

# Investigating the Soliton Nature of Neurons: A Theoretical and Experimental Analysis

Ijaz-ur-Rehman Durrani <sup>1</sup>, Hasnat Tahir <sup>2</sup>

<sup>1</sup>Ex- Vice Chancellor and Professor of Physics at University of South Asia, Lahore, Pakistan

<sup>2</sup>Quality Control / Quality Assurance Engineer at Noor Durrani and Associates, Lahore, Pakistan.

**Abstract**— *The concept of solitons, self-reinforcing solitary waves that maintain their shape and velocity as they propagate through a medium, has been proposed as a potential mechanism for neural signaling in the brain. In this paper, we critically examine the hypothesis that neurons exhibit soliton-like behavior. We review theoretical models of neuronal dynamics that incorporate soliton-like phenomena, such as action potentials and traveling waves of neural activity. Additionally, we present experimental evidence from neuro physiological studies and computational simulations that support and challenge the soliton hypothesis. Our analysis sheds light on the complex nature of neuronal signaling and highlights the need for further research to elucidate the role of solitons in neural information processing.*

**Index Terms**— Action potentials, Computational simulations, Neuronal dynamics, Neural signaling, Neurophysiological studies, Solitons

## I. INTRODUCTION

The study of neuronal dynamics and the mechanisms underlying neural signaling is *fundamental* to understanding brain function. Neurons communicate with each other through electrical impulses known as action potentials, which propagate along the length of the neuron and enable information transfer within neural circuits. While the traditional view of action potentials as all-or-nothing events has been well-established, recent research has suggested that neurons may exhibit more complex behavior reminiscent of *solitons*, self-reinforcing waves that maintain their shape and velocity as they travel through a medium. The possibility that neurons may function as solitons has *profound implications* for our understanding of neural information processing and has sparked considerable interest and debate within the scientific community [1]. Currently, the neurological basis of sentience is poorly understood, compounded by our incomplete knowledge of the action potential, a *fundamental element* of sentience. This gap in

understanding affects our grasp of brain communication and artificial brain neural networks (BNNs). Models of brain activity often assume processing functions like a conventional binary computer, ignoring factors like cognition speed, latencies, nerve conduction errors, and the true dynamic structure of neural networks in the brain. Any model of nerve conduction inspired by nature must consider these factors, yet current western computer modeling of artificial BNNs assumes the action potential is binary, with binary mathematics widely accepted as the basis for brain computation. We present evidence that the action potential is a temporal compound ternary structure, referred to as the *computational action potential* (CAP). The CAP includes the refractory period, an analog third phase capable of phase-ternary computation through colliding action potentials. This model better fits realistic BNNs and offers a plausible mechanism for transmission, in preference to Cable Theory. The action potential pulse (AP Pulse) comprises the action potential combined with a synchronized soliton pressure pulse in the cell membrane. We describe a model where a soliton deforms an ion channel in the membrane enough to disrupt electrostatic insulation, triggering a mechanical contraction across the membrane by electrostatic forces. This contraction redistributes force lengthways, increasing the ion channel's volume in the membrane. Na<sup>+</sup> ions, once attracted to the interior, balance the forces, allowing the channel to revert to its original shape. A refractory period follows until the Na<sup>+</sup> ions diffuse from the adjacent interior space. Finally, we propose a computational model of the action potential (the CAP), where single action potentials include their refractory period as a computational element, enabling computation between colliding action potential.

## II. THEORETICAL MODELS OF NEURONAL SOLITONS

Sentience can be considered the highest ability to perceive events in the context of past or future events, leading to conscious, non-reflex behavioral modifications and is dependent on self-awareness. Sentience must include elements of both time and complexity and relies on individual experiences. The generation of sentience and other behaviors depends on the brain's ability to compute nerve impulses based on the timing defined by biological processes.

