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## **Meshless Solutions to PDE Model for Calcium Signaling in Ventricular Myocytes**

Calcium dynamics plays a central role in studying the excitation-contraction (EC) coupling process which allows cardiac muscle cells to pump blood through the heart and around the body. For many reasons such as patient safety, mathematical models of calcium dynamics are becoming an increasingly powerful tool in the study of the heart and cardiomyopathy. Computational simulations can be used to model calcium movements in heart muscle cells and therefore simulate heart conditions.

In this paper, we model such calcium movements by a system of nonlinear diffusion-reaction partial differential equations (PDEs). Radial basis functions are used to provide a ‘mesh-free’ method for data reconstruction, as well as numerical solutions of the proposed PDE model. Two meshless methods are involved: the local radial basis function collocation method, and the localized method of particular solution methods. Two numerical methods discretize the time space in the differential model in an explicit and implicit way, respectively. The differences in terms of time step size and rate of convergence of the methods are compared. The numerical experiments show that predictions of calcium signaling process in ventricular myocytes with realistic transverse-axial tubular geometry and inhibited sarcoplasmic reticulum are extremely sensitive to the numerical methods used in the PDE model.

Computational simulations predict the calcium flows through the heart cells, which describe the excitation-contraction of the muscle. In particular, we compared the accuracy and efficiency of the computational methods in a realistic heart muscle cell. Due to the complexity of the cell geometry, currently it still takes an hour to model a single cycle of the EC process with only a small portion of the whole cell. However, the further assessment and improvement in the computational technique for solving the PDE model will enable more complex and detailed investigations of heart muscle disease.