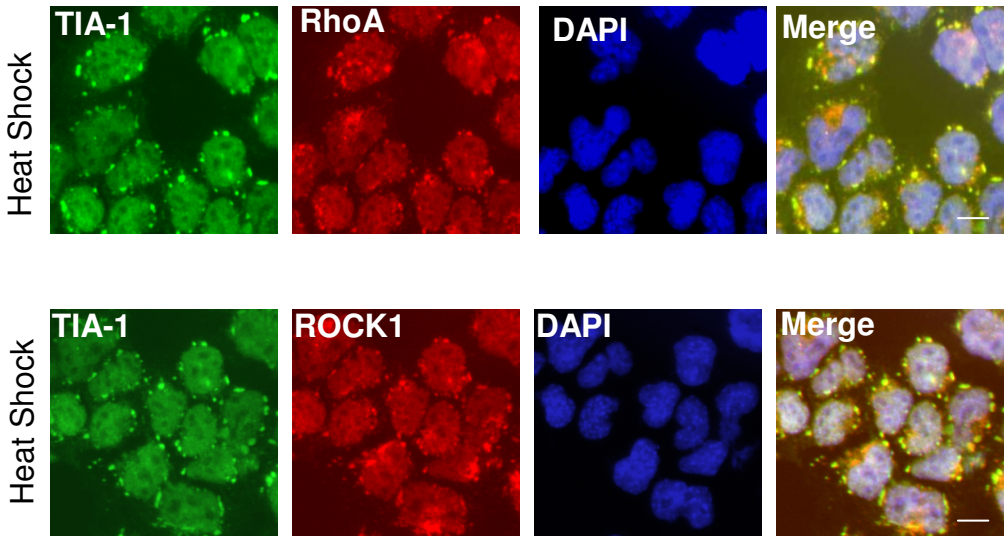
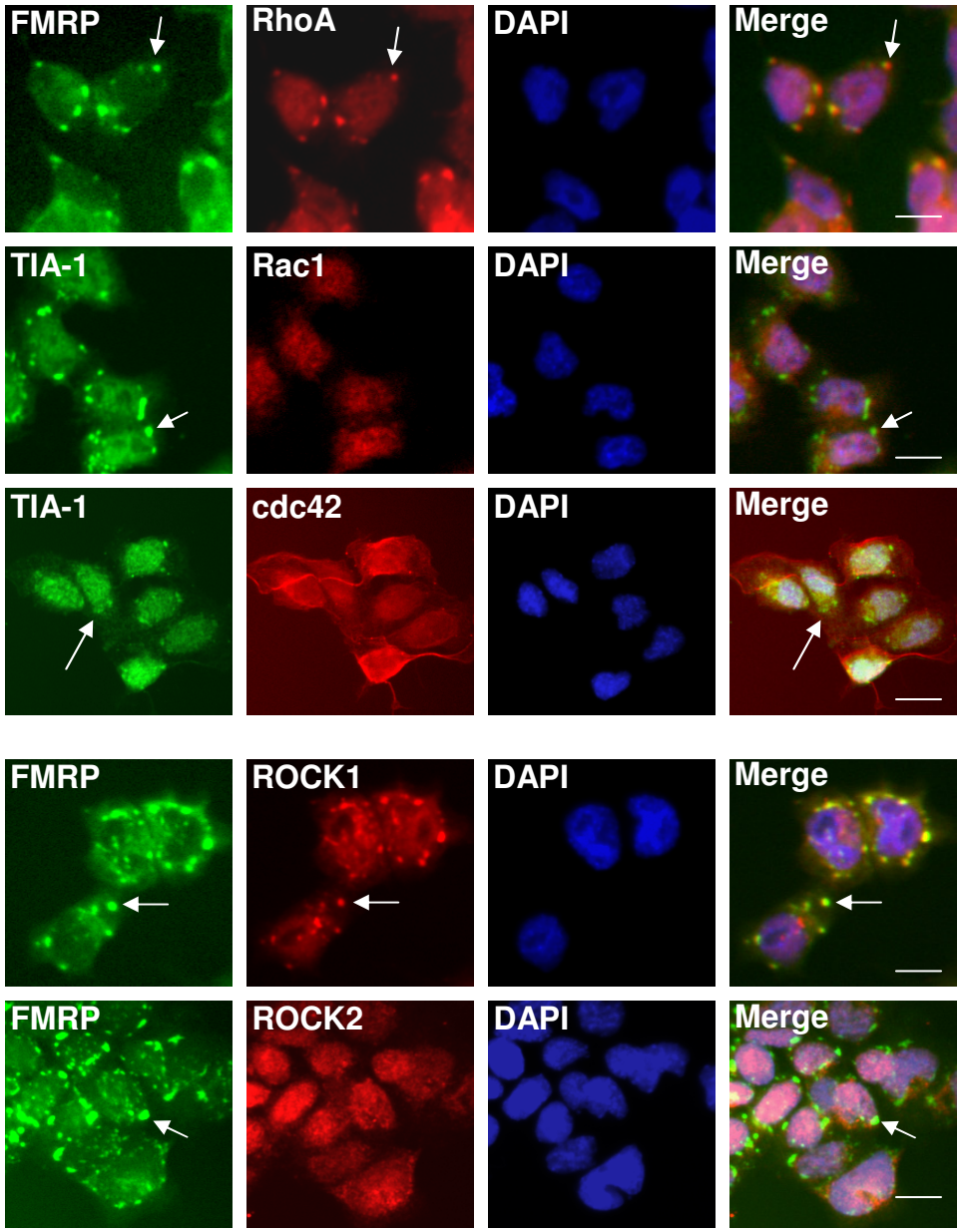
Supplemental Figure S1



