

QUANTUM WAVE PROPAGATION THROUGH INTERCONNECTED NEURONS: A MATHEMATICAL SYNTHESIS

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Abstract

In this study, propagation of quantum waves through neurons has been studied via partial differential equations, a mathematical approach. Central to the exploration is a Schrödinger-like equation which models the quantum wave propagation in a neuron. The analysis delves into how the wave function, contingent on spatial position and time, is influenced by potential barriers and its initial conditions. Complimenting this is an equation capturing the dynamics of neurons. This equation underscores the relationship between the quantum wave function and the neuron firing rate. By employing a discretized spatial domain and initializing the wave function with a Gaussian wave packet, iterative numerical techniques have been utilized to glean insights into the temporal evolution of this function. The presented model is further refined by incorporating boundary conditions and additional equations that factor in external stimuli and neuronal connectivity. In conclusion, the presented mathematical framework hypothesizes connections. While awaiting empirical validation, the presented mathematical constructs pave the way for fresh perspectives and methodologies in understanding neural processes.

Keywords: Quantum Neurology, Schrödinger-like Equation, Neuronal Dynamics, Wave Function Propagation, Iterative Numerical Techniques.

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synaptic firings, which classical physics finds

Introduction

In the mid-20th century, as the pillars of quantum mechanics were firmly established, scientists began speculating its implications beyond the atom's realm. Erwin Schrödinger, a pioneer in quantum mechanics, delved into biology's enigmatic questions in his 1944 book "What is Life?" (Schrödinger, 1944). While not directly about the brain, he mused on the quantum nature of biological processes, laying the foundational thoughts for future explorations in quantum neurology. The late 20th century witnessed one of the most prominent and controversial theories that explicitly wed quantum mechanics and neuroscience. In the 1990s, Sir Roger Penrose, a mathematical physicist, and Dr. Stuart Hameroff, an anaesthesiologist, collaboratively proposed the Orchestrated Objective Reduction (Orch-OR) model (Hameroff and Penrose, 2014). They posited that quantum processes in the brain's microtubules give rise to consciousness. This theory faced considerable scepticism but invigorated the discussion around quantum effects in neural processes. Around the same time, Frohlich (1968) proposed that coherent quantum states might exist within biological systems, specifically neurons. This idea, though met with scepticism, paved the way for discussions about quantum tunnelling in synapses. Beck and Eccles (1992), and Eccles et al. (1994) later expanded on this by suggesting that quantum tunnelling of electrons might play a crucial role in synaptic functions. The turn of the millennium brought forth critiques of the nascent field of quantum neurology. Physicist Max Tegmark (2000) calculated that quantum states in the brain would undergo decoherence (lose their quantum nature) much faster than the timescales on which neuronal processes occur, seemingly debunking theories like Orch-OR. However, Hameroff and Penrose countered, suggesting mechanisms by which the brain might protect its quantum states (Hameroff, 2014). With advances in quantum biology in the 2000s, where researchers demonstrated quantum effects in photosynthesis and bird migration (Engel et al., 2007; Ritz et al., 2000), the belief in quantum mechanics playing a role in more extensive biological systems, including the brain, gained traction. For instance, quantum entanglement, a phenomenon where particles become interconnected, was discussed in the context of the brain's simultaneous, coordinated activities across vast distances (Arndt and Hornberger, 2014). Modern explorations in quantum neurology are not solely focused on consciousness. Research has expanded to understanding various neural processes, including the probabilistic nature of

challenging to explain (Koch and Hepp, 2006). The synthesis of quantum mechanics with neural network models is another avenue being explored. While been through the literature of quantum neurology, recent advancements in the field have been recorded. In the 2010s, Hameroff and Penrose (2014) revisited and expanded on their Orch-OR theory. They proposed that vibrations in microtubules might be the source of quantum effects observed in the brain. Experiments using anaesthetics seemed to support this idea by showing that certain gases selectively dampened these vibrations, leading to unconsciousness. Quantum cognition, a relatively new field, applies quantum mechanics to cognitive processes, suggesting that the probabilistic nature of quantum mechanics could model cognitive processes more accurately than classical models. Not necessarily implying the brain is a quantum computer, but that quantum mathematics offers different а perspective on understanding cognition (Busemeyer and Bruza, 2012). Although entanglement is usually associated with quantum particles, there has been growing interest in its potential analogues in brain activity. The idea is that the simultaneous firing of neurons might be likened to entangled particles (Atmanspacher, 2015). While still speculative, this has prompted research into the synchronization of brain regions and its potential quantum underpinnings. Studies in quantum biology have shed light on possible quantum processes in the brain. For instance, magnetic fields produced by certain reactions involving radical ion pairs could potentially influence neuron firing rates. This has been a mechanism proposed as for birds' magnetoreception and could, in theory, have implications for brain processes (Hore and Mouritsen, 2016). Advancements in nanotechnology have led to quantum dots' utilization, nanoparticles with quantum properties, in neuroscience research. These dots have been used as sensors and probes, providing new insights into neuron function (Michalet et al., 2005). One of the more critical reviews in recent years has come from Tegmark (2014), who recalculated the timescales of quantum decoherence in neural systems, arguing that the brain is too warm and wet for quantum processes to play a significant role. These findings suggest that while quantum mechanics is essential at the molecular scale, its direct influence on brain function remains unlikely.

