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► **To cite this version:**

Hind Zaaraoui. Modeling of Human Reaction Creation following an Event arrival in the Brain: a quantum and a relativistic approach. 2021. hal-03432984v1

HAL Id: hal-03432984

<https://hal.science/hal-03432984v1>

Preprint submitted on 21 Nov 2021 (v1), last revised 15 Dec 2021 (v2)

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Modeling of Human Reaction Creation following an Event arrival in the Brain: a quantum and a relativistic approach

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Abstract

This work proposes a mathematical model about how a reaction is created in the human brain in response to a particular incoming Information/Event using quantum mechanics and more precisely path integrals theory. The set of action potentials created in a particular neuron N_2 is a result of temporal and spatial summation of the signals coming from different neighboring neurons N_x with different dendrite-paths. Each dendrite-path of N_2 is assumed to be determined by its respective synapse with its neurotransmitters and assumed to have an action S due to the neurotransmitter types (for example: excitatory or inhibitory). An external incoming signal information being initially modulated by receptor neurons (in eyes, ears...) travels through the neighboring neurons that are linked to the excited receptor neurons. A potential reaction responses are subsequently created thanks to a final *deformed* signal in the motor neurons by all the correlated neural paths. The total deformation at each neuron is created by different incoming dendrite-paths and their structures (inhibitory or excitatory neurotransmitters and their type), and of course the existence or not of the signal and its frequency coming from each path. Using path Integrals theory, we compute the probability of existence of the signal-Information or the potential reaction to the incoming information at each neuron. In this paper we also compute how much the signal-Information has been distorted between two neighboring linked neural points including if it arrives or not to the neighboring neurons. We propose an Information entropy similar to Shannon one and we demonstrate that this entropy is equivalent to *timespace* curvature in the Brain.

I. INTRODUCTION

In the Brain, the information disseminators are neurons and all their structures including their axons, dendrites, synapses with their respective neurotransmitters (inhibitory and excitatory). The signal unit, which is called action potential in the brain, coming from a particular neuron with its particular synapses and neurotransmitters, propagates to the linked neighboring neuron thanks to the neurotransmitters which will ensure the circulation of the *deformed* (or not) signal information until the neighboring neuron, see for example [1], [2], [3] and figure1.

If the neurotransmitters are inhibitory then the created signal in the dendrites are reducing the final computed signal amplitude which may totally eliminate the signal propagation. The signal does no more exist in space nor in time. On the contrary if the neurotransmitters are excitatory, the created postsynaptic signals in the dendrites enhance the computed signal which may ensure the continuation of the signal propagation distorted or not. From the dendrites to the neighboring neuron soma N_2 , all the incoming signals (inhibitory or excitatory) are summed in time and space [4], [5]. If the resulted amplitude exceeds a certain threshold then a new deformed signal is recreated in the studied neuron and retransmitted to the nearest linked neurons (figure 1 and [6]).

In our work we assume that each incoming dendrite-path to N_2 , determined by one synapse, provide a time phase shift of the signal coming from any neighboring neuron N_x this phase shift is what we will call the path action due to their respective type of neurotransmitters. A similar modeling has been proposed in [7] but between all the neuronal paths in the Brain. The aggregated signal at N_2 is finally modeled by the path-integrals method used in quantum mechanics [8].

The inevitable time travel between times t and $t + 1$ of any created signal Information from a neuron soma is its travel in the following path in synapses (if there are still neurotransmitters left), dendrites and neuron soma. All the components including *neurotransmitters, dendrites and neuron soma* are what we will call "Time" dimensions in the brain as the signal-Information by the studied neuron N_2 is not yet created.

The signal deformed by the *Time* arrives to the neighboring neuron soma which may be retransmitted or not, depending on the amplitude of the additionned arrived excitatory and inhibitory signals. *Space* is hence the axonic cone and axon where a potential signal may be created, or not, and retransmitted automatically in *Time*. See for example figure figure 1 for a better understanding of the structure of a neuron. One can see that *Space* and *Time* cannot be disconnected: a neuron axonal cone and axon exists if its dendrites, synapses, neurotransmitters and neuron soma exist reciprocally.

