SUPPLEMENTAL MATERIAL

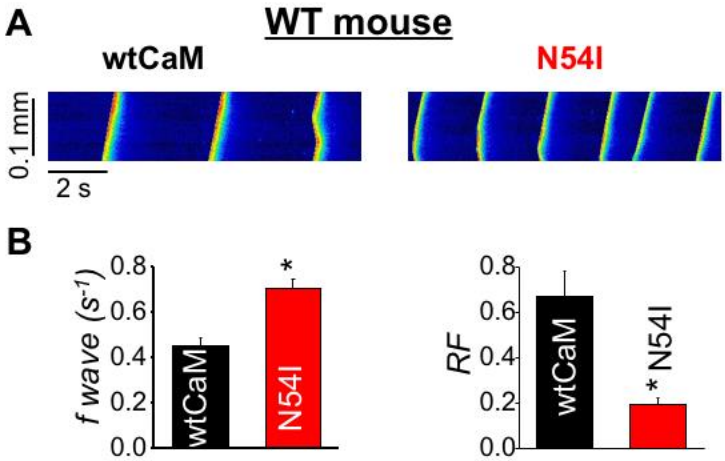

Figure S1. CPVT CaM N54I increased Ca waves frequency and shortened RyR2 refractoriness. **A)** representative line-scan images of SCWs in permeabilized WT myocytes exposed to cAMP. **B**) Average frequency of SCWs (n=46-51 cells) and refractoriness (n=21-22 cells), * $p<0.05$ vs wtCaM. CPVT, Catecholaminergic polymorphic ventricular tachycardia; CaM, Calmodulin; Ca, Calcium; RyR2, Ryanodine receptor 2; cAMP, Cyclic adenosine monophosphate; SCW, spontaneous Ca waves; WT, wild type.

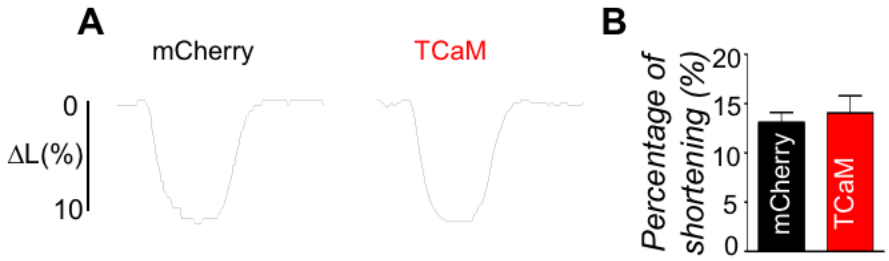

Figure S2. TCaM did not alter the extent of myocyte shortening in R33Q myocytes. **A)** representative shortening traces of R33Q myocytes infected with control (mCherrry) or TCaM virus. **B)** Average percentage of shortening (n=45-47 cells), * $p<0.05$ vs mCherry. TCaM, Therapeutic Calmodulin.

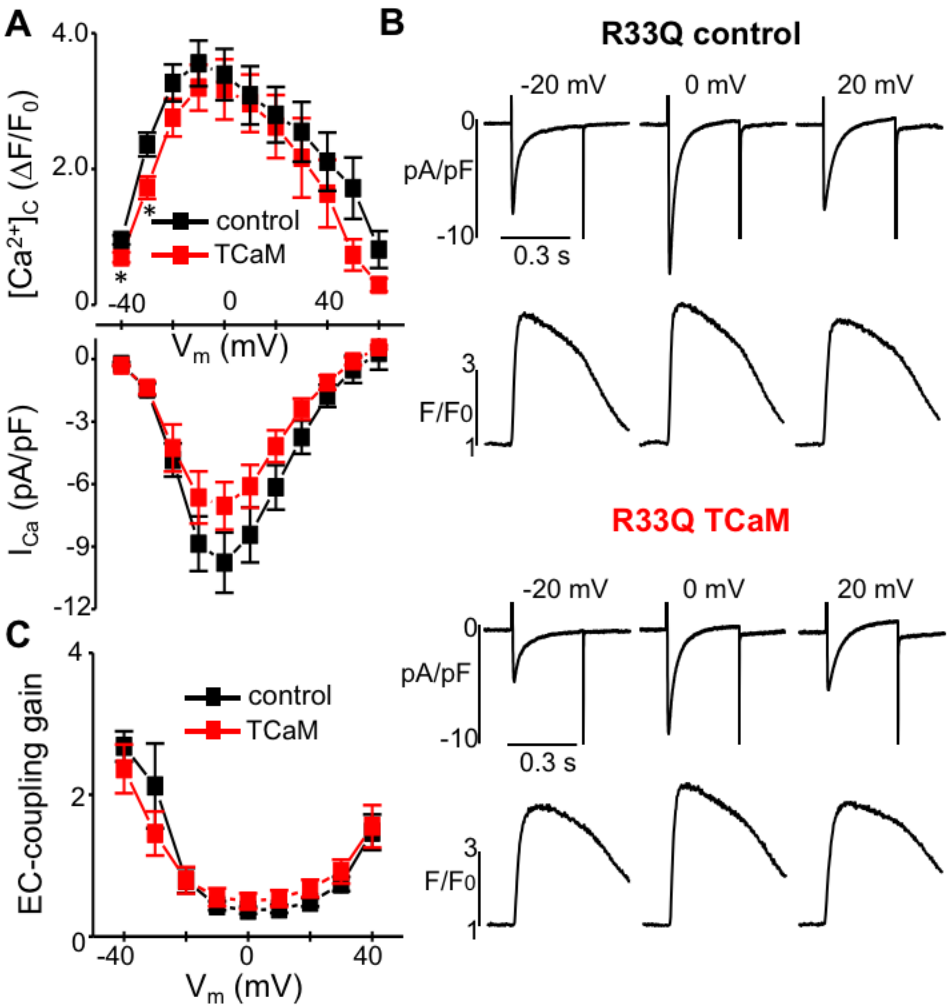

Figure S3. EC-coupling in R33Q myocytes noninfected (control) and infected with TCaM. A) Voltage-dependence of Ca currents (I_{Ca}) and corresponding Ca transients recorded in control R33Q myocytes (n=6) and in R33Q myocyte expressing TCaM (n=4). **B)** Representative traces of Ca transients and I_{Ca} evoked by depolarizing steps from -50 to -20, 0 , and 20 mV in control and TCaM myocytes, respectively. **C**) The EC-coupling gain in R33Q control and TCaM myocytes.*, P<0.05 vs control. EC, excitation contraction; TCaM, Therapeutic Calmodulin; Ca, Calcium; I_{ca}, Calcium current.