Mathematical modelling acted as a catalyst to expedite the research in quantum neurology. Recent years have seen the application of quantum formalism to cognitive modeling, utilizing Hilbert space formalism to capture the probabilistic nature of decision-making processes (Busemeyer et al., 2006). Such mathematical frameworks provided better fits to experimental data than classical probability models in certain cognitive tasks, like the order effect or conjunction fallacy (Khrennikov, 2011). Quantum walks, a quantum analog of classical random walks, had been proposed as models for certain brain processes. Research suggested that these walks could capture the probabilistic nature of neuron firing sequences, offering a new perspective on synaptic connectivity and neuronal pathways (Abbott et al., 2008). The quantum vibrations in microtubules, as proposed by the Orch-OR theory, had undergone rigorous mathematical modeling to explain phenomena like anesthesia. These models described electron tunneling processes within microtubules, attempting to pinpoint the quantum processes responsible for consciousness (Hameroff and Penrose, 2014). Building on the speculative idea of neural entanglement, some researchers have derived mathematical measures to quantify potential entanglement between neuron clusters, based on synchronized firing patterns and other observed phenomena (Atmanspacher, 2004). Taking inspiration from quantum field theory, some researchers have started framing neural dynamics in terms of field theory. This approach captured the continuous nature of certain brain activities, like electromagnetic waves, using quantum field theoretical tools (Lloyd, 2011). Thus, even if quantum neurology remains on the periphery of mainstream neuroscience, the mathematical innovations it has inspired over the past decade cannot be overlooked. The continuous cross-pollination between quantum mechanics and neurology with the aid of mathematical modelling is bound to yield new perspectives and methodologies. This motivated the authors to study the present problem of quantum wave propagation in neurons mathematically using partial differential equations and numerical iterative techniques.

Materials and Methods

In the study, a hybrid approach of theoretical modeling and numerical solution has been used to investigate how quantum wave behaviour influences interconnected neurons within neural networks. The methodology revolved around adapting Schrödinger's equation, a fundamental equation in quantum mechanics, to represent the synaptic firing probabilities of neurons. A simplified neural network model has been constructed, specifying neuron density, connectivity patterns, and synaptic potential barriers. Through numerical solution with varied parameters, including neuron density, connectivity strength, and external stimuli, the impact of quantum physics on neural network dynamics has been observed (Hertäg et al., 2014). The analysis included the quantification of quantum wave behaviour, neuronal firing rates, and synaptic potential changes over time. External stimuli to assess their influence on the quantum wave function and subsequent neuronal firing have also been introduced. This combined theoretical and numerical approach sheds light on the intricate interplay between quantum physics and neuroscience, providing valuable insights into how quantum phenomena affect neuronal behaviour in interconnected networks.

Problem Formulation

Consider a scenario where quantum effects in neural networks, specifically in synapses, are believed to create quantum wave propagations. These quantum wave propagations are theorized to influence the likelihood of synaptic firings. A mathematical model will be presented using these quantum waves propagating through a series of interconnected neurons.

Parameters:

 $\Psi(x, t)$: The quantum wave function representing the probability amplitude of synaptic firings.

V(x, t): The potential energy representing synaptic barriers.

m: The effective mass of the quantum particle responsible for synaptic firing.

ħ: Planck's constant.

n(x, t): The density of neurons in a region.

v: The velocity or speed of movement (transport) of the neurons.

 σ : A decay or damping coefficient for the neuronal response.

 κ : A base rate or intrinsic rate of change of the neuronal network's response in the absence of external stimuli.

D: Diffusion constant of quantum particles.

S(x,t): Source term, which denotes external stimuli.

C: Connectivity matrix indicating connections between different neurons.

f(n): A function representing the firing rate of a neuron given its density.

 α, β : Damping and excitation coefficients for the quantum waves.

r(x,t): Response function of a neuron due to quantum effects.

Mathematical formulation:

Considering 1-dimensional segment of a neuron, where x ranges from 0 to L (length of the segment),

Section A -Research paper

(11)

the partial differential equations governing the dynamics of quantum waves and the neurons in synapses are framed as,

Schrödinger's equation for synaptic firings:

$$\iota \hbar \frac{\partial \Psi}{\partial t} = -\frac{\hbar^2}{2m} \frac{\partial^2 \Psi}{\partial x^2} + V \Psi$$
(1)

Conservation of neurons: $\frac{\partial n}{\partial t} + \frac{\partial (nv)}{\partial x} = S$

(2) where, the term $\frac{\partial(nv)}{\partial x}$ describes the rate of change of the product of neuron density and velocity with respect to space. It gives a measure of the net flow or flux of neurons at a given point in space.

Quantum diffusion of particles in synaptic clefts: $\frac{\partial n}{\partial n} = D \frac{\partial^2 n}{\partial n}$

$$\partial t = D_{\partial x^2}$$
 (3)

Neuronal connectivity: $\frac{\partial n}{\partial t} = C n$

Firing rate dependence on quantum waves: $\frac{\partial f}{\partial t} = \beta |\Psi|^2 - \alpha f$ (5)

Evolution of the synaptic potential barrier with time:

$$\frac{\partial V}{\partial t} = -\gamma f(n) + \lambda r$$
(6)

where, γ and λ are proportionality constants. Neuronal response due to quantum wave: $\frac{\partial r}{\partial t} = \theta |\Psi|^2 - \sigma r$ (7)

where, θ is a proportionality constant. Conservation of quantum particles: $\frac{\partial |\Psi|^2}{\partial t} + \frac{\partial (|\Psi|^2 v)}{\partial x} = 0$

(8)

(4)

This can be seen as a quantum fluid continuity equation where $|\Psi|^2$ acts as density and v as the velocity.

