Supplemental Materials (Figures)

Figure S1. PKC activation has no effect on sarcomere structure in cultured cardiomyocytes as determined by α-actinin staining.

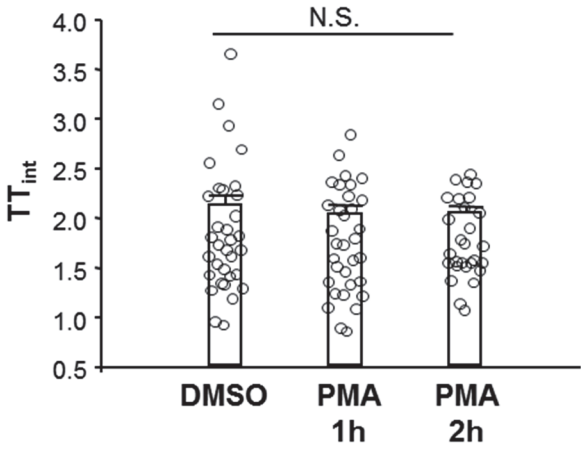

Figure S2. Short term effect (1h or 2h after treatment) of PMA on T-tubule integrity. T-tubule imaging was performed immediately after PMA treatment was completed.

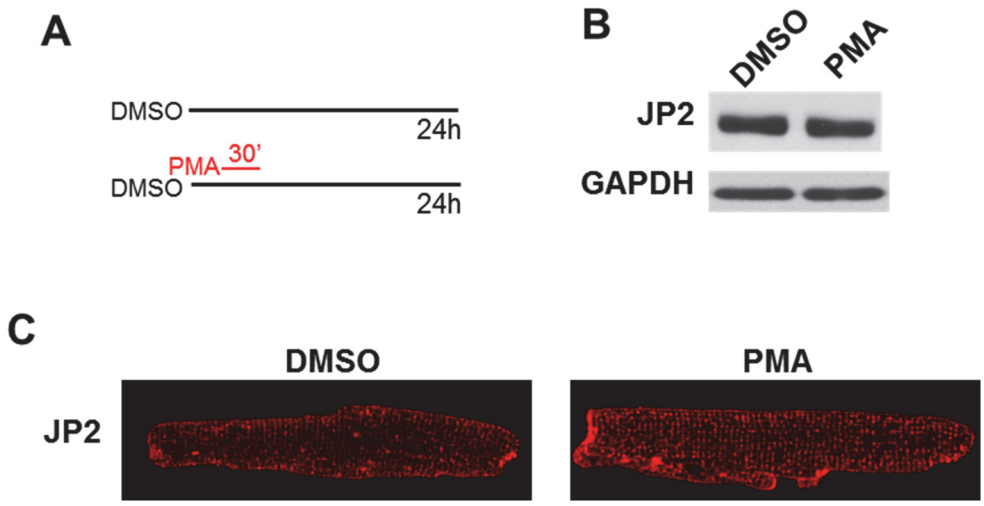

Figure S3. Transient PKC activation has no effect on junctophilin 2 (JP2) expression or subcellular distribution. A, Schematic presentation of experimental protocol. **B & C**, Analysis of JP2 expression by immunoblotting (**B**) and distribution by immunofluorescence (**C**).

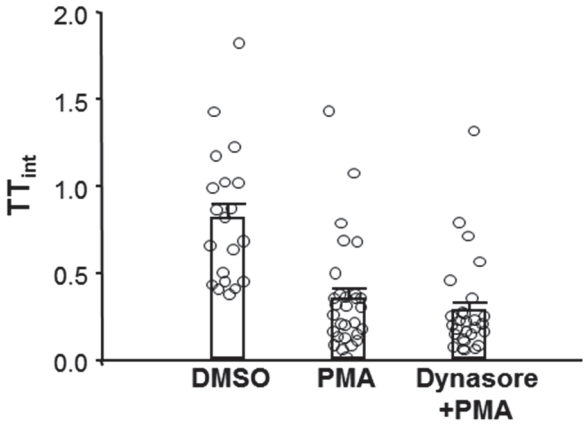

Figure S4. Endocytosis is not required for the effect of transient PKC activation on T-tubule remodeling. T-tubule integrity was determined at 24 h after 30 min PMA treatment and washout in the presence of the endocytosis inhibitor, Dynasore.

