Exploration

A Hypothesis about the Origin of Alzheimer's Disease in the Orch OR Theory

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Abstract

In the orchestrated objective reduction (Orch OR) theory of consciousness, information in the brain is proposed to be stored via quantum entanglement between fundamental units such as electron spins, electromagnetic fields, and the polarized molecules comprising neuronal microtubules. As an individual ages, environmental perturbations are hypothesized to progressively diminish the degree of quantum entanglement in these microtubules, thereby disrupting spin alignments and molecular polarizations. The resulting loss of quantum-coherent information is posited to manifest as memory loss and the cognitive decline characteristic of Alzheimer's disease (AD). Importantly, this hypothesis also accounts for epidemiological observations that lifelong mental stimulation and social engagement may delay AD onset. Individuals with active thought processes and frequent neuronal signaling—such as those with rich social interactions or intellectual responsibilities—can sustain the energy levels required to maintain molecular polarization and entanglement, thereby preserving stored information. In contrast, prolonged inactivity or isolation may fail to provide sufficient stimulation to counteract entanglement decay. We present a theoretical framework and mathematical model, grounded in Orch OR, that links the loss of quantum entanglement in brain microtubules to the neuropathology of AD. The model demonstrates how entangled electron-spin states deteriorate over time and how elevated neuronal activity (modeled as electrical signals) can mitigate this deterioration. The results offer a quantum biological perspective on why active, socially-engaged lifestyles correlate with reduced AD risk, while advanced age and passive isolation correspond to increased vulnerability. This hypothesis article is structured as a scholarly exploration of the idea that Alzheimer's disease may originate from the gradual loss of quantum coherence (entanglement) in the brain's information processing microstructures, bridging quantum physics and neurobiology in the context of aging and dementia.

Keywords: Alzheimer's disease, origin, theory of consciousness, Orch OR, quantum bit.

1. Introduction

One of the leading models proposed to explain how information is encoded and stored in brain cells is the Orch OR theory (Hameroff, 2012; Hameroff & Penrose, 2014). In Orch OR, developed by Stuart Hameroff and Roger Penrose, microtubules within neurons serve as sites of quantum information processing. Each neuron contains a cytoskeletal network of microtubules,

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and each microtubule is composed of many tubulin protein molecules that can exist in polarized states. The orientation of these molecular dipoles, the spins of their constituent electrons, the intensity of local electric fields between polarized molecules, or the potential differences across microtubule subunits can all function as *quantum bits* (qubits) that store information.

In essence, microtubule polarization and spin alignment provide a substrate for a proposed quantum biomemory. If a subset of these qubits were to lose their ability to maintain information (for example, through decoherence or structural damage), the data encoded by those qubits would be lost, potentially manifesting as memory impairment. It has been hypothesized that when enough microtubule qubits fail, the cumulative information loss could lead to neurodegenerative conditions such as Alzheimer's disease. Empirical evidence linking such information loss to AD is an active area of exploration. For instance, biochemical studies of AD have documented the destabilization of cytoskeletal structures and synaptic loss consistent with the idea of lost "bits" of information (Breijyeh & Karaman, 2020; Waldemar et al., 2007; Shahwar et al., 2022).

In recent years, research on psychosocial factors has further illuminated the connection between cognitive decline and lifestyle. Several studies have examined the relationship between Alzheimer's disease progression and factors like social isolation and mental inactivity. Ali et al. (2017) demonstrated in a rodent model that prolonged complete social isolation can induce neurodegeneration in the brain, significantly elevating markers of AD pathology. Their findings suggest that isolation is a risk factor that, when combined with other AD mechanisms, increases neuronal DNA fragmentation and exacerbates disease severity. Wilson et al. (2007) reported that feelings of loneliness in older adults are associated with a higher risk of developing dementia, including AD, although loneliness itself may not directly target the classical neuropathological hallmarks of the disease.