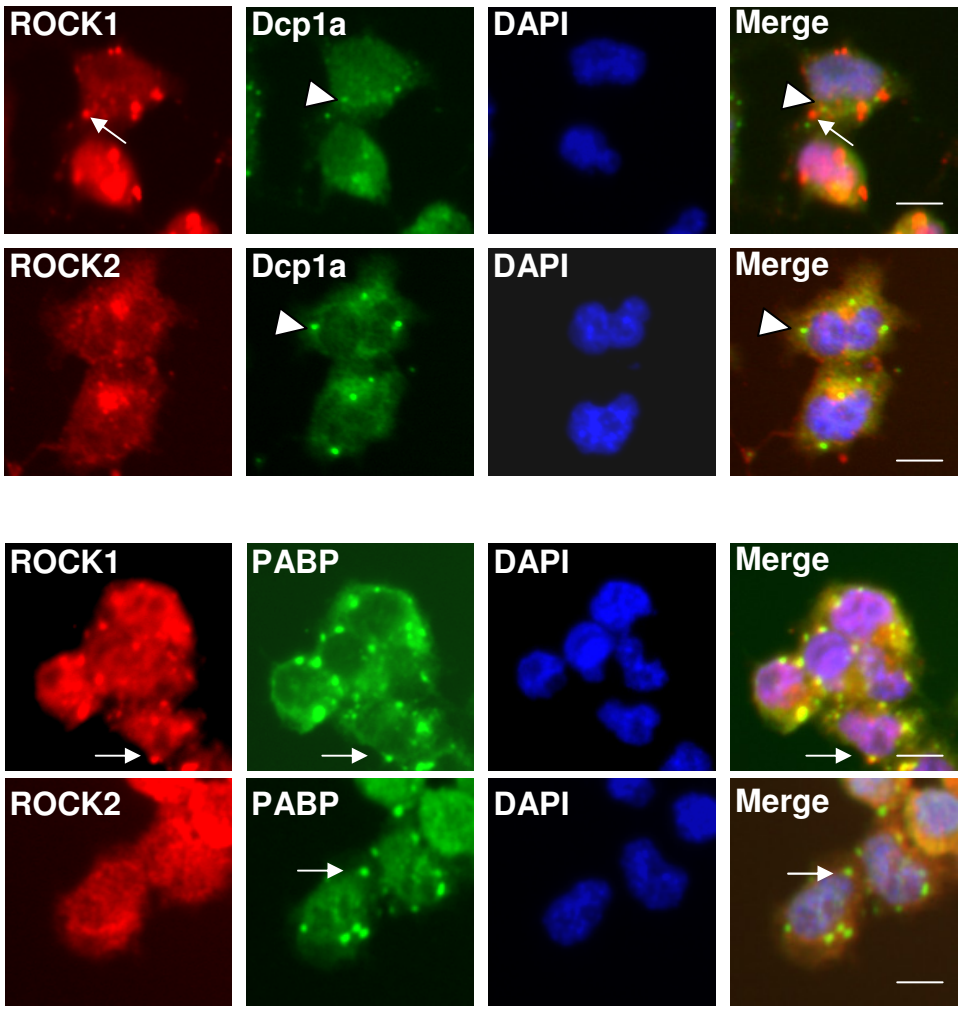
Supplemental Figure S1. RhoA and ROCK1 co-localize with another SG component, TIA-1, during heat shock stress. Bars, 25 µm.

