Heart failure patients often experience reduced exercise capacity and excessive muscle fatigue. One of the hypotheses concerning the mechanisms of fatigue is that intrinsic alterations in the sarcoplasmatic reticulum (SR) Ca\textsuperscript{2+} uptake and/or release properties limit activation of the contractile apparatus and reduce force output. SR Ca\textsuperscript{2+} ATPase (SERCAs) pumps cytosolic Ca\textsuperscript{2+} into the SR, enabling the muscle to relax following contraction. It has been reported a reduction in the activity or pump rate of SERCA in muscles after exercise. SERCA proteins exist as 3 family members. SERCA1, expressed in fast skeletal muscle fibers, SERCA2, expressed in slow skeletal muscle fibers and the heart and SERCA3, expressed in various non-muscle tissues. Loss of SERCA1 results in exercise-induced impaired muscle relaxation in humans (Brody’s disease). Little is however known about the effects of loss of SERCA2 on skeletal muscle function. We have generated Serca2\textsuperscript{flox/flox} MLC-1f\textsuperscript{wt/cre} (MLC1f-SERCA2KO) mice, in which the Serca2 gene is deleted in skeletal muscle. Serca2 gene excision was detected in soleus, extensor digitorum longus, tibialis anterior, gastrocnemius muscles and the diaphragma, but not in the heart or in non-muscle tissue. MLC1f-SERCA2KO mice appeared overall normal. In soleus muscle, Serca2 mRNA and SERCA2 protein were reduced to 12 ± 2 % (SEM) and 6 ± 2 % of control values, respectively. Serca1 and Serca3 mRNA or protein expression levels were unaltered. Preliminary in situ experiments showed that there was no difference between MLC1f-SERCA2KO and control soleus muscles in the maximum tetanic force generation and fatigue development. Our preliminary conclusion is that MLC1f-SERCA2KO soleus muscle shows normal contractile properties in spite of near loss of SERCA2. Further studies include measuring Ca\textsuperscript{2+} sensitivity, Ca\textsuperscript{2+} transients and force in single fibers, and investigate the expression and activity pattern of other Ca\textsuperscript{2+} transporting proteins and myofilament protein components.