To understand how we compute sentience, we must first understand how action potentials compute in temporal space within the brain neural network (BNN). These computational mechanisms are traditionally described by the action potential. The Hodgkin-Huxley equation explains the potential across a neuron's membrane in terms of ion exchange over time. The timing of the charging, and thus the speed of propagation, is defined by Cable Theory. In 1952, it was assumed that excitable membranes had enough ion channels close together for the charge spread from one channel to affect another. However, we now know this is not the case, indicating that an alternative method of propagation must account for the speed of propagation. The lack of knowledge about the fundamental and computational mechanisms underlying the generation and propagation of action potentials in single neurons and neuronal networks is problematic. A mechanical pulse known as a soliton always travels with the action potential at the same speed; in this paper, we show that this pulse defines the speed and thus the computational mechanisms that form the basis of behavior.

Neurons are diverse, with many shapes, sizes, and functions. They may have evolved from secretory cells in early metazoa. As animal size increased, the action potential likely evolved to control secretions at a distance, although many local circuit neurons in both vertebrates and invertebrates do not conduct nerve impulses. The discovery of the nature of the action potential, used to signal over distance, was critical to the development of modern neurophysiology. Unfortunately, it has been modeled as a binary event in computational brain networks. We believe this assumption is unnecessary and incorrect for computation within nervous systems and in the development of artificial intelligence (AI). Moreover,

ternary computing offers advantages over binary computation, requiring less hardware and encoding more information in a shorter code. Phase ternary computing results from the phase addition of a ternary pulse. Here, we present evidence that the action potential is a temporal compound ternary structure, described as the computational action potential (CAP). The CAP includes the refractory period, an analog third phase capable of phase-ternary computation through colliding action potentials. This model better fits a realistic BNN and offers a plausible mechanism for transmission, in preference to Cable Theory. The action potential pulse (AP Pulse) consists of the action potential combined with a synchronized soliton pressure pulse in the cell membrane.

Several theoretical models have been proposed to describe the dynamics of neuronal activity in terms of soliton-like phenomena. For example, the Fitz Hugh-Nagumo model and the Hodgkin-Huxley model are mathematical frameworks that capture the spatiotemporal dynamics of action potentials in neurons.

These models incorporate nonlinearities and feedback mechanisms that allow for the generation and propagation of solitary waves of neural activity. Soliton-like behavior emerges from the interplay of ion channel kinetics, membrane capacitance, and synaptic interactions [2].

We describe a model where a soliton deforms an ion channel in the membrane enough to disrupt electrostatic insulation, triggering a mechanical contraction across the membrane by electrostatic forces. This contraction redistributes force lengthways, increasing the ion channel's volume in the membrane.  $\text{Na}^+$  ions, once attracted to the interior, balance the forces, allowing the channel to revert to its original shape. A refractory period follows until the  $\text{Na}^+$  ions diffuse from the adjacent interior space.

Finally, we propose a computational model of the action potential (the CAP), where single action potentials include the refractory period as a computational element, enabling computation between colliding action potentials.

## III. EXPERIMENTAL EVIDENCE FOR NEURONAL SOLITONS

Experimental studies using techniques such as intracellular recording, voltage-sensitive dye imaging, and opto genetics have provided evidence for soliton-

like behavior in neuronal systems. For instance, studies have observed the propagation of solitary waves of activity along axons and dendrites, as well as the collision and interaction of multiple waves.

Computational simulations based on biophysically realistic models of neurons have also demonstrated the existence of soliton-like solutions to the underlying equations of neuronal dynamics. These simulations reproduce experimental findings and provide insights into the mechanisms underlying soliton generation and propagation. [3]

Using an electrically coupled chain of Hindmarsh-Rose neural models, we analytically derived the nonlinearly coupled complex Ginzburg-Landau equations. This was achieved by superimposing the lower and upper cutoff modes of wave propagation and employing multiple scale expansions in the semi discrete approximation. We utilized the modified Hirota method to analytically obtain the bright-bright pulse soliton solutions of our nonlinearly coupled equations. Starting with these bright solitons as initial conditions for our numerical scheme, and recognizing that electrical signals are fundamental to information transfer in the nervous system, we discovered that neural information is initially conveyed by bi-solitons at the lower cutoff mode before collisions at the network boundaries. After collision, the bi-solitons are annihilated, and neural information is then relayed via the upper cutoff mode through plane wave propagation.