The set of the potential actions resulting in N_2 is deformed compared to the set of potential actions coming from all the neighboring neurons N_x .

We show in this paper that the deformed created signal/information in a particular neuron N_2 is induced by the incoming dendrite-paths structures linked to the neighboring neurons N_x which is modeled by path integrals formula [8]. We assume that the total signal deformation at each neuron can be finally described by the information entropy similar to the Shannon entropy. Using this entropy on the incoming signal-information from N_2 dendrites, we demonstrate that at each neuron point N_2 there is a kind of spacetime *curvature* similar to the spacetime curvature described by Einstein's General Relativity.

In this paper we will firstly describe the timespace system and signal-Information in the Brain. We will then define our mathematical model of the information diffusion between neurons in the Brain. We will show how the information aggregated in a neuron is deformed as soon as it arrives in neuron soma and how the total deformation can be seen as the curvature of spacetime. The last section explains how from this model we deduce some implications in Human behaviors/reactions.

II. ARTICLE TYPES

This paper is an Original Research article in the computational and decision Neuroscience field: mathematical modeling for the reaction/decision creation in the Brain to an event considering neural structures and computational properties.

III. MANUSCRIPT FORMATTING

A. Spacetime definition of a Reaction to an incoming information in the Brain

In this section we describe how any Information evolves and may induce a Reaction created by the Brain. Any signal-Information, or in other words a set of action potential, evolution must be studied in space and time. But what do we mean by Space and Time in the Brain? We suppose that Space is where a detectable Information can be seen or detected or in other words any location where any information could exist. For the Brain it is any neuron that can lead to an external reaction more precisely it is the axonal cone, axon and axonal endings hence where neurotransmitters are released to create a physical reaction in a muscle or another neuron...

Time is the invisible dimension that can be seen in the Information evolution or the space before any potential reaction can be created. What makes the signal-Information evolve in time in the brain ? Neurotransmitters and Neuroreceptors types, dendrites and their structures + neuron soma are the Time components in the Brain. These neuronal components force information to move, to persist, to evolve, to become sometimes *distorted* or to disappear by the inhibitory neurotransmitters effect or because of a insufficient amplitude of the aggregated signal in neuron soma. The Information signal undergoes phase time shift thanks to the all postsynaptic components.

Before arriving at the neuron soma, a signal-Information has the wave property: it can interfere with itself thanks to spatial and temporal summation of postsynaptic potentials coming from many dendrites and perhaps from many neurons. In fact the signals are added to one another if they are close in Time and space in the neuron soma. The aggregated signal has the capacity to be transformed hence to a potential internal or external Reaction if the total amplitude exceeds a particular threshold. Therefore an Information has the wave properties between two linked neurons that share synapses figure 1) in Time dimensions, and has the particle properties as soon as it is recreated by summation in the axonal cone, the Space dimensions.

The Reaction to an incoming Information behaves hence as a particle in quantum mechanics [9]. As an Information arrives to the brain it propagates, interferes with itself thanks to Time components, and as soon as a reaction is needed, there is only one total seen created action: *one cannot choose in a restaurant fixed menu both dessert and cheese*. But what is this main *decided* Reaction ? The answer is in Le Bihan's work in [10]. In this paper it has been shown experimentally that brain spacetime presents a functional curvature