In a comprehensive review and meta-analysis, Evans et al. (2019) found that various aspects of social isolation are correlated with diminished cognitive function in later life, reinforcing the idea that engagement in social and intellectual activities has protective effects on brain health. On the other hand, advancing age remains the predominant risk factor for sporadic AD: nearly half of individuals over 85 are affected (Drachman, 2006). Drachman (2006) described the aging brain in terms of increasing entropy, suggesting that the accumulation of disordered, lost information is central to late-life cognitive decline. Motivated by these findings, our work considers the interplay between aging, social/mental activity, and quantum information processes in the brain. In particular, we explore the hypothesis that Alzheimer's disease can be understood, in part, as a consequence of age-related loss of quantum entanglement in neuronal microtubules – a loss that may be offset by sustained cognitive engagement and neuroelectric activity.

2. Theoretical Background

According to Orch OR theory, consciousness and memory are supported by quantum processes inside neurons, especially within microtubules. Microtubules are hollow cylindrical polymers of the protein tubulin, and many tubulin molecules within a microtubule can become *polarized*, meaning they carry an electric dipole alignment. These polarized molecules can pair with neighbors to form bioelectric capacitors that store localized electric fields. The presence of a

stored field (and its magnitude and orientation) can serve as a bit of information, as can the binary orientation (up vs. down) of an electron or nuclear spin in the molecule. In other words, each tubulin's polarization state or associated electron spin state can act as a qubit encoding information (e.g., "0" or "1"). Multiple physical degrees of freedom—electric charge separation, field direction, spin orientation—offer a high-capacity information storage system in the quantum domain (Meijer et al., 2021; Beshkar, 2020, 2022). Entanglement between these quantum degrees of freedom allows for distributed or redundant storage of information across different parts of the microtubule or even across different neurons. This means that a single piece of information might be stored in the *correlations* between entangled spins or polarizations, not just in a single localized bit. The Orch OR model posits that brain information is encoded in such entangled states linking fields, electrons, and molecules within microtubules.

To illustrate how entanglement can amplify information capacity, consider a simple analogy using two-level spin systems. Suppose we want to store the state of a lamp (on or off) as a bit of information in a microtubule. One way is to assign an electron's spin orientation to represent the lamp's state: for example, spin "up" = lamp on, and spin "down" = lamp off. This single spin can encode the binary information. Now imagine we also want to record the color of the lamp (say red, green, or blue in addition to on/off). A single spin bit is insufficient for multiple states of color. However, if we use an *entangled state* of two electron spins, we can encode more information. If the two spins are in an **antisymmetric** (singlet) entangled state, we might designate that as "lamp off" regardless of color; if they are in a **symmetric** (triplet) entangled state, we designate "lamp on." Within the symmetric state, there are three independent basis states (triplet sub-states) which we can map to the three possible colors (e.g., red, green, blue).

In this way, the entangled two-spin system can store not just a single bit (on/off) but additional information (color) in its quantum state. Moreover, one could imagine using the relative angle between the spin orientations as another continuous parameter for encoding information. In principle, the angle between two spin vectors could vary from 0 to 360 degrees, which means an entangled state could encode up to 360 distinct levels of information—vastly more than the simple binary case. This toy model demonstrates how entanglement and quantum superposition can dramatically increase the information storage capacity: by some estimates, entanglement between spins, fields, and molecular polarizations in microtubules could scale up information content hundreds-fold compared to classical bits. Although the "360 times" figure is a heuristic example, it underscores the potential richness of quantum brain information encoding under Orch OR assumptions.

Beyond Orch OR, other theoretical frameworks also support the notion of distributed, non-local information storage in biological systems. The *Quantum Hologram Theory of Consciousness (QHTC)* is one such framework which posits that consciousness is not confined to a specific location in the brain, but is instead a field phenomenon, much like a hologram (Valverde et al., 2022).

In QHTC, the brain is likened to a holographic processor: it converts sensory inputs and internal states into interference patterns (quantum wave interference) and then reconstructs these as the "images" of conscious experience. These interference patterns (quantum wavefunctions) are capable of storing a vast amount of information in distributed form, analogous to how a hologram stores an image across an entire volume such that each piece of the hologram contains the whole image in encoded form. The theory suggests that the wavefunction of the brain itself

could be a field of consciousness, integrating information across the brain and even the body. According to QHTC, the information relevant to consciousness could be coded in the polarization or spin states of not only molecules and electrons, but even photons, and this information is continually exchanged between the brain and other parts of the body (for example, the heart) via electromagnetic waves and possibly quantum-correlated signals in the blood and nervous system.

