New Ca^{2+}-buffers for enhanced performance

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Diastolic heart failure (DHF) is a clinical syndrome characterized by depressed myocardial relaxation performance and poor ventricular refilling. Defective intracellular calcium (Ca^{2+}) handling underlies one of the fundamental mechanisms of DHF. Manipulating the content and function of Ca^{2+} handling proteins in the heart has been the focus of intense study to develop effective therapies for DHF patients. Parvalbumin (Parv), a skeletal muscle Ca^{2+} binding protein, has been shown to facilitate myocardial relaxation both in vitro and in vivo. Parv acts as a unique "delayed" Ca^{2+} buffer and facilitates Ca^{2+} sequestration from cytosol. Here, we summarize studies employing gene transfer of Parv in cultured adult cardiac myocytes and in vivo to redress depressed diastolic function. By targeting defects in cardiac Ca^{2+} handling, Parv represents a promising therapeutic candidate for alleviating diastolic dysfunction in DHF.