Supplemental Figure Legends

Figure S1. Western blot analysis of PKCα after reintroducing FADD into FADD-/- MEFs. Cells were transfected with empty or FLAG-FADD plasmids for 24 h and then treated with the indicated concentrations of PMA for 12 h.

Figure S2. The PKCβII plasmid was overexpressed with or without the PP2Ac plasmid for 24 h in 293T cells. Cells were fractionated into the detergent-soluble supernatant (S) and detergent-insoluble pellet (P).

Figure S3. Phospho-TM and phospho-HM determined using western blot analysis. Similar protein levels of PKCβII were immunoprecipitated which was determined by HA antibody.

Figure S4. Western blot analysis of PKCβII, PKCδ and pERK in cytosolic extracts of cardiac muscle from control littermates (L) and FADD-D mice (D). Representative results from 10 pairs of mice are shown.

Figure S5. Transwell assay of FADD+/+ and FADD-/- MEFs. Represent results of Figure 7C.

Figure S6. Transwell assay of wild type (WT) and FADD-D MEFs. Represent results of Figure 8C

Figure S7. A schematic diagram illustrating how FADD and its phosphorylation regulate cytoskeleton and cell motility via PKC signaling. FADD is required for PP2A interacts with PKCα to reduce cPKC TM and HM phosphorylation. When FADD is deficient or phosphorylated, the interaction between cPKC and PP2A is impaired. As a result, endogenous cPKC S

Figure S1.

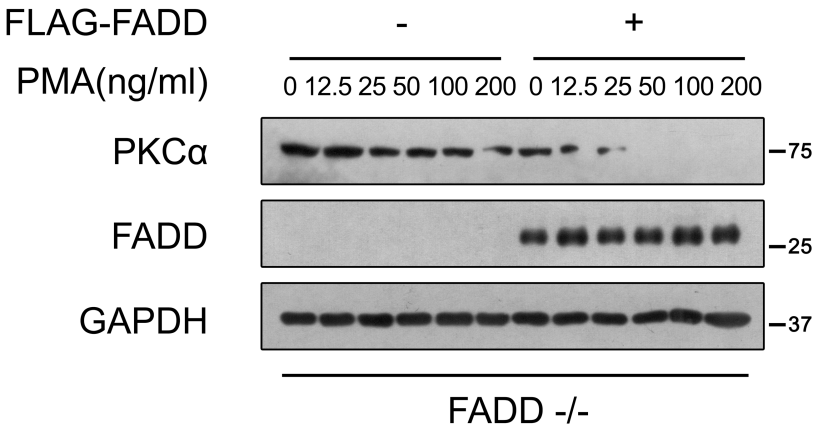


Figure S2.

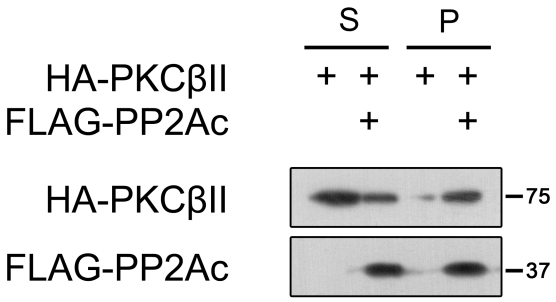


Figure S3.

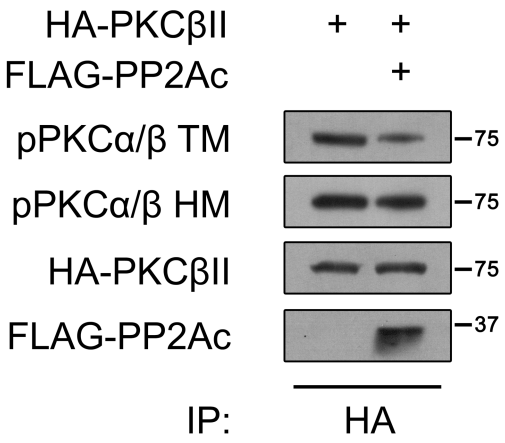


Figure S4.

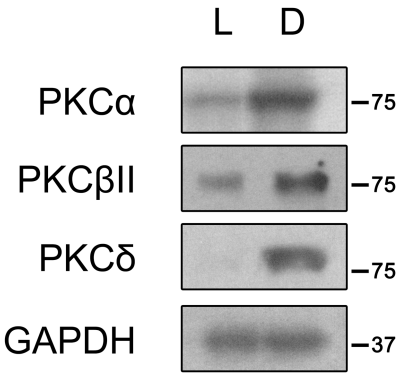


Figure S5.