While the specifics of QHTC are beyond the scope of this paper, it aligns with Orch OR in emphasizing non-local, wave-mediated information storage. Both theories allow for the possibility that memory and personality could be influenced by quantum states that extend beyond single neurons – an idea supported by intriguing observations such as personality changes following organ transplants, where the *information* carried by a donor organ might affect the recipient (Al-Karaki et al., 2024).

Given this background, let us consider what happens over time to these quantum information carriers. The entangled states and polarized structures in microtubules are presumably susceptible to environmental disturbances. Factors such as mechanical stress (e.g., pressure or shear forces in brain tissue), gravitational effects, thermal fluctuations, electromagnetic interference, and accumulations of electric charge can all act as *decoherence factors* that disrupt delicate quantum states. Over the lifespan of an individual, these environmental factors continuously impinge on neural tissue.

We hypothesize that as time passes, they gradually destroy the "bioelectric" structures – the polarization configurations and entangled spin states – that encode memory and cognitive information. In the quantum framework, this means that the fidelity of entanglement between microtubule components diminishes with age. Consequently, a significant portion of the information that was once stored in the brain's quantum state gets lost. When such loss reaches a critical threshold, the macroscopic symptoms of Alzheimer's disease, such as memory loss and cognitive dysfunction, become apparent. In summary, the theoretical background of our hypothesis is that AD can be viewed as a quantum information *degradation* problem: the breakdown of entangled molecular and spin states in the brain's microtubules erodes the stored information that underlies memory and identity. In the following sections, we introduce a mathematical model to formalize this concept and to explore how neuronal activity might counteract or accelerate this quantum information loss.

3. Mathematical Method

To quantitatively examine the loss of entanglement with time – and the potential mitigating effect of neural signaling – we construct a simplified quantum-mechanical model of two interacting molecules in the brain. Consider two molecules (for example, two tubulin dimers in a microtubule or two adjacent molecules within a neuron) that each contain an unpaired electron. We assume that one electron from the first molecule and one electron from the second molecule become *entangled* as a spin pair. According to the Pauli exclusion principle, if these two electrons form a paired system, their spins must be opposite (one spin-up, the other spin-down) when in the singlet state. We represent the general two-electron spin state as a superposition of one electron spin-up & the other spin-down, and vice versa:

$$F(S_1{:}S_2) \;=\; \cos(\alpha)\; S_1^{\uparrow}S_2^{\downarrow} \;+\; \sin(\alpha)\; S_1^{\downarrow}S_2^{\uparrow}$$

Equation 1

where F denotes the two-spin state function, S_1 and S_2 are the spin wavefunctions of electron 1 and 2 respectively, and the coefficients $\cos(\alpha)$ and $\sin(\alpha)$specify the quantum superposition amplitudes. The parameter α effectively determines the degree of entanglement or mixing between the two basis states ($S_1^{\uparrow}S_2^{\downarrow}$vs. $S_1^{\downarrow}S_2^{\uparrow}$). When $\alpha = 45°$(i.e. $\cos^2 \quad \alpha = \sin^2 \quad \alpha = 0.5$), the state is maximally entangled (equal superposition of the two spin configurations); when $\alpha = 0$ or 90 , the state collapses to a definite product state (completely unentangled). We further define $\sin(\alpha)$in a thermodynamic form suggestive of a Boltzmann factor:

$$\sin(\alpha) \;=\; \frac{e^{-E(S_1:S_2)/T_0(S_1:S_2)}}{1 + e^{-E(S_1:S_2)/T_0(S_1:S_2)}},$$

Equation 2

where $E(S_1{:}S_2)$ is the interaction energy of the two-spin system and T_0 is an effective temperature associated with the spin system. This form for $\sin(\alpha)$ensures that $0 \le \sin(\alpha) \le 1$and is reminiscent of the Fermi-Dirac distribution or logistic function, indicating that higher energy relative to temperature makes $\sin(\alpha)$ small (i.e., the spins are less likely to be aligned in the ↓↑configuration) . Intuitively, at very low temperature T_0 or very high energy cost E, the two spins will almost certainly be antiparallel ($\cos\alpha \approx 1\$,\ \sin\alpha \approx 0$), whereas at high temperature or low interaction energy the system can fluctuate into either configuration.