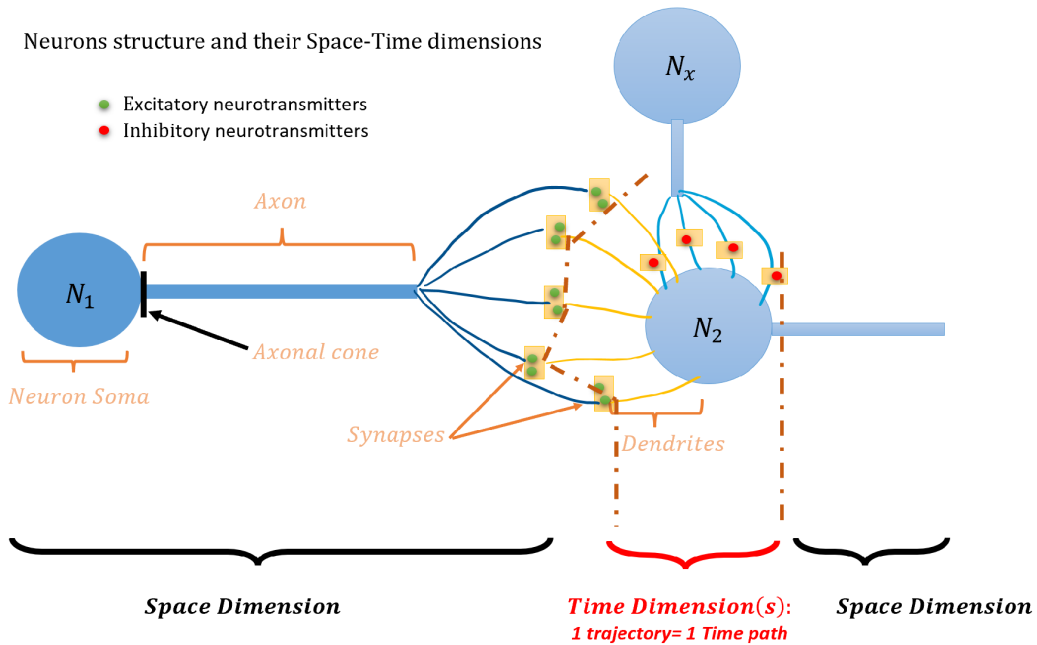


Fig. 1. Neighboring Linked neurons and their Spacetime structure

generated by brain activity, in a similar way gravitational masses give our 4-dimensional Universe spacetime its curvature. The *heaviest* correlated Reaction to the incoming Information will be the *decided* one. The aim of this paper is to reconcile quantum mechanics theory and general relativity theory following these both observations in the Brain.

But why there is only one first Reaction to an event? Because a Reaction is not only a set of excitatory neurons activated but also an internal inhibitory Information for other coming correlated reactions thanks to the presence of the inhibitory neurons (synapses with inhibitory neurotransmitters). In fact other correlated in time reactions may follow the first one if the inhibition intensity is not sufficient: we will continue to cry and/or to *act sad* if we are sad until the intensity of crying will decrease a little sadness intensity. The reaction of crying is also an input event in the Brain: it is the inhibitory event to the first crying reaction. Note that some less time correlated reactions may survive from the inhibitory effect of the *existing reactions* for a long time and could persist beyond the noise effects of the whole day's Information in real life and may *exist* lately perhaps in dreams.

An incoming Information in the brain is initially transduced to a signal-Information, filtered before following particular neural paths. The signal-Information in the brain is therefore a set of action potentials at a particular frequency that is deformed by particular neurons in the neuronal paths thanks to spatial and temporal summation in neuron soma. The action potential amplitude does not change, it's rather the frequencies of the signal unit that are changed. A final reaction is hence aggregated depending on every human experience or in other words depending on different neuronal trajectories structure for each human [11]: The sight of a dog for a given human is not objective. We can see it as a cute little animal and have the need to pet him or as a dangerous animal and run depending on one's past experiences. These two different reactions depend on the Brain SpaceTime evolution. If a dog attacked me before, my heaviest reaction I will have now is to run away. As a simple observer one will not know how the other person would react in presence of a dog if we do not know his history with dogs. The Time dimension in the brain exists and is described by the neuronal paths structures but is uninterpretable for all of us as neurons, synapses, neurotransmitters etc. are physically the same. The difference is just in neuronal path structures precisely in their geometry, their weight in terms of probabilities, and particularly in their neurotransmitters + neuroreceptors types (excitatory or inhibitory...).

