2}\right)\omega(t-t_0)\right]}\right\} \quad (29)$$

by which has been needed to define:

$$|\mathbf{G}(r,t)| = \frac{q_0\epsilon_N|\mathbf{E}(r)|}{\rho(t)r} \quad (30)$$

Because the Coulomb-fields and the near-field character of chip running as antenna, the efficiency can be dependent on the inverse distance so that $\mathcal{E}(r^n)$ since a generalization of fields would suggest to write a normalized field electric as $|\mathbf{E}(r)| \equiv \gamma/r^n$, with γ a normalization constant. In Fig.6 the efficiencies to complete the synapse with Coulomb-like fields are displayed. One can note that for high values of n the distributions acquire a peaked morphology. In a speculative manner, for high values of n the efficiency would acquire a Gaussian-like shape. On the other side it is also valid to postulate the idea that the Weibull distributions might emerge. By knowing the probabilistic character of the Weibull family, then one could associate the set of efficiencies to a territory purely probabilistic. Therefore, it is speculative also to state the all those formalisms modeling the pushing out natural neurotransmitters by ANT might be dependent of stochastic more than a pure deterministic scenario [24]. Therefore the success at the functionality of a prospective IBT might be exclusively inside the territory of randomness to some extent.

V. Conclusion

Along this paper it was treated carefully a theory that is directly associated to the problem of the lost of transit of artificial neurotransmitters between two adjacent neurons. Thus, under the hypothesis that the chip Neuralink would behave as a near-field antenna then the radiated electromagnetic waves in particular the electric field component, might to guide although in a dispersive manner the implanted neurotransmitters to push out the bunching of natural neurotransmitters as well as ANT can also replace the role of them. From a naive usage of efficiency that incorporates Coulomb fields, various distributions have been plotted. These are interpreted in a dual manner, as curves based at Gaussian and Weibull forms, and as a probabilistic success of action to distance of near-field created by the chip Neuralink. The done investigation in this paper is not conclusive but also requires of experimental data to make robust predictions of the realistic effect of implanted chips to improve sequels of neural diseases.

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