Supplemental Figure S2



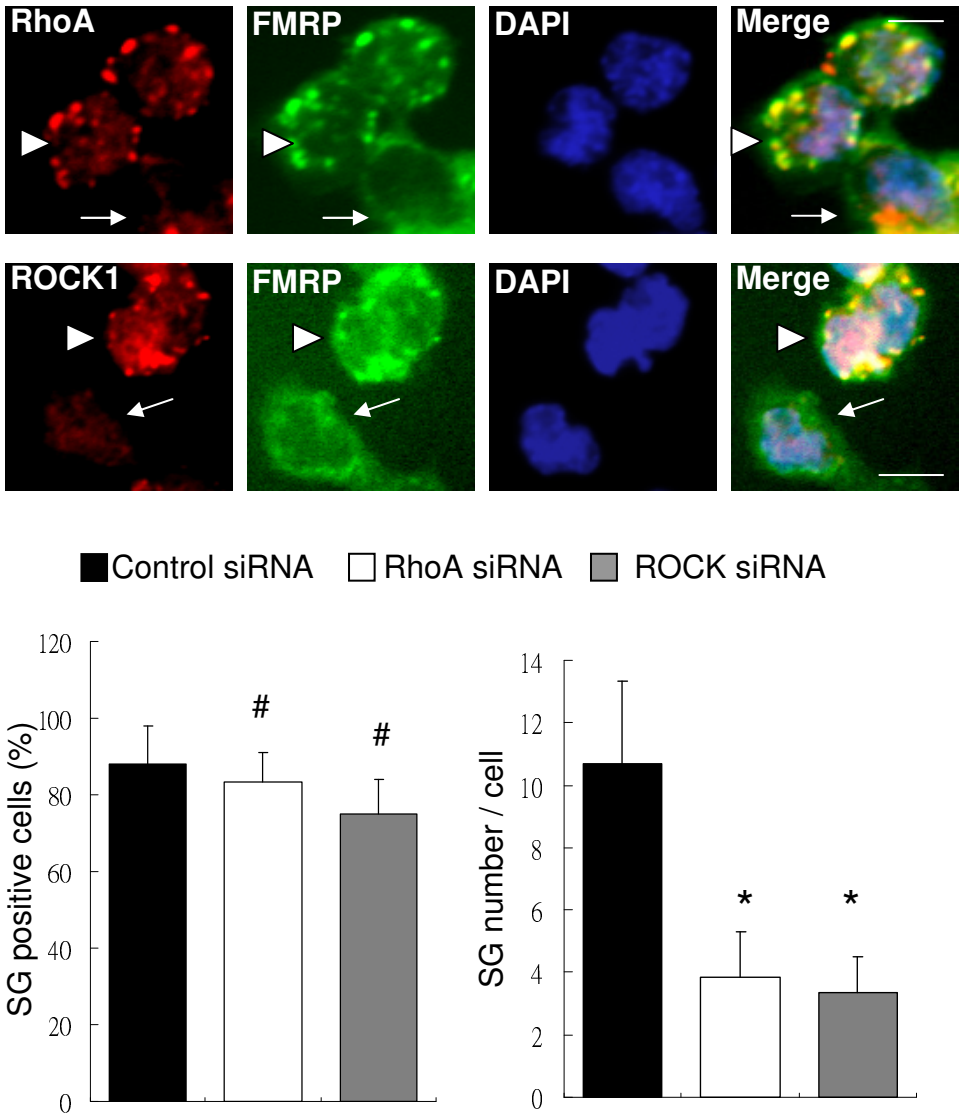
Supplemental Figure S2. Immunohistochemistry with antibodies indicated shows alternative stress induction, arsenite treatment, causes SG formation similar to heat shock treatment. Clear granules are marked by arrows. Bars, 25 µm.

