Review of: "Approaching EEG Pathological Spikes in Terms of Solitons"

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Potential competing interests: No potential competing interests to declare.

I recently read the Qeios posting [“Approaching EEG pathological spikes in terms of solitons”] by Tozzi and wanted to share some thoughts, if I may.

Concerning the statement (pg. 2): “The energy of solitons is carried adiabatically through the medium, i.e., without transferring heat or mass...” While I agree that the electrical activity of the underlying neurons can and does sum to produce the oscillations seen in EEG recordings, the basis of the underlying action potentials that generate this electrical activity is not adiabatic. Action potential generation has been shown to generate net heat (as reported in Abbott et al. 1958; see citations below) and is an active dissipative process which requires coupling to the flow of metabolic energy to be generated and maintained (as shown by the work reviewed in Ritchie 1973). Thus, if to be a soliton the action potential must be generated and pass along without net heat production, it seems that the action potential does not meet this requirement? (If you know of any recent studies which have measured the actual heat from action potentials with more modern methods and have found there to be no net heat, why not cite such work in support of the claim that action potentials are adiabatic?) That does not mean that an analysis of how the underlying waveforms might interact to produce the EEG patterns we often observe would not be of interest; indeed, perhaps modeling the EEG waves as solitons as a conceptual starting point might lead to some interesting perspectives. It simply implies that the underlying action potential mechanism is not likely to be that of a soliton?

As many likely know, the Heimburg-Jackson soliton/action potential model (Heimburg et al. 2006) proposes that a lipid phase transition occurs in the plasma membrane of the neuron, and that during this phase transition, the local surface potential of the membrane shifts due to changes in the lipid density with the passage of a lipid phase transition along the surface of the membrane. It is implied that there would be a difference in the surface potential shifts that occur on the outer face of the plasma membrane versus the inner face of this membrane, and the suggestion is made that the difference between these two potentials produces the pattern of electrical changes over time that we categorize as an action potential. However, there are several problems with the application of this model to neurons as a means to account for action potentials. One is that, to the best of my knowledge, there is no clear evidence that during an action potential in a neuron, there is the sort of lipid phase transition occurring that this model requires. (If you know of a recent study which presents such evidence, why not cite it in support of the claims made by this soliton-AP model?) The various mechanical and optical features that are noted to be associated with the action potential can be accounted for in a manner consistent with the modern electrophysical action potential model, which is derived from the work of Hodgkin and Huxley. Another

problem is that this soliton-AP model that Heimburg and Jackson are proposing suggests that surface potential changes have macroscopic influences. However, the surface potentials of membranes are noted in the literature to only have influences across about a nanometer of distance and so are microscopic (see Wang 2012, and Galassi et al. 2021). Thus, for this Heimburg-Jackson soliton-AP model to function, the nature of surface potentials would have to be shown not to be microscopic, but rather macroscopic in nature. I am not aware of any work which supports such a view, but if you know of any report of surface potentials in aqueous physiological media (with dipole molecules such as water and ions present as these shield surface potentials rather effectively) being able to have macroscopic influences, and so are able to have influences across the macroscopic distances as seen by action potentials, then why not cite such work in your article so that readers can see support for this claim?

Also, on pg. 8 of this article, it states: "Two colliding solitons penetrate through each other, exchange their amplitude and velocity, and emerge fully intact as the exact pulses that entered the collision (Poznanski et al., 2017)." It should be noted that the claims of action potential passage through one another upon collision have not, to my knowledge, been confirmed by observation. What the Poznanski et al. (2017) article does is present what is calculated to happen based on theory; it does not actually confirm through actual observation the passage of action potentials in actual living neurons. You might wish to see the comments made by Berg et al. (2017), who notes that typically upon collision in neurons, action potentials undergo "annihilation," not penetration of each other, and there are biophysical and biochemical mechanisms noted to account for this happening. Thus, again, to my knowledge, there is no confirmation of the claim from Heimburg's lab group that action potentials "penetrate" one another upon collision. And this then would seem to be yet another way that the action potentials in neurons differ from what would be expected of solitons?

Thus, in your article, when you close with the question of how solitons might be generated in neurons to create the pattern of electrical changes we call an action potential, that is a question I very much share. And, so far, I have not found convincing evidence that action potentials are solitons, at least not in the manner suggested by the Heimburg-Jackson soliton-AP model.

Finally, in the interests of full disclosure, here are the citations of two articles I have posted, in my retirement, on the topic of the soliton-AP model. I would note that I am not opposed to the model, per se; it is just that I have not, yet, seen compelling evidence for its operation in neurons...

Meissner S.T.- 2018-Proposed tests of the soliton wave model of action potentials, and of inducible lipid pores, and how non-electrical phenomena might be consistent with the Hodgkin-Huxley model- Physics arXiv, 45 pgs, arXiv:1808.07193v1 [physics.bio-ph], http://arxiv.org/abs/1808.07193

Meissner S.T- 2022-Additional proposed tests of the soliton/wave-action potential model, and how the thermodynamic/theory-based philosophical approach abandons the scientific method. Preprints 2022, 2022080248 (doi: 10.20944/preprints202208.0248.v1).

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Citations of articles noted above:

Abbott B.C., A.V. Hill, J.V. Howarth- 1958-The positive and negative heat production associated with a nerve impulse- Proceedings of the Royal Society of London B, 148: (#931, 2/18) 149-187.

Berg R.W., M.T. Stauning, J.B. Sørensen, H. Jahnsen- 2017-Comment on "Penetration of action potentials during collision in the median and lateral giant axons of invertebrates"- Physical Review X 7: 028001, 3 pgs. doi: 10.1103/PhysRevX.7.028001.

Galassi V.V., N. Wilke- 2021-On the coupling between mechanical properties and electrostatics in biological membranes- Membranes 11: 478, 24 pgs., https://doi.org/10.3390/membranes11070478

Heimburg T., A.D. Jackson- 2006-On the action potential as a propagating density pulse and the role of anesthetics- arXiv:physics/ob10117v2 [physics.bio-ph] 13 pgs.

Poznanski R.R., L.A. Cacha, Y.M.S. Al-Wesabi, J. Ali, M. Bahadoran, P.P. Yupapin, J. Yunus- 2017-Solitonic conduction of electrotonic signals in neuronal branchlets with polarized microstructure- Scientific Reports 7: 2746, 12 pgs., doi: 10.1038/s41598-017-01849-3

Ritchie J.M- 1973-Energetic aspects of nerve conduction: The relationships between heat production, electrical activity and metabolism- Progress in Biophysics and Molecular Biology 26: 147-187.

Wang L- 2012-Measurements and implications of the membrane dipole potential- Annual review of Biochemistry 81: 615-635, doi: 10.1146/annurev-biochem-070110-123033