Given this definition, we can compute the probability that the two-spin system is found in the specific state $S_1^{\downarrow}S_2^{\uparrow}$ (one up, one down in the opposite order):

$$P\left(S_1^{\downarrow}{:}\,S_2^{\uparrow}\right) \;=\; \sin^2(\alpha)\,.$$

Equation 3

Substituting the expression above for $\sin(\alpha)$, this probability can be written explicitly as:

$$P\left(S_1^{\downarrow}{:}\,S_2^{\uparrow}\right) \;=\; \sin^2(\alpha) \;=\; \frac{e^{-2E(S_1:S_2)/T_0(S_1:S_2)}}{(1 + e^{-E(S_1:S_2)/T_0(S_1:S_2)})^2}.$$

Equation 4

This result shows that the entanglement-related probability (since $\sin^2\alpha$ quantifies the weight of the entangled part of the state) depends on the ratio of energy to temperature for the two-spin subsystem. A higher effective temperature T_0or lower interaction energy E makes the entangled configuration more probable, whereas a low temperature or high energy difference suppresses entanglement in favor of a definite spin order. In essence, thermal agitation can sustain or randomize spin orientations, supporting entangled superpositions, while energy biases can lock the system into a particular order (destroying equal superposition).

In addition to spin orientation, we must also consider the *existence* or *occupancy* of the particles themselves in their entangled state. Especially in a biological context, particles (such as electrons, or excitons, or other quasiparticles) might transiently exist or not exist in certain states due to interactions like annihilation, transfer, or delocalization. To incorporate this, we introduce another two-state quantum function G that represents the joint existence (Ex or non-existence (NonEx) of the entangled pair of particles (e.g., whether both electrons are present in the microtubule structure simultaneously or not). We write a similar superposition for the existence aspect:

$$\mathrm{G}(\mathrm{Ex}_1/\mathrm{NonEx}_1 : \mathrm{Ex}_2/\mathrm{NonEx}_2) = \cos(\Theta)\ (\mathrm{NonEx}_1\ \mathrm{NonEx}_2) + \sin(\Theta)\ (\mathrm{Ex}_1\ \mathrm{Ex}_2)$$

Equation 4

Here, $(\mathrm{NonEx}_1\ \mathrm{NonEx}_2)$is the state where neither particle exists (for instance, both electrons have left the entangled state or are absent), and $(\mathrm{Ex}_1\ \mathrm{Ex}_2)$ is the state where both particles exist (both electrons are present and available to be entangled). The coefficient $\sin(\Theta)$ (analogous to $\sin\alpha$ earlier) quantifies the amplitude for both particles existing together. We define this amplitude in a similar logistic form:

$$\sin(\Theta) = \frac{e^{-\mathrm{E}(\mathrm{Ex}_1:\mathrm{Ex}_2)/\mathrm{T}_1(\mathrm{Ex}_1:\mathrm{Ex}_2)}}{1 + e^{-\mathrm{E}(\mathrm{Ex}_1:\mathrm{Ex}_2)/\mathrm{T}_1(\mathrm{Ex}_1:\mathrm{Ex}_2)}}$$

Equation 5

where $\mathrm{E}(\mathrm{Ex}_1:\mathrm{Ex}_2)$is some energy associated with the existence of the two particles as a pair (for example, an energy penalty or binding energy required for both to simultaneously exist in the entangled state), and T_1 is an effective "existence" temperature that might differ from the spin temperature T_0. This form implies that if it is energetically unfavorable for both particles to exist together (large $\mathrm{E}(\mathrm{Ex}_1:\mathrm{Ex}_2)$) and the system is cold (T_1 low), then $\sin(\Theta)$ will be small (the particles won't co-exist). Conversely, at high T_1 or low energy requirement, simultaneous existence is likely.

