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Opinion

A Case for Electromagnetic Approach to Cellular Activities

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Cellular transport proteins are of primordial importance to cell survival. These proteins span the entire cellular membrane. They sometimes form channels that allow for the passage of nutrients that would not otherwise be able to traverse the cellular membrane due to their overall charge. These essential factors are set in a certain local electromagnetic landscape which coordinates their function and morphology. This electromagnetic environment is facilitated and maintained by the establishment of membrane potentials which regulate the activities of these aforementioned transporters.

Charged species, internal or external to the cell interior, are either able or unable to cross the cellular membrane via transport proteins. This crossing is refereed by facilitated or active transports. Membrane potentials result from and are maintained by the crossing or lack thereof of these charged species and the crossing of the charged species is regulated by the membrane potential [1]. I contend that this electromagnetic environment, which the membrane potential is an integral part of, contains the key to understanding most if not all of the cell's activities including its morphology, enzyme kinetics, immune system response just to name just a few.

For example, it is known that often a cell will have different transporters for the same nutrient, but they differ in their affinities for the nutrient in question. In today's language, we would say that the protein transport network contains redundancy, that is, different ways of doing the same thing. As an aside, let me submit that, at least on a molecular level, redundancy seems to be crucial for and one of the hallmarks of evolution. For example, redundancy in their biochemical and/or genetic pathways and/or mechanisms seems to be the methods by which the parasites Trypanosoma brucei rhodesiense and Tryponosoma brucei gambiense, causative agents of Human African Trypanosomaisis, HAT, are able to evade not only the immune system's response but also the injurious effects of trypanocidal drugs even though their only source of energy is glycolysis [2]. But I digressed!

There is also a very interesting similar but different phenomenon where a transporter can switch affinity for the same substrate. A good example is the Arabidopsis nitrate transporter CHL1 that can switch from high to low affinity once the protein is phosphorylated [3]. But that is just it: phosphate groups are negatively charged species and their attaching to the protein is an electrodynamic phenomenon which can possibly cause atomic rearrangement that is turn can modify the active site precipitating a different type of electrical activity that we translate into affinity.

Another example is cellular 'sensing' of metals which at every level of the process, whether changes in gene expression, compartmentalization, storage and transport, seems to involve some sort of local electromagnetic fluctuations at its core [4]. What of the 'sensing' of uncharged species? Does it bear the imprint of electromagnetic influences? It is known that in S. cerevisiae the 'sensing' of glucose, an uncharged species in that specific yeast milieu, is uncoupled from its uptake [5]. However, the 'sensing' part in this particular environment still involves electromagnetic interactions [6], and therefore they still prove to be of consequence as this 'sensing' affects growth in the case at hand [5].

Finally, molecular 'docking' methodology is yet another area where an electromagnetic formalism should prove useful. Molecular docking methodology is concerned with the behavior of small molecules in the binding site of a target protein. Molecular docking programs in the context of drug discovery assigns an affinity score to each candidate ligand; the score is slated to be the sum of the electrostatic and van der Waals energies of said ligand [7].

It is true that presently monitoring, at the molecular level, the electromagnetic interactions of the enormous number of metabolites and reactions involved in cellular activities is nearly computationally impossible. However, we can attack the problem in small steps by probing small intracellular compartments for which a vast array of experimental data is available. This is necessary because until we do take a serious and courageous look using a full electromagnetic formalism, our understanding of the fundamental way the cell operates will be obstructed and our results will only make sense on the average and probably the wrong average at that!.

References

1. Fontus M, Ortoleva P (2011) Electro metabolomics modeling of microbes: Applications in fuel cells and environment analysis. Journal of Biotech Research 3: 37-50.
2. McGowen T, Fontus M (2017) Proposing drug target(s) to combat trypanosoma infection. PURSUE Undergraduate Research Journal 1(1): 25-39.
3. Liu KH, Tsay YF (2003) Switching between the two action modes of the dual-affinity nitrate transporter CHL1 by phosphorylation. EMBO J 22(5): 1005-1013.
4. Bird AJ (2015) Cellular sensing and transport of metal ions: Implications in micronutrient homeostasis. J Nutr Biochem 26(11): 1103-1115.
5. Youk H, Oudenaarden AV (2009) Growth landscape formed by perception and import of glucose in yeast. Nature 462(7275): 875-879.
6. Moriya H, Johnston M (2004) Glucose sensing and signaling in *saccharomyces cerevisiae* through the Rgt2 glucose sensor and casein kinase I. Proc Natl Acad Sci USA 101(6): 1572-1577.
7. Pagadala N, Syed K, Tuszynski J (2017) Software for molecular docking: A review. Biophys Rev 9(2): 91-102.