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Modeling the interplay between interneuron and pyramidal cell during seizures

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Summary

We present an ionic current model composed of Hodgkin-Huxley type neurons aided by variable ion concentration dynamics to investigate the role of various mechanisms in neuronal interplay seen during seizure-like events [1].

Methods

We model both cells using a two compartmental model with (1) axo-somatic, and (2) dendritic compartments. The equilibrium potentials for various ion concentrations and leak conductance are updated based on instantaneous ion concentrations inside and outside the cell using Goldman-Hodgkin-Katz equation. The K+/Na+ concentration in the interstitial volume surrounding each cell was continuously updated based on K+/Na+currents across the neuronal membrane, K+/Na+ pumps, uptake by the glial network surrounding the neurons, and lateral diffusion of K+ within the extracellular space.

Results

We investigated the cellular mechanism shaping the interplay between interneurons and pyramidal cells using compartmental models of two cells coupled through synaptic inputs and extracellular K+ diffusion. We find physiological conditions under which the two cells are locked into interplay during seizure-like events. The two cells exhibit the interplay when (1) the lateral K+ diffusion is taken into account, (2) Inhibitory and excitatory synaptic strengths are within certain range, and (3) The persistent sodium current is included in the pyramidal cell.

Conclusion

We conclude that the extracellular diffusion of K+ions and the persistent sodium current play a major role in shaping the interplay between IN and PC during seizure-like events.

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