The probability that both particles *exist* (as opposed to one or both being absent from the entangled state) is then:

$$\mathrm{P}(\mathrm{Ex}_1\mathrm{Ex}_2) = \sin^2(\Theta) = \frac{e^{-2\mathrm{E}(\mathrm{Ex}_1:\mathrm{Ex}_2)/\mathrm{T}_1(\mathrm{Ex}_1:\mathrm{Ex}_2)}}{(1 + e^{-\mathrm{E}(\mathrm{Ex}_1:\mathrm{Ex}_2)/\mathrm{T}_1(\mathrm{Ex}_1:\mathrm{Ex}_2)})^2}$$

Equation 6

This mirrors our earlier result for the spin state, now describing the occupancy aspect.

Now we combine the spin entanglement and the existence probability to get the *total* probability that we have an entangled two-particle state with one spin up and one spin down *and* both particles present. Assuming the spin orientation aspect and the existence aspect are independent (to first approximation), the joint probability will be the product of the two probabilities:

$$\begin{aligned}\mathrm{P(Two\ Entangled\ Spinors)} &= \mathrm{P}\left(\mathrm{S}_1^{\downarrow}:\mathrm{S}_2^{\uparrow}\right) \times \mathrm{P}(\mathrm{Ex}_1\mathrm{Ex}_2) \\ &= \sin^2(\alpha) \times \sin^2(\Theta) = e^{-2\left(\frac{E(S_1:S_2)}{T_0(S_1:S_2)}\right)}\left(1+e^{-\left(\frac{E(S_1:S_2)}{T_0(S_1:S_2)}\right)}\right)^{-2}\end{aligned}$$

Equation 7

This expression encapsulates the probability that a given pair of molecules is in the entangled state of interest, incorporating both the alignment of their spins and the co-existence of the particles. The dependence on two temperatures T_0 and T_1 reflects potentially different thermal influences on spin orientation and particle existence, respectively. Entanglement is favored by higher temperatures (which promote superposition and occupancy) and is suppressed by higher energies (which disfavor the needed configurations).

Extending this model to larger structures, we consider two entire molecules (each composed of many potential entangled pairs) – for instance, two microtubule segments – that share multiple entangled electron pairs. If each molecule has N such entangled spin pairs with the other, the overall probability that *all* these pairs maintain entanglement is the product of the probabilities for each entangled spin pair (assuming independence). Thus, if the entangled pairs are indexed as $1:2,3:4,\ldots,(\mathrm{N}-1):\mathrm{N}$, we can write the entanglement probability for the two molecules in energy-momentum space as:

$$\mathrm{P(Two\ Entangled\ Molecules\ in\ Energy\text{-}Momentum\ space)} = \prod_{\mathrm{k}=1}^{\mathrm{N}/2} \mathrm{P}\left(\mathrm{Two\ Entangled\ Spinors}_{\mathrm{k}}\right)$$

Equation 8

where each P("$\{\mathrm{Two\ Entangled\ Spinors}\}_{\{\mathrm{k}\}}$is given by an expression like the one above (with appropriate energies for each pair). This multiplication reflects that if any one entangled pair is lost, the two molecules are not fully entangled. In practice, entanglement across many pairs could degrade gradually – our model simplifies this as an all-or-nothing product for tractability.

So far, our probabilities are calculated in the energy-momentum domain (i.e., they depend on energy parameters E and temperatures T but are not explicitly time-dependent). To understand how entanglement changes *over time*, we perform a Fourier transform to move from the energy domain into the time domain. In quantum mechanics, transitioning from energy E to time t can be done by an inverse Fourier transform kernel $\mathrm{e}^{\mathrm{iEt}/\hbar}$ (with $\hbar$ set to 1 in natural units). For our purposes, we integrate over all the energy variables associated with the entangled pairs, introducing time dependence for each via complex exponentials. The probability amplitude in the time domain is obtained by:

$$\begin{aligned}&\mathrm{P(Two\ Entangled\ Molecules\ in\ Space\text{-}Time)} \\ &= \int \left(\prod_{\mathrm{k}} \mathrm{dE}_{\mathrm{k}}\right) \mathrm{P(Two\ Entangled\ Molecules\ in\ E\text{-}M)} \times \left(\prod_{\mathrm{k}} \mathrm{e}^{\,\mathrm{iE_k\,t_k}}\right),\end{aligned}$$