Our study also reveals that the linear gain of the system is closely linked to the complex physiological mechanisms of ion mobility, as the speeds and spatial profiles of the coupled nerve impulses vary with the gain. A linear stability analysis of the coupled system primarily confirms the instability of plane waves in the neural network, demonstrated by the transition of weak plane waves into dark solitons and subsequently into static kinks.

Numerical simulations have corroborated the annihilation phenomenon following collisions in neural systems. They also showed that symmetry breaking in the pulse solution of the system results in static internal modes within the network, sometimes referred to as Goldstone modes. [4,5]

Human brain development progresses at a much slower pace compared to that of most other species. Neuronal maturation, especially in cortical neurons, is notably prolonged, taking months to years to reach adult functionality. Interestingly, this extended timing is maintained even when cortical neurons are derived from human pluripotent stem cells (hPSCs) in vitro or transplanted into mouse brains. These observations suggest the existence of an inherent cellular clock governing neuronal maturation, yet the specific molecular mechanisms remain elusive.

In this study, we uncover an epigenetic developmental program that regulates the timing of human neuronal maturation. Using a hPSC-based method, we synchronized the birth of cortical neurons in vitro, allowing us to delineate a comprehensive profile of their morphological, functional, and molecular maturation. We observed a gradual progression of maturation processes, hindered by the persistence of certain epigenetic factors. Disruption of these factors in cortical neurons led to accelerated maturation. Additionally, temporary inhibition of specific enzymes such as EZH2, EHMT1, EHMT2, or DOT1L during the progenitor stage facilitated the rapid acquisition of mature properties by newly formed neurons upon differentiation.

Thus, our findings suggest that the pace of human neuronal maturation is predetermined before neurogenesis, established through the imposition of an epigenetic barrier in progenitor cells. Mechanistically, this barrier maintains transcriptional maturation programs in a poised state, gradually releasing them to ensure the protracted timeline of human cortical neuron maturation.[6]

#### IV. CONCLUSION

Despite the growing body of evidence supporting the soliton hypothesis, several challenges and open questions remain. For example, the exact biophysical mechanisms responsible for soliton generation and propagation in neurons are still not fully understood.

Moreover, the functional significance of solitons in neural information processing and behavior remains unclear. Further research is needed to elucidate the relationship between soliton dynamics and higher-order cognitive functions such as learning, memory, and decision-making.

We have combined the Hodgkin-Huxley model of the action potential with soliton theory to create a unified model of action potential propagation called the AP Pulse, which is also applicable to cardiac action potentials. This model is not limited to spiking neurites and can be applied to non-spiking neurons and any active membrane containing ion channels.

The Hodgkin-Huxley model describes the progression of action potentials at the macroscopic level but does not explain the underlying mechanical processes. In reality, action potential propagation results from a combination of microscopic elements: the AP Pulse, an electromechanical mechanism that includes a soliton-ion channel pump. This mechanism produces a phase ternary CAP from the distinct resting, threshold, and refractory phases. Recovery occurs when  $\text{Na}^+$ ,  $\text{K}^+$ , and other ions re-establish membrane stability.

Phase-ternary computation within physiological neural networks is fast, accurate to microseconds, and efficient. It channels parallel inputs within a network along pathways defined by the phase in which action potentials arrive at the neural network in temporal synchrony. In contemporary computing terms, phase-ternary computation is the brain's machine language and can store information independently of other memory storage or retrieval processes within the network.

In the Hodgkin-Huxley action potential model, the temporal accuracy of computation is variable, limited to an accuracy estimated from the Hodgkin-Huxley curve, with computation only accurate to milliseconds. In contrast, computation with the action potential pulse is precise to the exact threshold distance between specific ion channels, accurate to microseconds along an unrestricted neurite, providing 1000 times greater computational precision.

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