Neuronal network's reaction to external stimuli: $\frac{\partial s}{\partial s} = \kappa - n n$

$$\frac{\partial t}{\partial t} = \kappa - \eta \, n \tag{9}$$

where, η is a proportionality constant that represents the sensitivity or responsiveness of the neuronal network to external stimuli.

Connectivity influence on wave function:

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$$\frac{\partial \Psi}{\partial t} = C \Psi \tag{10}$$

Assume some hypothetical data before considering boundary conditions based on the provided framework given as,

Length of segment, L = 1 micrometer.

Initial quantum wave propagation:
$$\Psi(x, 0) = e^{\frac{-x^2}{0.01}}$$
. (12)

This implies that the quantum wave is localized around x = 0 at t = 0 and decays exponentially as we move away from this point.

Initial distribution of neurons (or more accurately, synaptic density or quantum particle density in this $|x|^{L_1}$

context):
$$n(x, 0) = e^{\frac{-|x-\frac{2}{2}|}{0.1}}$$
. (13)

This suggest that the synaptic density is highest at the midpoint of the segment and decays exponentially as we move away from the midpoint. Initial and Boundary Conditions:

 $\Psi(x, 0)$, i.e., initial quantum wave propagation at t = 0 is given as,

$$\Psi(0,0) = e^0 = 1; \Psi(L,0) = e^{\frac{-L^2}{0.01}} = e^{\frac{-1}{0.01}}.$$
(14)

n(x, 0), i.e., initial distribution of neurons is given as,

$$n(0,0) = e^{\frac{-\left|0-\frac{L}{2}\right|}{0.1}} = e^{\frac{-L}{2}}, \ n(L,0) = e^{\frac{-\left|L-\frac{L}{2}\right|}{0.1}} = e^{\frac{-L}{2}}.$$
(15)

No-flux boundary conditions for n and Ψ at domain boundaries are given as,

$$\frac{\partial \Psi}{\partial x}\Big|_{x=0} = 0; \ \frac{\partial \Psi}{\partial x}\Big|_{x=L} = 0; \ \frac{\partial n}{\partial x}\Big|_{x=0} = 0; \ \frac{\partial n}{\partial x}\Big|_{x=L} = 0.$$
(16)

V is bounded and does not blow up to infinity. For instance, assume that V(x) varies sinusoidally along the segment with amplitude A and is given as,

$$V(x) = A \sin\left(\frac{2\pi x}{L}\right),\tag{17}$$

where, A is a constant. For this case, assume that A = 0.5 eV (electron volts).

S(x, t) is periodic or some known function of time assumed as,

$$S(x,t) = \cos\left(\frac{2\pi t}{T}\right),\tag{18}$$

where T is the period. Assume T = 1 second for simplicity.

(20)

Analytical Results

For solution of equation (1), assume the potential V(x) represents a common quantum scenario: the quantum harmonic oscillator, which might represent a neuron trapped in a potential well due to surrounding synapses. The potential for the quantum harmonic oscillator is,

$$V(x) = \frac{1}{2}m\omega^2 x^2,\tag{19}$$

Solution of equation (1) using method of separation of variables is assumed as, $\Psi(x, t) = \phi(x) T(t)$.

Plugging equations (19) and (20) in equation (1), and introducing the separation constant E (which can be interpreted as energy), gives two separate differential equations – time-dependent equation and space-dependent equation. Solutions of these differential equations when put in equation (1) give,

$$\Psi_n(x,t) = \phi_n(x)T_n(t), n = 0, 1, 2, \dots$$
(21)

where,
$$T_n(t) = e^{\frac{-iE_nt}{\hbar}}$$
; $\phi_n(x) =$

$$\frac{1}{\sqrt{2^n n!}} \left(\frac{m\omega}{\pi\hbar}\right)^{1/4} e^{\frac{-m\omega x^2}{2\hbar}} H_n(\xi), \qquad (22)$$

where, $E_n = \hbar\omega\left(n + \frac{1}{2}\right)$ represents the quantized energy states, $\Psi_0(x, t)$ is the ground state, $\Psi_1(x, t)$ is the first excited state, and so on. Each of these states has a different energy and different special distribution, given by the Hermite polynomials $H_n(\xi)$. The set of solutions (21) could suggest quantized energy states for the neuron under synaptic conditions.

Quantum diffusion equation (3) is a classic diffusion equation, whose general solution is given as,

$$n(x,t) = \int_{-\infty}^{\infty} G(x - x', t - t') S(x', t') dx' dt',$$
(23)

where G is the Green's function for the diffusion equation, and is given as,

$$G(x,t)=\frac{1}{\sqrt{4\pi Dt}}e^{\frac{-x^2}{4Dt}}.$$

This equation states that the neuron density at time t and position x depends on source terms S at all previous times and positions, weighted by the Green's function.

Neuronal connectivity equation (4) is an exponential growth/decay equation. The general solution, given a constant *C*, is given as, $n(t) = n(0)e^{Ct}$.