B. Mathematical model for the information dissemination in the Brain

1) *Information-Reactions system model:* Between two Brain neurons N_1 and N_2 directly linked there are synapses. We suppose in our model that there are as many synapses as paths between these two neurons. However the neuron N_2 can have other synapses (hence other paths) connected to other neighboring neurons N_x figure1.

All the signals coming from the neighboring neurons N_x to N_2 are modulated by each path and reach the N_2 neuron soma where they are aggregated according to their arrival spatial and temporal phase thanks to the spatial and temporal additivity property of the postsynaptic action potentials in the dendrites. This spatial and temporal summation will ensure interference in the case of time delay between signals coming from any dendrites, or in the case of spatial distance between dendrites incoming to the soma. A same signal-Information coming from N_1 can be deformed due to different path structures between N_1 and N_2 namely by the geometry of paths and their respective neurotransmitters type.

Each neuron N_2 is activated by the signal transmission aggregated coming from the linked neuronal neighborhood N_x . This signal or Information amplitude is denoted by $\psi(N_x, t)$ the wave function of signal neuron N_x at time t that is complex as we allow that signals can interfere with one another. If $\psi(N_{x_0}, t) = 0$ then there is no signal coming from the neuron N_{x_0} at time t .

When traveling in the N_x axon until the collaterals and axonal terminaison, the signal $\psi(N_x, t)$ remains the same; the only difference is that the signal speed changes along the path due to membrane channel composition and particularly to their diameters. It's known that in the axon (depending on each neuron) the speed of the signal propagation is the highest comparing to the speed in axonal terminaisons and dendrites. As soon as the signal arrives to the synapse, the signal causes the exocytosis of neurotransmitters: the wave is converted into matter which subsequently disappears just after being linked to the neuro-receivers and retransformed to their respective signals: inhibitory signals if the neurotransmitters are inhibitory or excitatory signal if the transmitters are excitatory [12]. The total signal-Information coming to N_2 is finally deformed due to all these paths components' action and also due to paths coming from other correlated neighboring neurons.

The following modeling is particularly similar to the one that H. Zaaraoui has suggested in [13] for the Information dissemination in the universe.

The signal-Information from N_x to N_2 in the Brain Time dimensions undergoes in each Time path at each time unit du figure2 a normalized action $d\bar{S}$ per unit time, or a phase shift, defined as following:

$$\frac{d\bar{S}}{du} = \frac{L}{\hbar} \Rightarrow \bar{S} = \int \frac{L}{\hbar} du \quad (1)$$

$\frac{d\bar{S}}{du}$ is the action unit of the neurotransmitters in a particular path. L is a path Lagrangian and \hbar is supposed to be a constant (the reduced Planck constant for Universe laws) [19]. $\frac{1}{\hbar}$ acts as the normalization of the energy density in time where if Lagrangian L is equal to one energy unit L_1 (one action potential for brain) for one time unit t_1 , hence:

$$\int_0^{t_1} \frac{L_1}{\hbar} du = 1 \quad (2)$$

where $\frac{L_1}{\hbar}$ would be equivalent to the probability density to find a quantum of energy at u in probability theory, with \hbar the maximal quantity of energy that can hold a temporal distance unit, the Time distance between two neurons N_1 and N_2 . With this definition, one can note that \bar{S} is the mean of $\frac{L}{L_1}$ which is the number of energy units, or in the brain the number of action potentials at time u .