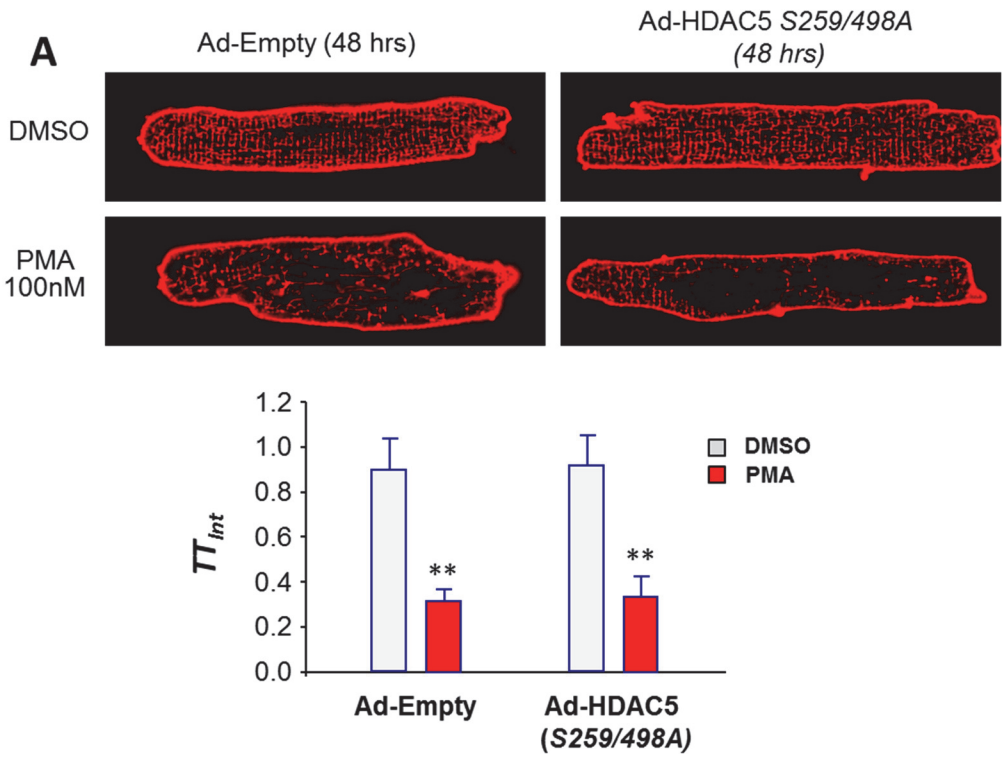

Figure S5. Adenoviral infection of HDAC5 mutant (S259/498A, 48 hours) does not prevent PMA-induced T-tubule damage. HDAC5 S259/498A mutant represses myocytes from PKC-hypertrophic signaling by disabling CaMKII-phosphorylation of HDAC5. n=16, 19, 18 and 15 cells/group. **, $p<0.01$ vs DMSO treatment.

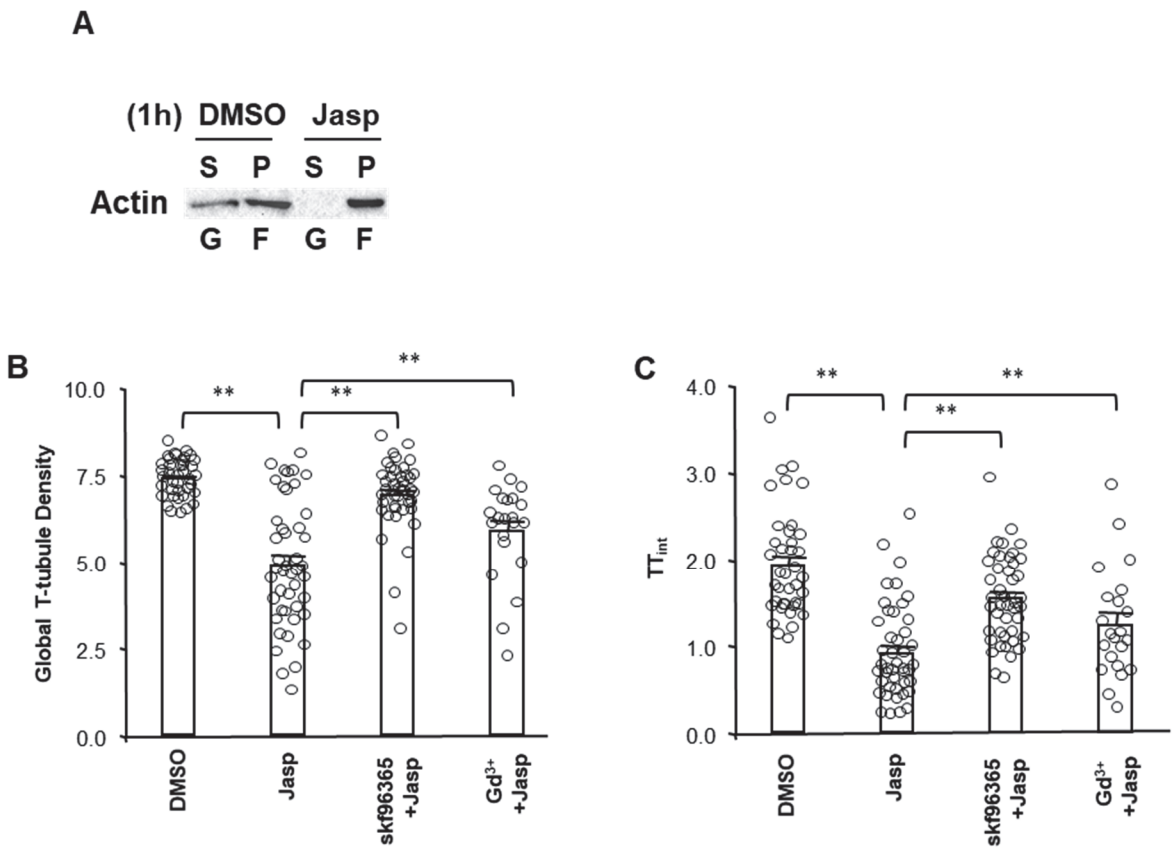

Figure S6. Excess de novo F-actin synthesis damages T-tubules, and stretch activated channels inhibitors attenuates T-tubule damage. A, Western blot of F-actin polymerization following 1 hour treatment with Jasplakinolide (Jasp), which stabilizes F-actin and promotes de novo F-actin polymerization. **B-C**, Jasp induced T-tubule remodeling and protective effect of skf96365 and Gd^{3+}. Both skf96365 / Ga^{3+} and Jasp were continuously present in culture medium until T-tubule imaging at 24 hours later. n=39, 45, 44 and 22 cells per group, respectively. ** $p < 0.01$.