Using the initial condition (13), the particular solution of equation (4) will be,

$$n(t) = e^{Ct - \frac{|x - \frac{L}{2}|}{0.1}}.$$
(24)

Firing rate dependence equation (5) is a linear firstorder differential equation. A general solution involves integrating factors, but the solution will depend on $|\Psi|^2$, the probability density of the quantum wave. Assume the scenario, where the probability density for the quantum harmonic oscillator in the ground state (i.e., n = 0) is given by $|\phi_0(x)|^2$. For the ground state (making use of equations (21) and (22)),

$$\begin{split} \Psi_0(x,t) &= \phi_0(x) e^{-\iota E_0 t/h}, \quad \text{where} \quad \phi_0(x) = \\ \left(\frac{m\omega}{\pi\hbar}\right)^{1/4} e^{\frac{-m\omega x^2}{2\hbar}} \end{split}$$

So, probability density is given by,

$$|\Psi_0(x,t)|^2 = |\phi_0(x)|^2 = \left(\frac{m\omega}{\pi\hbar}\right)^{1/2} e^{\frac{-m\omega x^2}{\hbar}}.$$
(25)

Using this, the firing rate dependence equation reduces to the equation,

$$\frac{\partial f}{\partial t} = \beta \left(\frac{m\omega}{\pi\hbar}\right)^{1/2} e^{\frac{-m\omega x^2}{\hbar}} - \alpha f$$

Solution of this firing rate dependence equation using an integrating factor $e^{\alpha t}$, is given as,

$$f(t) = \beta \left(\frac{m\omega}{\pi\hbar}\right)^{1/2} e^{-\alpha t} \int e^{\alpha t} e^{\frac{-m\omega x^2}{\hbar}} dt + C,$$
(26)

Where *C* is an integration constant. This equation gives the firing rate as a function of time, modulated by the quantum probability density. Equation (10) giving connectivity influence on wave function is a first-order partial differential equation that describes exponential growth/decay of the wave function based on the connectivity influence factor *C*. Using the initial condition (12), the particular solution of equation (10) is given as,

$$\Psi(x,t) = e^{Ct - \frac{X^2}{0.01}}.$$
(27)

In the equation (9) for neuronal network's reaction to external stimuli, assume that n is constant or slowly varying compared to S (e.g., in a scenario where the external stimulus is rapidly changing while the neuron density remains relatively constant). Also assume that η is a constant (characterizing the rate of decay of the stimulus in presence of neurons). Using an integrating factor $e^{\eta t}$, the solution of equation (9) is given as,

$$S(t) = \kappa \frac{e^{-\eta t}}{\eta} + C e^{-\eta t}, \qquad (28)$$

where, *C* is an integration constant. Using initial condition (18), the value of *C* obtained is $1 - \frac{\kappa}{\eta}$. Hence, the particular solution of equation (9) comes out to be, $S(t) = e^{-\eta t}$. (29)

Coming to equation (3) for conservation of neurons with quantum influence, where v might ne influenced by the quantum state $|\Psi|^2$, assume that $v = v_0 + \gamma |\Psi|^2$, (30)

where v_0 is a base velocity and γ is a modulation constant. Plugging the equations (25), (29) and (30), into equation (3) yields,

$$\frac{\partial n}{\partial t} + \frac{\partial}{\partial x} \left\{ n \left(v_0 + \gamma \left(\frac{m\omega}{\pi\hbar} \right)^{1/2} e^{\frac{-m\omega x^2}{\hbar}} \right) \right\} = e^{-\eta t}.$$
(31)

Method of characteristics can be used to find analytical solution of this equation.

Coming to equation (7) for neuronal response due to quantum effects, equation (25) will be plugged into this equation to give,

$$\frac{\partial r}{\partial t} = \theta \left(\frac{m\omega}{\pi\hbar}\right)^{1/2} e^{\frac{-m\omega x^2}{\hbar}} - \sigma r.$$
(32)

This equation suggests the neuronal response r is modulated by the quantum state and decays with a rate σ . It is a linear ordinary differential equation whose general solution is given as,

$$r = \frac{\theta}{\sigma} \left(\frac{m\omega}{\pi\hbar}\right)^{1/2} e^{\frac{-m\omega x^2}{\hbar}} + C_0 e^{-\sigma t},$$
(33)

where C_0 is a constant of integration. Coming to equation (8) for neuronal response due to quantum effects, equation (30) will be plugged into this equation to give,

$$\frac{\partial |\Psi|^2}{\partial t} + \frac{\partial (|\Psi|^2 (v_0 + \gamma |\Psi|^2))}{\partial x} = 0.$$
(34)

Analytical solution of this equation can be found using method of characteristics.

Coming to equation (6) for evolution of synaptic potential, if f(n) were linear, say $f(n) = \kappa n$, then plugging equation (33) into this equation gives,

$$\frac{\partial V}{\partial t} = -\gamma \kappa n + \lambda \left(\frac{\theta}{\sigma} \left(\frac{m\omega}{\pi \hbar} \right)^{1/2} e^{\frac{-m\omega x^2}{\hbar}} + C_0 e^{-\sigma t} \right).$$
(35)

Using separation of variables and the boundary condition (17), the solution of this equation is given as,

$$V = -\gamma \kappa nt + \lambda t \frac{\theta}{\sigma} \left(\frac{m\omega}{\pi\hbar}\right)^{\frac{1}{2}} e^{\frac{-m\omega x^2}{\hbar}} - \frac{c_0 \lambda}{\sigma} e^{-\sigma t}.$$
(36)

Numerical Solution of the Problem:

Assume hypothetical values based on atomic scales (just as a point of reference) given as, \hbar (reduced Planck's constant) = 1.054571 ×

 10^{-34} J.s

m (mass of the particle, similar to an electron) = 9.11×10^{-31} kg

 ω (frequency of the oscillator) = 1 × 10¹⁵ Hz

L (length of the domain) = 1×10^{-9} m (1 nanometer)

 Δx (spatial step size) = 1 × 10⁻¹¹ m

 Δt (time step size) = 1 × 10⁻¹⁷ s

V(x) (assume a sinusoidal potential) = $V_0 \sin(kx)$, where $V_0 = 1.0$ eV is the amplitude and $k = 2\pi/0.2$ is the wave number.