The signal-Information propagating in a particular path with incidence r into N_2 undergoes a total phase shift of \bar{S}_r from t_0 to t_f figure3, and the modified signal in this path is:

$$\psi(N^r, t_f) = \psi(N_x, t) e^{i\bar{S}_r} = \psi(N_x, t) e^{i(\frac{S_r}{\hbar} - \delta_{inh,r,t}\pi)} \quad (3)$$

where $S_r = \int_{t_0}^{t_f} L_r du$ the path action, and $\delta_{inh,r,t}\pi$ is equal to π if the Information mediators are inhibitory neurotransmitters coming with incidence r at time t . $\delta_{inh,r,t} = 0$ if the mediators are an excitatory neurotransmitters. Adding $-\delta_{inh,r,t}\pi$ term to the action of a path allows the inhibition of the signal. In fact, if the neurotransmitter in a particular path is inhibitory hence $e^{i\frac{S_r}{\hbar} - \pi} = -e^{i\frac{S_r}{\hbar}}$ which ensures the suppression of a part of the signal in N_2 while summing all the incoming signals. N^r is the virtual image of the neuron

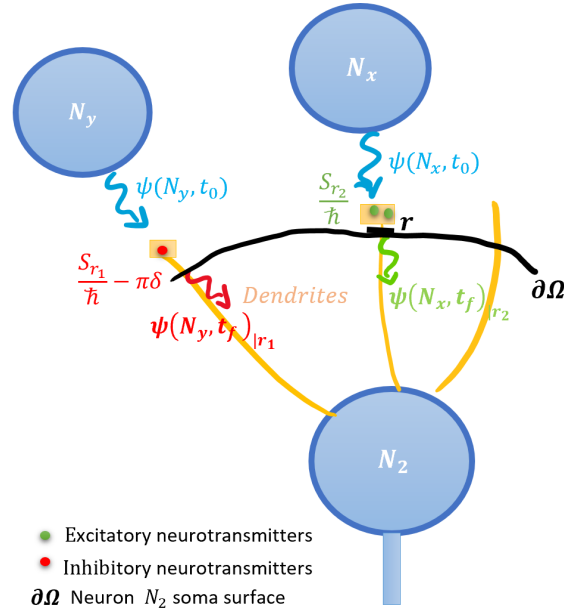


Fig. 3. Signal-Information propagation with Time path action

(knowing the value of $\delta_{inh,r,t}$), with complex density where R and T are the random variables defined as follows:

- R represents the random variable of the signal existence coming from the virtual point N^R i.e. random variable of the existence of dendrite-path with incidence r and the presence of the *signal* coming with incidence r ,
- T is the random variable of the summation possibility of signals coming from the neighboring points i.e it represents the time summation capacity: interference in time.

Hence we can see mathematically $\psi(N_2, t_f)$:

$$\psi(N_2, t_f) = \mathbb{E}[D(q_R(T))|\delta_{inh,R,T}] \quad (7)$$

Now that an Information may be reconstructed thanks to temporal and spatial summation of action potentials wave functions, we want to know how much an incoming Information has been deformed due to different Time dendrite-paths including their respective neurotransmitters between all the neighboring N_x or N^r and N_2 . We will show that a particular information entropy of wave function of paths arriving to N_2 define the scalar curvature term \mathcal{R} [14] in Hilbert-Einstein action [15], [16].

C. Information Entropy and Brain SpaceTime curvature

In this section we want to measure the average level of disorder of the signal-Information coming from N_x received by a Neuron point N_2 , or in other words how much the signal coming from the neighboring neuron N_x is distorted compared to the resulted signal in the neuron N_2 . We introduce here an entropy that is between the Shannon entropy in information theory [17] and the one introduced in the paper [18] which includes Lagrangian ([13]). We recall that Shannon entropy measures the information quantity sent by an information source. The more different information the source emits, the greater the entropy (or uncertainty about what the source emits).