Equation 9

where k indexes each entangled component (spin pairs and existence degrees of freedom). In this schematic formula, each E_k corresponds to an energy like $E(S_i{:}\,S_j)$ or $E(Ex_p{:}\,Ex_q)$ for a particular entangled pair, and t_k is the corresponding time variable conjugate to that energy. The product of exponentials $\prod e^{iE_k t_k}$ represents the Fourier kernel (with $i = \sqrt{-1}$) that introduces time into the probability, and $\prod dE_k$ indicates integration over all those energy variables. In effect, we superpose contributions from all possible energy values to see how the entanglement probability evolves as a function of time. This multiple integration yields a function R that depends on time *inversely*, as well as on the temperature parameters:

$$P(\text{Two Entangled Molecules in Space-Time}) = R\backslash(t^{-1}S1{:}\,S2,\ t^{-1},\ \ldots;\ T_0(S1{:}\,S2),\ \ldots, T_1(Ex1{:}\,Ex2),\ \ldots),$$

Equation 10

where R is some resultant function of the inverse times $1/t$ (for each entangled component) and the various T_0, T_1 values. While the exact form of R is complicated, we can qualitatively analyze its implications. Crucially, the dependence on t^{-1} means that as *time t progresses (increases), the argument $\frac{1}{t}$ decreases*, often driving P downwards. Indeed, the model indicates that as $t \to \infty$, the entanglement probability P tends toward zero: in other words, *with the passage of time, the entanglement between two molecules reduces and eventually shrinks to zero*. This result formalizes the intuitive idea that quantum entangled states in the brain are not perfectly preserved forever; they decay or decohere over time, leading to loss of the information those states carry. In a biological system at finite temperature, this decay might be rapid unless counteracted by some mechanism. The function R also explicitly includes the temperatures T_0, T_1, confirming that the rate of entanglement loss depends on the thermal environment: higher effective temperatures might slow the decay by providing energy to sustain entanglement, whereas lower temperatures might preserve coherence in some models, but here lower T actually made certain entangled configurations less probable in the first place. In summary, the mathematical model predicts an inexorable, time-dependent decline of entanglement between microtubule molecules, modulated by thermal factors.

The next step is to link this decay to physiological conditions in the brain. In particular, we consider how brain *activity* – in the form of electrical signals and currents – might influence the effective temperature parameters or directly enter the expression for P. Neurons communicate via electrical impulses (action potentials) and chemical signals, which create time-varying electric fields and currents in brain tissue. These electromagnetic effects could impact the quantum states of microtubule components. In our model, one way to incorporate this is to allow the temperature terms (especially T_1, the "existence temperature") to be functions of neuronal electrical activity. Following Sepehri et al. (the authors of our source hypothesis), we introduce an empirical relation between the temperature T_1 and the electrical current in neurons:

$$T_1 = k^{\{-1\}E(current)} = k^{\{-1\}(\{\{\text{Voltage}\}\times\{\text{signal}\}\}\times t)},$$

Equation 11

where $\mathrm{E(current)}$is an energy associated with the neural current (for example, the work done by a voltage source moving charge), expressed here in terms of the product of voltage and some measure of signal frequency, divided by current and multiplied by time t. The constant $\mathrm{k}^{\{-1\}}$ is a proportionality factor translating electrical units into temperature units. While this relation is heuristic, the idea is that active neuronal firing (high-frequency signals, higher voltages across membranes) can effectively raise the local "temperature" in the microtubule environment – not strictly the thermodynamic temperature, but the parameter $\mathrm{T_1}$governing occupancy of quantum states. In other words, vigorous brain activity can inject energy into the microtubule system, potentially sustaining entangled states that would otherwise collapse.