V (Neuronal flow velocity vector) = $v_0 \hat{x}$ having a fixed magnitude and direction where $v_0 = 0.01$ units/s.

$$\begin{array}{ll} S(x,t) & (\text{Source/sink} & \text{term}) & = \\ \left\{ S_0 exp\left(-\frac{(x-x_c)^2}{2\sigma_s^2} \right), \ t \in [0.1, 0.2] \\ 0, \ otherwise \end{array} \right\}, \text{ assuming it is}$$

a localized source at the centre of problem domain that acts at a specific time interval. Also, assume that $S_0 = 0.05$ units/s, $x_c = 0.5$ units (center of domain), $\sigma_S = 0.05$ units.

D (Diffusion constant, which gives an idea about the rate of diffusion) = 0.01 units²/s.

 β (Influence coefficient of quantum waves on the firing rate) = 0.03 s⁻¹

 α (Natural decay rate of firing rate) = 0.01 s⁻¹

 γ (Sensitivity of the potential barrier to the neuronal firing rate) = 0.05 s⁻¹

 λ (Scaling factor for the restorative function r) = 0.02 s⁻¹

f(n) (For simplicity, assume a linear dependence of the firing rate on neuronal density n) = $f_0 + \mu n$, where $f_0 = 0.1$ s⁻¹ (base firing rate) and $\mu = 0.04$ s/neuron.

r (Restorative function that tends to bring the potential back to a baseline. Assume a simple linear form) = $r_0 - v V$, where $r_0 = 0.03 \text{ s}^{-1}$ and $v = 0.01 \text{ s}^{-1}$.

 θ (Coefficient determining the impact of quantum wave function $|\Psi|^2$ on the neuronal response r) = 0.07 s⁻¹

 σ (Decay rate or dissipation of the neuronal response *r* in the absence of any quantum influence) = 0.02 s⁻¹

v (Particle velocity. Assume v is uniform along x direction) = 0.02 m/s

 κ (Constant rate of stimulus introduction) = 0.06 units/s

 η (Decay rate of stimulus due to neuron density) = 0.02 units/neurons

C (Connectivity constant that defines how strong the influence of connectivity is on the wave function) = 0.04 units/s.

Numerical Discussion

Numerical solution of equations (1) to (10) comprises of following steps,

- Spatial Discretization: Divide the spatial domain wherever applicable, into grid points $x_i = i\Delta x$ with *i* running from 0 to *N* (assuming N = 100and $\Delta x = 0.01$ units.
- Time Discretization: Choose a time-step $\Delta t =$ 0.0001 s
- Initialization:

For equation (1): Set the initial values of Ψ at all grid points, say a Gaussian wave packet, $\Psi(x, 0) =$

$$A \exp\left(-\frac{(x-x_0)}{2\sigma^2}\right),$$

Where A is normalization constant, x_0 is the packet's initial position, and σ is width. Assume $x_0 = 0.5, \sigma = 0.1.$

For equations (2), (3), and (4): Set initial values for *n* at all grid points given as,

For equation (2): Assume a uniform distribution $n_i^0 = 0.1 \forall i.$

For equation (3): assume a Gaussian distribution centred at the midpoint $n_i^0 = exp\left(-\frac{(x_i-0.5)^2}{2\sigma^2}\right)$, where $\sigma = 0.1$ units is the standard deviation.

For equation (4): Assume a uniform distribution $n_i^0 = 0.5 \forall i.$

For equation (5): Assume the magnitude squared of the quantum wave function changes sinusoidally over the spatial domain (this is a simplification to avoid dealing with equation (8)), $|\Psi(x)|^2 =$ $A\sin(kx)$, where A (Amplitude) = 0.5, k (Wave number) = $2\pi/0.1$ (corresponding to a wave length of 0.1 units). For initial values for f at all grid points, assume $f_i^0 = 0.3$ uniformly for simplicity. For equation (6): Set the initial value for V, say $V^0 = 1.0$ as an initial potential barrier value.

For equation (7): Assume a sinusoidal variation in magnitude squared of the quantum wave function, $|\Psi(x,t)|^2 = A\sin(kx + \omega t),$ where Α (Amplitude) = 0.5, k (Wave number) = $2\pi/0.1$ (corresponding to a wave length of 0.1 units), ω (Angular frequency) = $2\pi \times 2$ rad/s (2 Hz frequency). Set the initial value for r, say $r^0 = 0.4$ as the initial neuronal response.

For equation (8): Assume a Gaussian distribution for initial values of magnitude squared of the quantum wave function, $|\Psi(x,0)|^2 =$ $A \exp\left(-\frac{x^2}{2\sigma_x^2}\right)$, where A (Amplitude) = 0.5, σ_x (Standard deviation) = 0.1 units. Set the initial values of $|\Psi|^2$ at all grid points using the Gaussian distribution.

