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Poster presentation

Sodium gating capacitance and the optimization of the squid giant axon for metabolic energy usage

Patrick Crotty* and William B Levy

Address: Department of Neurological Surgery, University of Virginia Health System, Charlottesville, VA 22908, USA

Email: Patrick Crotty* - prc9m@virginia.edu

* Corresponding author

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Background

In previous work [1,2], we observed that the ionic fluxes during an action potential (AP) in the squid giant axon can be divided into three functionally separate components. Of these, the component responsible for the depolarizing phase of the AP, and hence its velocity, attains a minimum as a function of the ion channel densities and the axon diameter very near the experimental values of these parameters when the AP velocity is constrained to be at a single value. Since the ion channel fluxes are proportional to the metabolic energy consumption via the ATPase Na^+/K^+ exchanger, this suggests that evolution, subject to an external constraint on AP velocity, has optimized ion channel densities and axon diameters for the energy associated with the velocity. The energy minimum is close to, although not identical with, a similar minimum in the total membrane capacitance. The total capacitance consists of the intrinsic membrane capacitance (about 0.88 μF/cm^2) and a term proportional to the active Na^+ channel density (about 1 nF/mS of Na^+), the so-called sodium "gating capacitance," which arises from movements of charged segments of the Na^+ protein during conformal changes. In the present work, we investigate and resolve the discrepancy in the locations of the energy and membrane capacitance minima.

Methods

The Hodgkin-Huxley squid giant axon model was simulated using NEURON and NMODL. The axon diameter and the ion channel densities were taken as two independent parameters, with the channel densities (consisting of voltage-gated Na^+, voltage-gated K^+, and nonspecific leak channels) varied by a common factor and parameterized by the maximum sodium conductance. Note that this also necessitated varying the sodium gating capacitance by this factor. Constraining the velocity to be at a single value, we determined how the shape and height of the action potential varied along the resulting isovelocity curve.

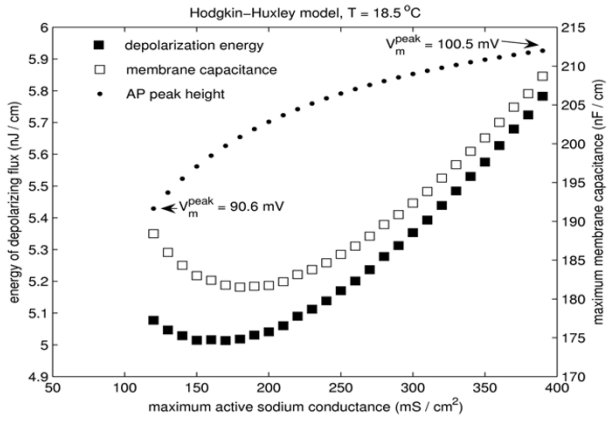

Figure 1
Action potential peak height, depolarization energy and membrane capacitance as functions of sodium conductance.

Results

Our results are summarized in Figure 1. All quantities are plotted on the 21.2 m/s isovelocity curve in axon diameter-channel density phase space. The amount of charge per unit axial length on the membrane capacitor at the peak of the action potential is $q_P = c_m V_m^{peak}$, where c_m is the total membrane capacitance per unit axial length. This charge is approximately equal to the total depolarizing charge crossing the membrane during the action potential, and hence is proportional to the metabolic energy. Since V_m^{peak} increases with the ion channel densities, values of c_m further to the right are more heavily weighted in the product $c_m V_m^{peak}$. This causes the minimum in the charge and energy curves to be further to the left than in the c_m curve alone.

Conclusion

The discrepancy between the locations of the energy and capacitance minima is resolved by considering the amount of charge placed on the membrane capacitor at the peak of the action potential. The AP peak-height rises with the ion channel densities, and therefore, the ion flux per unit membrane surface area across the membrane and the associated metabolic energy also increase. When this increase is taken into account, the location of the net charge minimum is at the same channel densities as the depolarizing energy minimum. This also illustrates that energy, rather than capacitance, is what evolution has minimized.

References

1. Crotty P, Sangrey T, Levy WB: **Metabolic energy cost of action potential velocity.** *J Neurophysiol* 2006, **96:**1237-1246.
2. Levy WB, Sangrey T, Friesen WO, Crotty P: **Reply to Jurisic and Bezanilla's letter to the editor.** *J Neurophys* 2006, **96:**960.