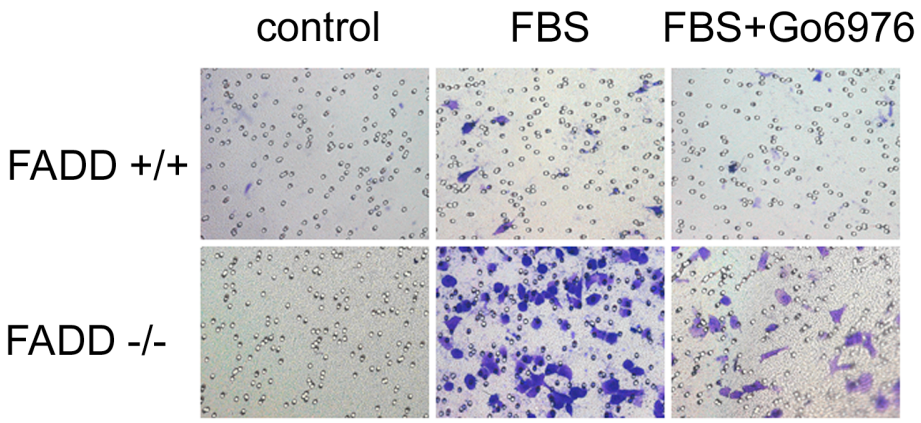


Figure S6.

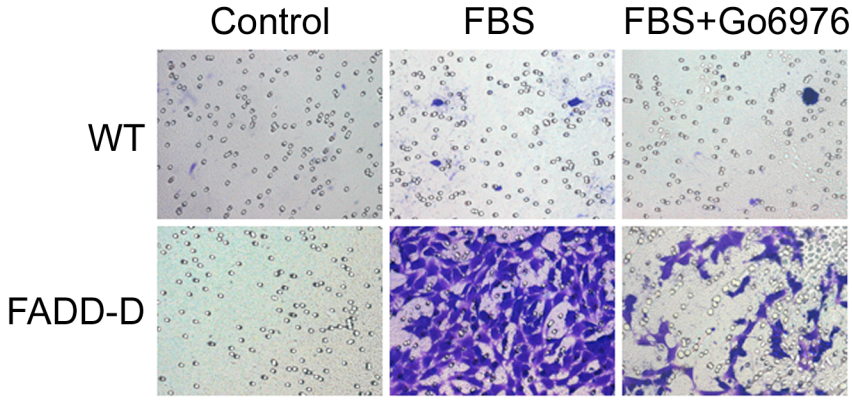
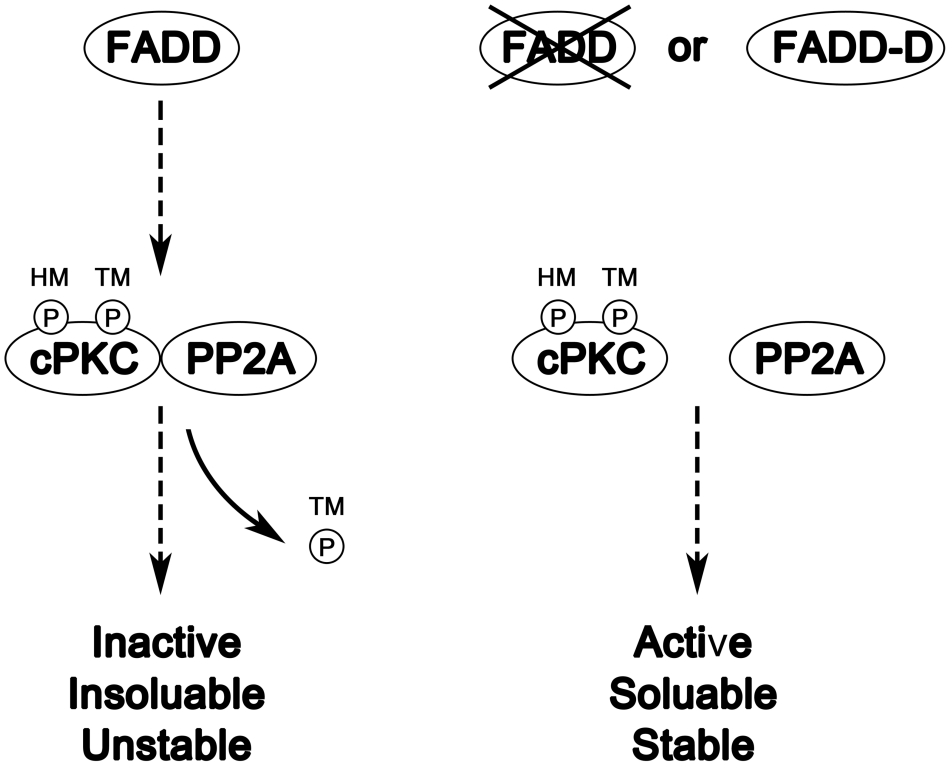


Figure S7.


FADD
FADD
or
FADD-D
HM
TM
P
P
cPKC
PP2A
TM
P
HM
TM
P
P
cPKC
PP2A
Inactive
Insoluable
Unstable
Active
Soluable
Stable