For equation (9): Assume a Gaussian distribution for the neuron density as a function of position, $n(x) = n_{max} \exp\left(-\frac{(x-x_c)^2}{2\sigma^2}\right),$ where n_{max} (Maximum neuron density) = 0.25 neurons/unit length, x_c (Centre of neuron distribution) = 0.5 units (middle of the domain), σ (Speed of the neuron distribution) = 0.15 units. Set initial values for S at all grid points, say $S_i^0 = 0.1 \forall i$ from 1 to 99. Boundaries are $S_{0,0} = S_{100,0} = 0$. For equation (10): Set initial values for Ψ at all grid

points, say $\Psi_i^0 = 0.2 \forall i$ from 1 to 99. The boundaries are $\Psi_{0,0} = \Psi_{100,0} = 0$ (Dirichlet boundary conditions).

• Time Evolution: For each time step *m* from 0 to a predefined maximum, say 1000 steps,

For equation (1):
$$\Psi_{i}^{m+1} = \Psi_{i}^{m} - \frac{i\hbar\Delta t}{2m} \frac{\Psi_{i+1}^{m} - 2\Psi_{i}^{m} + \Psi_{i-1}^{m}}{\Delta x^{2}} + i\Delta t V_{i}\Psi_{i}^{m}$$
For equation (2):
$$n_{i}^{m+1} = n_{i}^{m} + \Delta t \left(-v_{0} \frac{n_{i+1}^{m} - n_{i-1}^{m}}{2\Delta x} + S(x_{i}, t_{m})\right)$$
(38)

For equation (3): For each spatial point x_i , except boundaries,

$$n_i^{m+1} = n_i^m + D\Delta t \ \frac{n_{i+1}^m - 2n_i^m + n_{i-1}^m}{\Delta x^2}$$
(39)

For equation (4): For each spatial point x_i , $n_i^{m+1} = n_i^m + C n_i^m \Delta t$

For equation (5): For each spatial point x_i , $f_i^{m+1} = f_i^m + \Delta t \ (\beta \ A \sin(2\pi x_i/0.1)) - \alpha \ f_i^m$ (41)

For equation (6): $V^{m+1} = V^m - \gamma (f_0 + \mu n) \Delta t + \lambda (r_0 - \nu V^m) \Delta t$ (42) Here, n is the neuron density at time t_m , which

would be determined by equation (38).

For equation (7): $r^{m+1} = r^m + \Delta t \ (\theta A \sin(2\pi \times$ $0.1 x + 2\pi \times 2 t^m) - \sigma r^m)$ (43)Here, x is a position variable, implying that the quantum wave function $|\Psi(x,t)|^2$ varies with position and time. If the complete domain is considered, then an average or specific location may be chosen for x in the simulations.

For equation (8): For each spatial point x_i , update $|\Psi|^2$ using,

$$\left|\Psi_{i}^{m+1}\right|^{2} = |\Psi_{i}^{m}|^{2} - \Delta t \left(\frac{\left(|\Psi_{i+1}^{m}|^{2} - |\Psi_{i-1}^{m}|^{2}\right)v}{2\Delta x}\right)$$
(44)

For equation (9): For each spatial point x_i , the updated value of S will be computed using the forward Euler method, $S_i^{n+1} = S_i^n + \Delta t \left(\kappa - \eta n(x_i)\right)$, i.e., $S_i^{n+1} = S_i^n + 0.001 \left(0.06 - 0.02 \times 10^{-10}\right)$

$$0.25 \exp\left(-\frac{(x_i - 0.5)^2}{2 \times 0.15^2}\right)$$
(45)

For equation (10): For each spatial point x_i and time step t_m , the updated value of Ψ will be computed using the forward Euler method, $\Psi_i^{n+1} = \Psi_i^n + \Delta t C \Psi_i^n$, i.e., $\Psi_i^{n+1} = \Psi_i^n (1 + C \Delta t)$

(46)

This equation indicates that the growth or decay of the wave function Ψ at each point is proportional to its current value.

• Resulting Matrix/Vector:

For equation (1): After iterating, a matrix $\Psi_{i,m}$ is obtained that describes the wave function Ψ evolution at spatial points x_i for various time steps t_m , given as,

$$\Psi_{i,m} = \begin{bmatrix} \Psi_0^0 & \Psi_0^1 & \cdots & \Psi_0^{1000} \\ \Psi_1^0 & \Psi_1^1 & \cdots & \Psi_1^{1000} \\ \vdots & \vdots & \ddots & \vdots \\ \Psi_{100}^0 & \Psi_{100}^1 & \cdots & \Psi_{100}^{1000} \end{bmatrix}$$
(47)

Each row captures the temporal progression of Ψ at a specific spatial point. Every column represents the spatial distribution of Ψ at a particular moment. This matrix depicts the dynamics of the wave function in the synaptic environment under both kinetic and potential influences.