Supplemental Figure S3



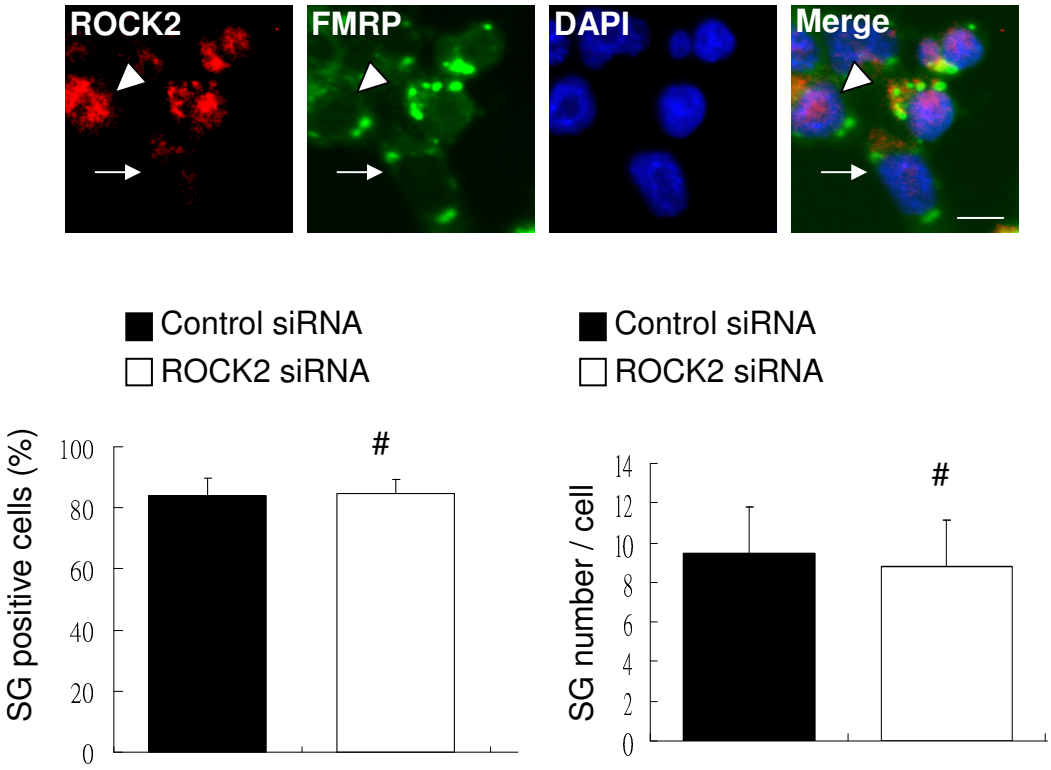
Supplemental Figure S3. ROCK1 shows colocalization with another SG component, poly-A binding protein (PABP), but not with P-bodies marker protein, decapping enzyme homolog 1a (Dcp1a). ROCK2 does not show colocalization with either PABP or Dcp1a. Clear SGs or PBs were indicated by arrows and arrow heads respectively. Bars, 25 μm.