The spatio-temporal entropy of a signal ψ at time t_f in location N_2 is defined in this paper by:

$$\mathfrak{S}(\psi(N_2, t_f)) = \mathcal{L}_m \sqrt{|g|} + \int_{\partial\Omega} \int_{\Delta t} -p(r, t) \times (\mathfrak{L}_{m_r} + \gamma(r, t)) dt dr \quad (8)$$

$p(r, t)$ is the density of the incoming signal amplitude per surface and time at N_2 with incidence r for the signal-Information in the Brain where $p(r, t) = |\psi(N^r, t)|^2$. If there is no signal-Information present in N_r ,

the virtual image of N_x in N_2 in the path q_r , or there cannot be a Time dendrite-path between N_r and N_2 hence $p(r, t) = 0$: p encompass the *probability* or more precisely the density of energy/mass per surface and time of initial incoming Information with incidence r . $\gamma(r, t)$ is the spatio-temporal phase shift at time t of the path incoming from r without the matter Lagrangian part:

$$\gamma(r, t) = \frac{S}{\hbar} - \delta_{inh,r,t}\pi \quad (9)$$

S , or noted before S_r , is the action on the signal-Information unit in a particular Time path with incidence r that can be defined as following:

$$\frac{S}{\hbar} = \int^t \frac{1}{2} \rho \left(\frac{dq}{du} \right)^2 du. \quad (10)$$

where ρ represents the mass equivalent to the energy amplitude of the action potential ($\sim 30mV$ before arriving to synapses) thanks to the Einstein's mass-energy equivalence formula $E = mc^2$. $\frac{dq}{du}$ is the speed of that quantum of Information (action potential) in the axon, dendrites etc.

In the brain, there is no complex interactions between action potentials from different Time paths but only propagation in each path and then summation or inhibition of signals to create or not a new signal in the neuron soma. We can deduce then that $\mathcal{L}_m \sqrt{|g|} \sim \int_{\partial\Omega} \int_{\Delta t} -p(r, t) \mathcal{L}_m dt dr$ where \mathcal{L}_m is the Lagrangian or the phase of the initial outgoing signal-Information from N_x figure3. $\sqrt{|g|}$ aggregates the effect of the density p of the incoming information which can be also assimilated to the mean correlation or the mean distance degree between neurons. This term will be more explained later in the paper.

As $p(r, t)$ represents the incoming amplitude energy density per surface and time in brain, to normalize $p(r, t)$, we multiply and divide by $\frac{M_p}{S_p t_p}$ where M_p is the maximum of equivalent mass of a set of action potential that could come during a minimal time t_p and surface S_p (analogous to Planck constants in Physics). We consider now $\xi(\psi(N_2, t_f))$ without the Lagrangian density of matter \mathcal{L}_m :

$$\xi(\psi(N_2, t_f)) = \frac{M_p}{S_p t_p} \int_{\partial\Omega} \int_{\Delta t} -P(r, t) \times \gamma(r, t) dt dr \quad (11)$$

where $P(r, t) = \frac{p(r, t)}{\frac{M_p}{S_p t_p}}$.

In the neuron soma there is a temporal summation of all the action potentials arrived during Δt , a duration which after the neuron may create or not a new action potential or a signal-Information unit that will be sent to neighboring linked neurons with the condition that the sum of signal amplitude exceeds a certain threshold. This duration is the *tolerance* time that any information unit with respective weight coming from anywhere may be aggregated to create at N_2 at time t a new information unit: an action potential in Brain.

If the total amplitude exceeds the threshold before achieving duration Δt , the elementary signal-Information is directly created in Space dimensions.

Using the same calculus method as [13], we have:

$$\xi(\psi(N_2, t_f)) = \frac{M_p^2}{S_p t_p \hbar} \int_{\partial\Omega} \int_{\Delta t} -P(r, t) \times \left[\int^t \frac{1}{2} \frac{\rho}{M_p} \left(\frac{dq_r}{du} \right)^2 du - \frac{\hbar}{M_p} \delta_{inh,r,t} \pi \right] dt dr \quad (12)$$