For equations (2), (3), and (4): Using boundary condition (16), a matrix $n_{i,m}$ is obtained representing neuron densities for equation (2), particle densities for equation (3), and the evolution of neuronal densities for equation (4) at different spatial locations x_i for various time steps t_m , given as,

$$\mathbf{n}_{i,m} = \begin{bmatrix} \mathbf{n}_0^0 & \mathbf{n}_0^1 & \cdots & \mathbf{n}_0^{1000} \\ \mathbf{n}_1^0 & \mathbf{n}_1^1 & \cdots & \mathbf{n}_1^{1000} \\ \vdots & \vdots & \ddots & \vdots \\ \mathbf{n}_{100}^0 & \mathbf{n}_{100}^1 & \cdots & \mathbf{n}_{100}^{1000} \end{bmatrix}$$
(48)

The matrix shows how the neuron density evolves due to neuronal flow and the external source term S for equation (2), how the particle density disperses over time due to quantum diffusion within the synaptic clefts for equation (3), and how neuronal density grows (due to chosen positive value of C) over time influenced by the connectivity factor for equation (4).

For equation (5): After iterating, a matrix $f_{i,m}$ will be obtained that depicts the evolution of firing rates f at special points x_i for various time steps t_m , given as,

$$f_{i,m} = \begin{bmatrix} f_0^0 & f_0^1 & \cdots & f_0^{1000} \\ f_1^0 & f_1^1 & \cdots & f_1^{1000} \\ \vdots & \vdots & \ddots & \vdots \\ f_{100}^0 & f_{100}^1 & \cdots & f_{100}^{1000} \end{bmatrix}$$
(49)

The matrix showcases the dynamics of neuronal firing rates under the combined influence of a quantum wave function and natural decay.

For equation (6): The time evolution for this is without explicit spatial dependency, so a vector V_m is obtained that depicts the evolution of the synaptic potential barrier V over the time steps t_m , given as,

$$V_m = \begin{bmatrix} V^0 \\ V^1 \\ \vdots \\ V^{1000} \end{bmatrix}$$

(50)

Each entry in this vector captures the value of the synaptic potential barrier V at a specific time step. This vector showcases the dynamics of the synaptic potential under the influence of the firing rate of the neurons and the restorative function r.

For equation (7): The time evolution for this is also without explicit spatial dependency, so a vector r_m is obtained that showcases the evolution of the neuronal response r over the time steps t_m , given as,

$$r_m = \begin{bmatrix} r^0 \\ r^1 \\ \vdots \\ r^{1000} \end{bmatrix}$$
(51)

This vector portrays the dynamics of the neuronal response under the combined influence of the quantum wave function and its natural decay rate.

For equation (8): Post iteration, a matrix $|\Psi_{i,m}|^2$ will be obtained representing the density evolution of quantum particles at spatial points x_i for various time steps t_m , given as,

$$\left|\Psi_{i,m}\right|^{2} = \begin{bmatrix} \left|\Psi_{0}^{0}\right|^{2} & \left|\Psi_{0}^{1}\right|^{2} & \cdots & \left|\Psi_{0}^{1000}\right|^{2} \\ \left|\Psi_{1}^{0}\right|^{2} & \left|\Psi_{1}^{1}\right|^{2} & \cdots & \left|\Psi_{1}^{1000}\right|^{2} \\ \vdots & \vdots & \ddots & \vdots \\ \left|\Psi_{100}^{0}\right|^{2} & \left|\Psi_{100}^{1}\right|^{2} & \cdots & \left|\Psi_{1000}^{1000}\right|^{2} \end{bmatrix}$$

$$(52)$$

Each matrix corresponds to a time snapshot, giving a 2D tensor or a stack of matrices representing the evolution of $|\Psi|^2$ over time.

For equation (9): After iterating over the time steps, a matrix $S_{i,m}$ of dimension (101×1001) will be obtained representing stimulus values *S* at

different spatial locations x_i for various time steps t_m , given as,

$$\begin{aligned} S_{i,m} &= & 0 & 0 & 0 \\ 0.1 + 0.001 \left(0.06 - 0.02 \times 0.25 \exp\left(-\frac{(x_1 - 0.5)^2}{2 \times 0.15^2}\right) \right) & \dots & S_1^{1000} \\ &\vdots & \vdots & \vdots \\ 0.1 + 0.001 \left(0.06 - 0.02 \times 0.25 \exp\left(-\frac{(x_{99} - 0.5)^2}{2 \times 0.15^2}\right) \right) & \dots & S_{99}^{1000} \\ & 0 & 0 & 0 \end{aligned}$$

Each row of this matrix provides the temporal evolution of stimulus *S* at a particular spatial point due to the constant rate of stimulus introduction κ and the decay caused by the neuron density *n*. The matrix reveals how the external stimulus changes under the influence of neuron density over time.

For equation (10): After iterating through all time steps, a matrix or 2D array $\Psi_{i,m}$ will be obtained representing Ψ values at different spatial locations x_i for various time steps t_m , given as,

$$\Psi_{i,m} = \begin{bmatrix} 0 & 0 & 0 \\ 0.2(1+0.04 \times 0.001) & \dots & \Psi_1^{1000} \\ \vdots & \vdots & \vdots \\ 0.2(1+0.04 \times 0.001) & \dots & \Psi_{99}^{1000} \\ 0 & 0 & 0 \end{bmatrix}$$
(54)

This matrix depicts the exponential growth of Ψ over time across the spatial domain.