Substituting this relation back into the probability, we can rewrite the entanglement probability in space-time to explicitly include the influence of neural signals:

$$P(\{Two\ entangled\ molecules\}\{Space{:}Time\})$$

$$= R($$

$$t^{-1(S_1:S_2)} \dots$$

$$t^{-1(Ex_1:Ex_2)} \dots$$

$$t^{-1(S_{N-1}:S_N)} \dots$$

$$t^{-1(Ex_{N-1}:Ex_N)} \dots$$

$$\frac{Voltage \cdot Signal}{(Current \cdot t)(S_1:S_2)} \dots$$

$$\frac{Voltage \cdot Signal}{(Current \cdot t)(Ex_1:Ex_2)} \dots$$

$$\frac{Voltage \cdot Signal}{(Current \cdot t)(S_{N-1}:S_N)} \dots$$

$$\frac{Voltage \cdot Signal}{(Current \cdot t)(Ex_{N-1}:Ex_N)}$$

$$)$$

Equation 12

where for brevity we have not written out all arguments; the key change is that each occurrence of $\mathrm{T_1}$ in R is replaced by an expression proportional to $\frac{Voltage \cdot Signal}{(Current \cdot t)}$ The effect of this substitution is significant: the factors of $\mathrm{t}^{\{-1\}}$ (which caused decay with time) may be partly canceled or mitigated by factors of t introduced via the $\mathrm{T_1}$ substitution. **In qualitative terms, the model now indicates that the presence of an electrical signal (voltage pulses and currents from neural activity) reduces the effective role of time in diminishing entanglement, thereby increasing or sustaining the strength of entanglement between molecules.** In short, ongoing neural signaling can slow down the entanglement decay or even temporarily boost the

entanglement probability, whereas in a quiescent brain with little electrical activity, entanglement would fade more rapidly. This theoretical outcome aligns with the hypothesis that mental activity (neuronal firing and information processing) helps preserve the quantum coherence that encodes memory and cognition, thus protecting against Alzheimer's-type information loss.

4. Results

The above theoretical model leads to several qualitative results that resonate with epidemiological and clinical observations in Alzheimer's disease. **First, it suggests that individuals who remain mentally active and socially engaged should experience a slower loss of quantum information in their brains, translating to a later onset or reduced risk of AD.** In our model, frequent neuronal firing (stimulated by continuous cognitive and social interaction) provides a kind of "quantum support" for microtubule entanglement, maintaining molecular polarization and spin coherence despite the passage of time. This finding is consistent with real-world patterns: active people who are regularly challenged by intellectual tasks or who interact with society (for instance, through work, family, or community engagement) are empirically less prone to develop Alzheimer's disease than those who lead isolated or unengaged lives. Such active and responsive individuals are constantly thinking, which in neural terms means their brains are constantly exchanging electrical signals between neurons.

Our model indicates that these signals help preserve the polarization of microtubule molecules – in effect keeping the "memory qubits" energized and entangled. Polarized, entangled molecules are better able to store information and contribute to conscious processes, so the cognitive function of active individuals remains intact longer. Epidemiological studies indeed show that higher cognitive reserve, often built by education, complex occupation, and social engagement, correlates with a delayed manifestation of dementia symptoms. The theoretical underpinning provided by our model gives a quantum-biological explanation for this: mental activity fights entropy in the brain's quantum information structure.

Second, the model reinforces that increasing age inherently brings a greater risk of Alzheimer's disease due to the cumulative decoherence of microtubule qubits over time. Even in the absence of other risk factors, time is detrimental to quantum coherence. As the model showed, as t increases, $P_{\{\{\text{entanglement}\}\}}$ tends to shrink unless counteracted by injected energy. With advancing age, molecules gradually lose their polarization ability; the entanglement between spins in the brain's microtubules correspondingly diminishes. In quantum terms, fewer qubits remain entangled and thus fewer are effective in storing information. From a neurological perspective, this means that some of the information held in the brain is lost over time – a process that would first affect short-term memory and gradually long-term memory, as observed in early AD. Our model thus provides a potential explanation for why the incidence of Alzheimer's disease and related dementias climbs steeply in old age.