$-P(r, t) \left[\int^t \frac{1}{2} \frac{\rho}{M_p} \left(\frac{dq_r}{du} \right)^2 du - \delta_{inh,r,t} \pi \right]$ represents the variation in time of spatio-temporal square distance that a signal-Information travels in Brain from two nearest points N_x to point N_2 taking the dendrite-path with an incidence r in N_2 at time t_2 figure2. We note the variation in time of square distance of the particular Time dendrite-path with incidence r without the inhibition part $\frac{\partial g_{x_r,t}}{\partial t}$:

$$\frac{\partial g_{x_r,t}}{\partial t} = -P(r, t) \left[\int_{t_1}^t \frac{\rho}{M_p} \left(\frac{dq_r}{du} \right)^2 du \right] \quad (13)$$

and the variation in time of square distance of the inhibitor Time path with incidence r $\frac{\partial \bar{g}_{x_r,t}}{\partial t}$ where:

$$\frac{\partial \bar{g}_{x_r,t}}{\partial t} = P(r, t) \frac{\hbar}{M_p} \delta_{inh,r,t} \pi \quad (14)$$

The distance between two points N_x and N_2 is infinite if no Information can propagate between them i.e. if $P(r, t) = 0$ for all Time incidence r at N_2 . In other words the initial condition is a Dirac distribution in

the sense that it is infinite at time $t = 0$. $\frac{\partial g_{x_r,t}}{\partial t}$ is surely to be negative if the mediators are excitatory (as $\delta_{inh,r,t}\pi = 0$). This means that the distance between the two neurons is decreasing between times t_1 and t if $P(r, t)$ is strictly positive i.e. the path exists, the neurotransmitters are excitatory and the signal-Information exist (which is included in P). This square distance is an equivalence to the correlation factor between neurons to a particular incoming signal-Information.

However if the mediators are inhibitory then $-\delta_{inh,r,t}\pi = -\pi$ which may increase the distance between the neurons as $\frac{\partial \bar{g}_{x_r,t}}{\partial t}$ could be positive. This leads to a kind of a virtual spatio-temporal expansion between these two nearest points N_x and N_2 . We rewrite the Information entropy:

$$\S(\psi(N_2, t_f)) = \frac{M_p^2}{S_p t_p \hbar} \left[\frac{1}{2} \int_{\partial\Omega} \int_{\Delta t} \frac{\partial g_{x_r,t}}{\partial t} dt dr + \frac{\hbar}{M_p} \int_{\partial\Omega} \int_{\Delta t} P(r, t) \delta_{inh,r,t} \pi dt dr \right] \quad (15)$$

We consider now as in [13]:

$$\begin{aligned} -\vec{n} dS &= dr \text{ surface unit} \\ -\vec{n} &= \text{incoming normal vector to } dS \\ \vec{F} &= \overrightarrow{\text{grad}} \frac{\partial g_{x_r,t}}{\partial t} \\ \vec{\Pi} &= \overrightarrow{\text{grad}} \left(\frac{\hbar}{M_p} P(r, t) \delta_{inh,r,t} \pi \right) = \overrightarrow{\text{grad}} \frac{\partial \bar{g}_{x_r,t}}{\partial t} \end{aligned} \quad (16)$$

where $\vec{\Pi}$ can be rewritten as follows:

$$\vec{\Pi} = \frac{\hbar}{M_p} \pi P(r, t) \overrightarrow{\text{grad}}(\delta_{inh,r,t}) \quad (17)$$

Hence the scalar product of $\vec{n} dS$ and $\vec{\Pi}$:

$$-\vec{n} dS \cdot \int^t \vec{\Pi} du = -\frac{\hbar}{M_p} \pi P(r, t) \delta_{inh,r,t} dS \quad (18)$$

\vec{F} is the resultant vector of a particular path which narrows the distance between N_x and N_2 neurons and $\vec{\Pi}$ is the vector that increases this distance. One can note that to N_2 the distance is identical to any direct neighboring neuron N_x as the distance is only the sum of all these vectors from any incoming path to N_2 figure2. The distance between two any linked neighboring points N_x and N_2 depends on the Time paths and on the initial incoming signal-Information. Space and Time are hence relative depending on what is going on between the nearest two spatial points and on the incoming/present signal-Information.