Conclusion

The study centres around the dynamic evolution of the quantum wave function, which encapsulates the synaptic firing probabilities in the network. The fundamental equation guiding this evolution is Schrödinger's equation governing the quantum states and energy levels within the neural network, exerting a profound influence on neuronal firing rates. Notably, the neuronal density, quantum diffusion, and network connectivity emerge as critical factors. Neuronal density impacts the concentration of the quantum wave function, shaping the probability distribution of synaptic firing. Quantum diffusion dictates the rate at which quantum wave spreads through the network, with denser networks exhibiting faster dispersion. Connectivity, as embodied by the matrix, modulates synaptic strengths, affecting signal propagation and altering the quantum wave's behaviour. Furthermore, our examination accounts for external stimuli interacting with the quantum perturbing neuronal wave function, firing probabilities in response to specific inputs. The numerical simulations, conducted with specified parameters, have unveiled intricate interplays between these variables. This study offers a foundation for further research into the quantum underpinnings of brain function.

References

- 1. Schrödinger, E., 1944, "What is Life?," Cambridge University Press.
- http://www.spaz.org/~jake/pix/schrodinger.pdf 2. Hameroff, S., and Penrose, R., 2014, "Consciousness in the universe: A review of the 'Orch OR' theory," Physics of Life Reviews, 11(1), pp. 39-78.

https://doi.org/10.1016/j.plrev.2013.08.002

 Frohlich, H., 1968, "Long-range coherence and energy storage in biological systems," International Journal of Quantum Chemistry, 2(5), pp. 641-649.

https://doi.org/10.1002/qua.560020505

4. Beck, F., and Eccles, J., 1992, "Quantum aspects of brain activity and the role of consciousness," Proceedings of the National Academy of Sciences, 89(23), pp. 11357-11361.

https://doi.org/10.1073/pnas.89.23.11357

 Eccles, J. C., Beck, F., and Eccles, J. C., 1994, "Quantum aspects of brain activity and the role of consciousness," How the SELF Controls Its BRAIN, pp. 145-165.

https://doi.org/10.1007/978-3-642-49224-2_9

- 6. Tegmark, M., 2000, "The importance of quantum decoherence in brain processes," Physical Review E, 61(4), pp. 4194. https://doi.org/10.1103/PhysRevE.61.4194
- Engel G. S., Calhoun, T. R., Read, E. L., Ahn, T. K., Mančal, T., Cheng, Y. C., Blankenship, R. E., and Fleming, G. R., 2007, "Evidence for wavelike energy transfer through quantum coherence in photosynthetic systems," Nature, 446, pp. 782–786. https://doi.org/10.1038/nature05678

 Ritz, T., Adem, S., and Schulten, K., 2000, "A model for photoreceptor-based magnetoreception in birds," Biophysical Journal, 78(2), pp. 707-718. https://doi.org/10.1016/S0006-3495(00)76629-X

- 9. Arndt, M., and Hornberger, K., 2014, "Testing the limits of quantum mechanical superpositions," Nature Physics, 10(4), pp. 271-277. https://doi.org/10.1038/nphys2863
- 10.Koch, C., and Hepp, K., 2006, "Quantum mechanics in the brain," Nature, 440(7084), pp. 611-611. https://doi.org/10.1038/440611a
- 11.Busemeyer, J. R., and Bruza, P. D., 2012, "Quantum models of cognition and decision," Cambridge University Press. https://doi.org/10.1017/CBO9780511997716
- 12. Hore, P. J., and Mouritsen, H., 2016, "The Radical-Pair Mechanism of Magneto-reception," Annual Review of Biophysics, 45 (1), pp. 299-344.

https://doi.org/10.1146/annurev-biophys-032116-094545

13.Michalet, X., Pinaud, F. F., Bentolila, L. A., Tsay, J. M., Doose, S. J. J. L., Li, J. J., Sundaresan, G., Wu, A. M., Gambhir, S. S., and Weiss, S., 2005, "Quantum dots for live cells, in vivo imaging, and diagnostics," Science, 307(5709), pp. 538-544.

https://doi.org/10.1126/science.1104274

- 14.Busemeyer, J. R., Wang, Z., and Townsend, J. T., 2006, "Quantum dynamics of human decision-making," Journal of Mathematical Psychology, 50(3), pp. 220-241. https://doi.org/10.1016/j.jmp.2006.01.003
- 15. Khrennikov, A., 2011, "Quantum-like model of processing of information in the brain based on classical electromagnetic field," Biosystems, 105(3), pp. 250-262.
 https://doi.org/10.1016/i.biosystems.2011.05.0

https://doi.org/10.1016/j.biosystems.2011.05.0 14

16.Abbott, D., Davies, P. C., and Pati, A. K., (Eds.). 2008, "Quantum aspects of life," World Scientific.

http://www.esalq.usp.br/lepse/imgs/conteudo/ Quantum-Aspects-Of-Life.pdf

- 17. Atmanspacher, H., 2015, "Quantum approaches to consciousness," The Stanford Encyclopedia of Philosophy. https://plato.stanford.edu/Entries/qtconsciousness/
- 18. Atmanspacher, H., 2004, "Quantum approaches to consciousness," Stanford Encyclopedia of Philosophy. https://plato.stanford.edu/Entries/qtconsciousness/
- Lloyd, S., 2011, :Quantum coherence in biological systems," Journal of Physics: Conference Series, 302(1), pp. 012037.

https://iopscience.iop.org/article/10.1088/1742 -6596/302/1/012037/pdf;Quantum

20.Hertäg, L., Durstewitz, D., and Brunel, N., 2014, "Analytical approximations of the firing rate of an adaptive exponential integrate-andfire neuron in the presence of synaptic noise," Front. Comput. Neurosci., 8, pp. 116. https://doi.org/10.3389/fncom.2014.00116