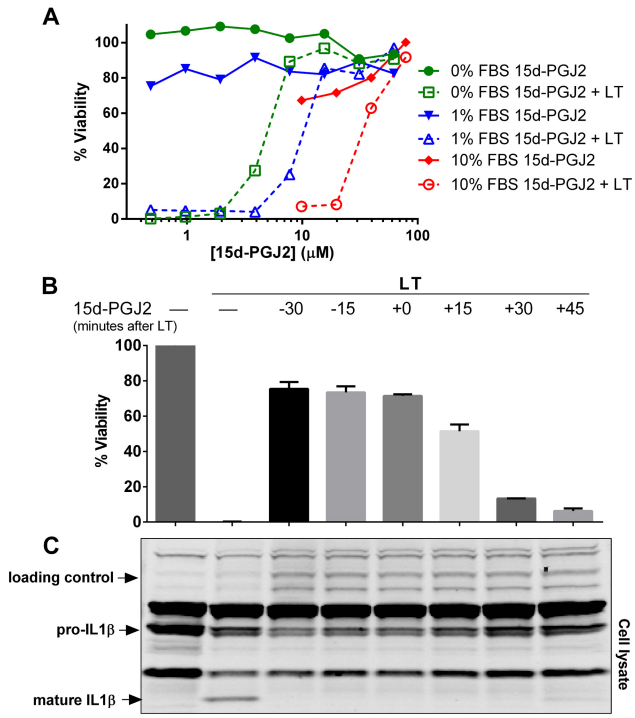
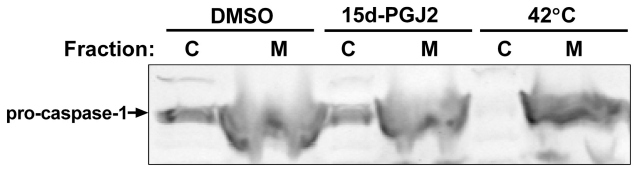
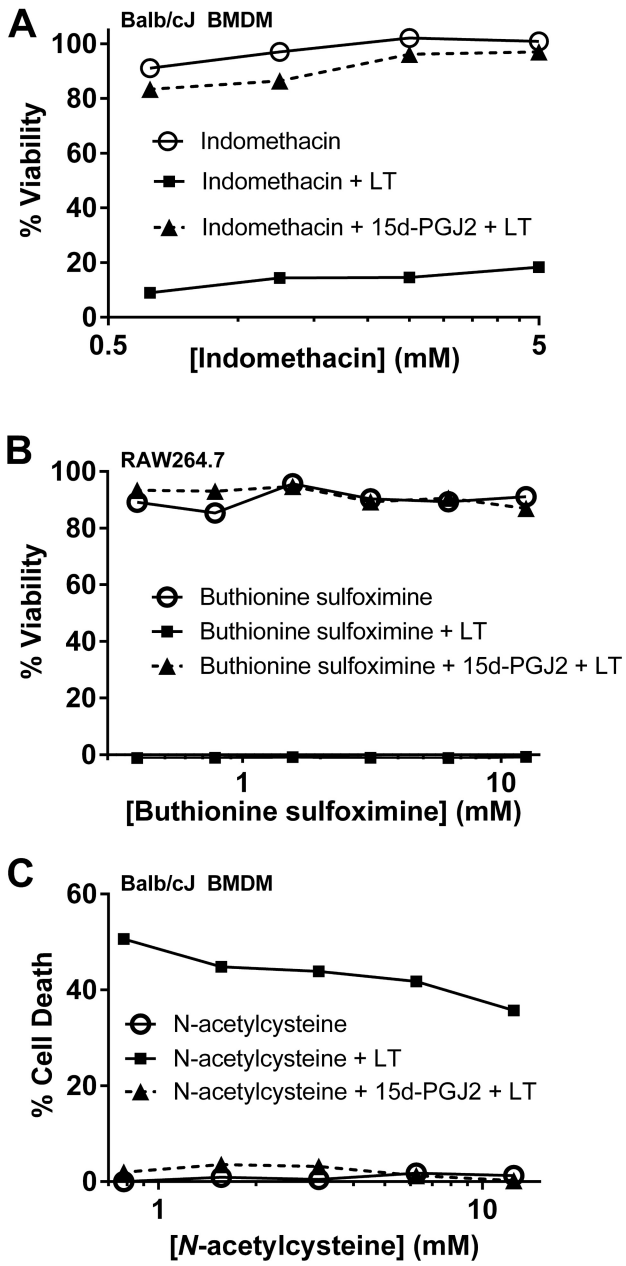


Supplemental Figure 1. 15d-PGJ2 is inactivated by FBS and inhibits the inflammasome at late timepoints. (A) Balb/cJ BMDMs were treated with variable concentrations of 15d-PGJ2 (30 min) in the presence of 10, 1, or 0 % FBS. Cells were challenged with LT (1 μg/mL, 2 h) and cell viability measured by MTT staining. (B) 15d-PGJ2 (50 μM) was applied to RAW264.7 cells at various timepoints before (-) or after (+) LT treatment. Cell viability was assessed at 2 h post-LT challenge by MTT staining. Each condition was assayed in duplicate in an experiment. Shown curves are representative of at least two or more experiments. In (B), error bars represent standard error of the mean. (C) RAW264.7 cells were primed with LPS (1 μg/mL, 2 h). 15d-PGJ2 (50 μM) was applied to cells at various timepoints before (-) or after (+) LT treatment. Cell lysates made at 1.5 h post-LT intoxication were analyzed for IL-1β maturation by Western blot.



Supplemental Figure 2. The mechanism of inflammasome inhibition by 15d-PGJ2 differs from that of heat shock. RAW264.7 cells were incubated with 15d-PGJ2 (50 μM) or heat shocked (42°C) for 1.5 h. Sucrose lysates were separated by centrifugation at 10,000 x g for 10 min at 4°C. Cytosolic (C) and membrane (M) fractions were analyzed for caspase-1 content by Western blot.



Supplemental Figure 3. Cyclooxygenase or glutathione manipulation has no effect on 15d-PGJ2-mediated protection from pyroptosis. RAW264.7 cells or Balb/cJ BMDMs were treated with variable concentrations of (A) indomethacin (4 h), (B) buthionine sulfoximine (2 h), or (C) N-acetylcysteine (2 h). 15d-PGJ2 (50 μM, 30 min) was added before LT (1 μg/mL, 2 h) challenge. Cell viability was analyzed by MTT staining, and cell death was analyzed by propidium iodide staining. Each condition was assayed in triplicate. Shown results are representative of two or more similar experiments.