Supplemental Figure S4



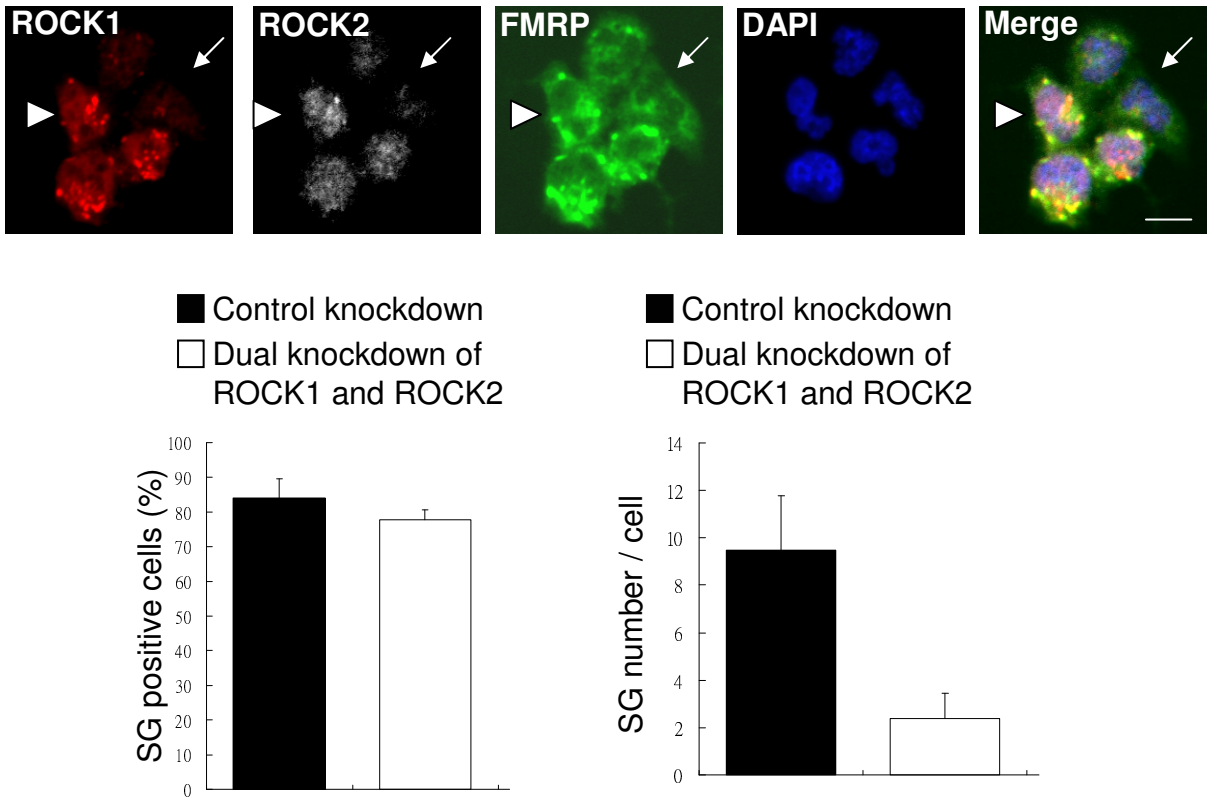
Supplemental Figure S4. Arsenite-treated, RhoA (top) or ROCK1 (bottom) silenced cells, show similar effect on SG formation compared to heat shock treatment. (*$P < 0.05$; #$P > 0.05$). Efficiently and inefficiently silenced cells were marked by arrows and arrow heads, respectively. Bars, 25 μm.

Supplemental Figure S5



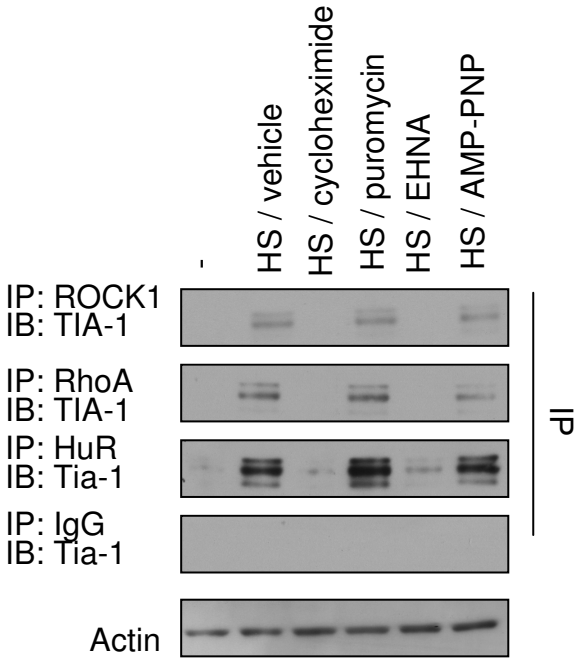
Supplemental Figure S5. Silencing ROCK2 does not affect SG formation. (#$P > 0.05$). Efficiently and inefficiently silenced cells were marked by arrows and arrow heads, respectively. Bars, 25 μm.

