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Generating dendritic Ca^{2+} spikes with different models of Ca^{2+} buffering in cerebellar Purkinje cells

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Ca^{2+} mechanisms, present mainly on the dendritic tree of cerebellar Purkinje cells (PC) [1], significantly influence its activity pattern [2,3], synaptic integration [4], etc. Particularly, the intracellular dynamics controlling Ca^{2+}concentrations can play a crucial role in the physiological interaction between the Ca^{2+} channels and Ca^{2+}-activated K^+ (KCa) channels [5]. The simplest, but commonly used model, the Ca^{2+} pool with a short relaxation time, will fail to simulate interactions occurring at multiple time scales. On the other hand, detailed computational models including various Ca^{2+} buffers and pumps [6] can result in large computational cost due to radial diffusion in large compartments, which may need to be avoided when simulating morphologically detailed PC models.

We present a method using compensating mechanisms to replace radial diffusion and compared the dynamics of different Ca^{2+} buffering models during generation of dendritic Ca^{2+} spikes during somatic bursting or depolarization [1]. As for the membrane mechanisms, we used a recently constructed single compartment model of a PC dendritic segment with the Ca^{2+} channels of P- and T-type and KCa channels of BK- and SK-type, which can generate the Ca^{2+} spikes comparable to the experimental recordings [7]. The Ca^{2+} dynamics models are (i) a single Ca^{2+} pool, (ii) two Ca^{2+} pools respectively for the fast and slow transients, (iii) detailed Ca^{2+} dynamics with calbindin, parvalbumin, pump and diffusion, and (iv) detailed Ca^{2+} dynamics with calbindin, parvalbumin, pump and diffusion compensation [6]. The simulated membrane voltage was compared with electrophysiological data.

Our results show that detailed Ca^{2+} dynamics models with buffers, pumps, and diffusion have significantly better control over Ca^{2+} activated K^+ channels and lead to physiologically more realistic simulations of Ca^{2+} spikes. Furthermore, the effect on Ca^{2+} dynamics of removing diffusion from the model can largely be eliminated by the compensating mechanisms. Therefore, physiologically realistic Ca^{2+} concentration dynamics can be simulated at reasonable computational cost.

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