Hence we have, as the method used in [13], using Gradient theorem:

$$\begin{aligned} \S(\psi(N_2, t_f)) &= \frac{M_p^2}{S_p t_p \hbar} \left[\frac{1}{2} \int_{\partial\Omega} \int_{\Delta t} \int^t -\vec{F} \cdot \vec{n} du dt dS \right. \\ &\quad \left. + \frac{\hbar \pi}{M_p} \int_{\partial\Omega} \int_{\Delta t} \vec{\Pi} \cdot \vec{n} du dt dS \right] \end{aligned} \quad (19)$$

Using Ostrogradsky theorem and approximations, and finally thanks to the action principle or the entropy minimizing we have the total Einstein equation of general relativity (see the demonstration in [13]):

$$Ric - \frac{1}{2} \mathcal{R} + \Lambda g = \frac{1}{\kappa} T \quad (20)$$

:

where Ric is the Ricci tensor, \mathcal{R} is the scalar curvature, Λ is the cosmological constant, κ is the Einstein constant and T is the energy-momentum tensor. But what does this equation mean in Brain terms?

Implications:

The curvature (Ric , \mathcal{R}) variables describe the impact of the excitatory neurotransmitters and their respective dendritic-paths on the signals-Information dissemination. They describe the way neurons and their structures would impact the incoming signal-Information T . T is the information of all the signals propagating in all the neurons from their axonal cones until there axonal endings at the moment when we study the Brain. If there

were no signals propagating, hence $T = 0$ there is no reaction activated. Λ is the impact of the inhibitory neurons if they are activated by the propagating signals. It is one of the reasons why some reactions are delayed or inhibited. g may represent the correlation between neurons i.e. the *Information* distance between neurons depending on the signals present when we study the brain. Two neurons can be infinitely distant if there are no paths or no signals propagating between them ...

We can conclude by this equation that the gravity effect is seen in the Final Reaction to an incoming Information: The *decided* Reaction is the *heaviest* and the most *correlated* one to the incoming Information/Event. A signal-Information propagates in the neuronal paths that are most correlated to the initial incoming signals in the Brain until arriving to the *heaviest* and the most correlated final *observed* reaction: this the gravity effect on the incoming Information/Event of the existing reactions in the Brains.

D. Implications on Human behaviors

Gravity in the Brain: An incoming information activates the most correlated and heaviest action i.e. neurons that have the most number of excitatory Time paths (dendrites-paths with excitatory neurotransmitters) linked to the initial activated neurons thanks to the general relativity equation demonstrated in [13].

Black Holes in the Brain: It is just like a neuron that its dendrites are destructed or have most of its dendrites are inhibitory. Any incoming information to these type of neurons is lost and no action related to these neurons can be created.

Quantum decoherence in the brain: The presence of inhibitory neurons i.e. with inhibitory neurotransmitters ensures the quantum decoherence of later reactions to an incoming Information. As soon as a reaction is created by the brain, this reaction becomes a new Event /information that is processed in the Brain and has an inhibitory effect on all reactions correlated to the first one: hence the only one first reaction created is the first one. Other correlated reactions may later follow.

CONFLICT OF INTEREST STATEMENT

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

AUTHOR CONTRIBUTIONS

The author gives a new way on how one can model the creation of a reaction in the Brain to an event using quantum mechanics and precisely paths integrals theory and the path action, where a path is defined by one synapse with its particular neurotransmitters and its action is the postsynaptic action of these neurotransmitters. The main contributions are the mathematical models for the signals propagation and deformation.

FUNDING

No Fundings.

PROOF OF THE FINAL EQUATION

All the theoretical demonstration of the formulas are demonstrated in [13]

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