The probability of memory-loss events increases as the quantum information substrates age and decay. Moreover, the model aligns with clinical observations that AD involves a breakdown of microtubule and cytoskeletal structure (for example, the formation of neurofibrillary tangles of tau protein destabilizes microtubules). In our framework, such structural breakdown could be

both a cause and a consequence of lost polarization/entanglement: once microtubules can no longer support entangled states, they may disintegrate, and conversely, if they disintegrate due to tau pathology, they can no longer support entanglement, accelerating information loss.

These results together articulate the central hypothesis: **Alzheimer's disease can be seen as a progressive decoherence of the brain's quantum information state, and active mental life is protective because it continually "re-entangles" the brain's qubits, whereas advanced age without cognitive engagement leads to unchecked entanglement loss**. In the next section, we discuss the broader implications of this hypothesis and how it converges with existing knowledge about AD.

5. Conclusion

In summary, the Orch OR model of quantum brain dynamics offers a novel lens through which to view the origin of Alzheimer's disease. According to this model, mental information is encoded not only in classical synaptic connections but also in the form of electromagnetic fields and quantum states (spin orientations, polarization states) that are *entangled* across the microtubule networks within neurons. Over time, however, various environmental and intrinsic factors cause a degradation of these delicate quantum-coherent structures. As the polarizable molecules and spin-based qubits in the microtubules lose their entanglement, the information that was stored in those quantum correlations is partially erased. We propose that when this loss reaches a critical level, the clinical symptoms of Alzheimer's disease emerge – essentially as a macroscopic manifestation of quantum information loss in the brain's neural architecture. This perspective complements classical accounts of AD (which focus on plaques, tangles, and neuronal death) by suggesting that *information* itself is a quantity that can be lost from the brain independent of overt cell death, through the subtle unraveling of quantum states. It also echoes the idea of the aging brain as an increase in entropy or information randomness (Drachman, 2006) – here cast specifically as loss of entangled order.

Our entanglement-based model also provides a coherent explanation for why an active, intellectually engaged lifestyle is associated with resilience to Alzheimer's disease. The theoretical analysis showed that continuous neural signaling – which corresponds to cognitive activity and sensory engagement – can counteract the time-driven decay of entanglement. In an active brain, synaptic firing and oscillatory electrical activity supply the microtubules with renewed energy (conceptualized via the T_1 term related to voltage and current), thereby maintaining molecular polarization and protecting entangled states from dissipating. Consequently, individuals who routinely challenge their brains (through learning, problem-solving, social interaction, etc.) essentially keep "recharging" their quantum bits, delaying the point at which information loss causes cognitive impairment.

This model is consistent with observational studies: for example, epidemiologists have noted that professionals who remain engaged in mentally demanding work or hobbies into old age—such as scientists, physicians, or musicians—tend to have a lower incidence of AD or a later onset, compared to less stimulated individuals. Likewise, elders in enriched, socially interactive environments (e.g. living with family or in vibrant communities) fare better cognitively than those in understimulating settings (such as isolating nursing homes). Our quantum perspective

suggests that the brains of active individuals maintain better microtubule coherence, whereas those of isolated individuals succumb faster to entanglement erosion.

Finally, the hypothesis outlined in this paper invites a fusion of ideas from quantum physics and neuroscience: it suggests that preventing or treating Alzheimer's disease might involve not just classical interventions (like reducing amyloid or tau pathology) but also stabilizing the brain's quantum states. While direct experimental evidence for quantum processes in cognition remains a subject of debate, this framework encourages interdisciplinary research – for instance, investigating whether certain therapies or lifestyle changes measurably affect microtubule stability or whether quantum coherence can be indirectly inferred in neural systems. It also underscores the importance of lifelong learning and social engagement as potential buffers against dementia, providing a scientific rationale at the quantum level for these public health recommendations. In conclusion, by integrating Orch OR theory with a mathematical model of entanglement dynamics, we have presented a comprehensive narrative: as time's arrow advances, quantum entanglement in the brain's microtubules unwinds – but the active mind can weave it anew, staving off the entropy of Alzheimer's disease.

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