Supplemental Figure S6



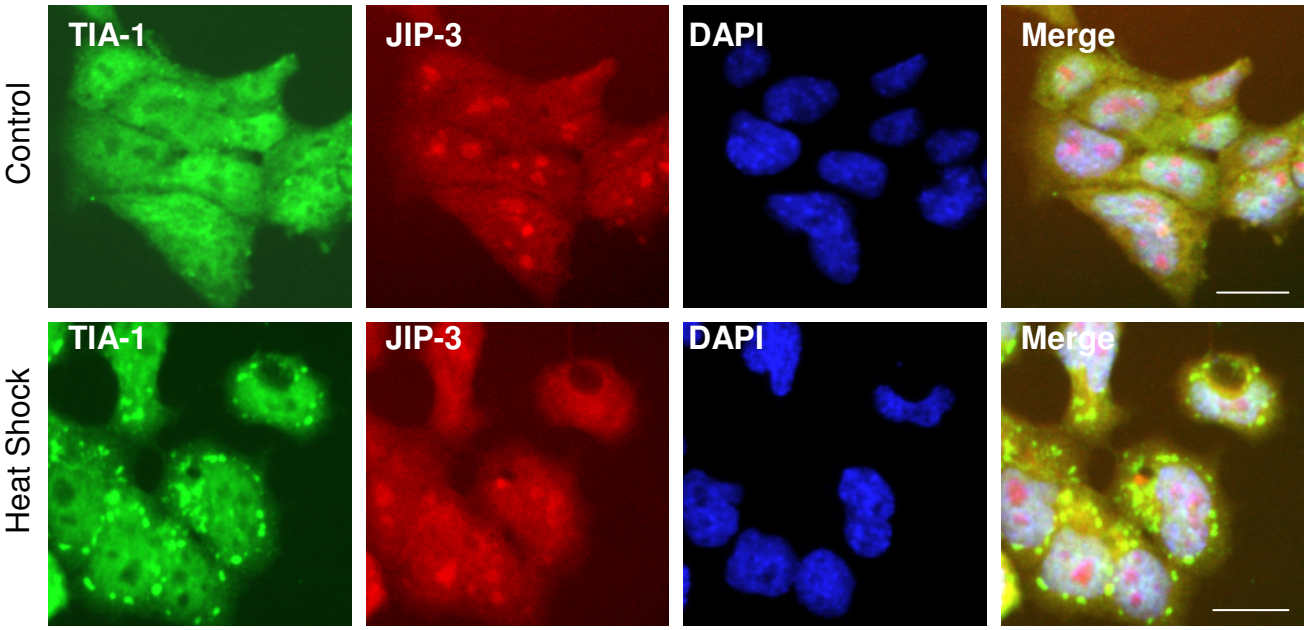
Supplemental Figure S6. Dual knockdown of ROCK1 and ROCK2 has similar effect as single silencing ROCK1 regarding SG formation. Efficiently and inefficiently silenced cells were marked by arrows and arrow heads, respectively. Bars, 25 µm.

Supplemental Figure S7



Supplemental Figure S7. Blocking SG formation with cycloheximide, EHNA but not puromycin, AMP-PNP, inhibits the interaction between TIA-1 with RhoA, ROCK1 or HuR.

Supplemental Figure S8



Supplemental Figure S8. JIP-3 is not colocalized with SG marker protein, TIA-1, during heat shock stress. Bars, 25 µm.