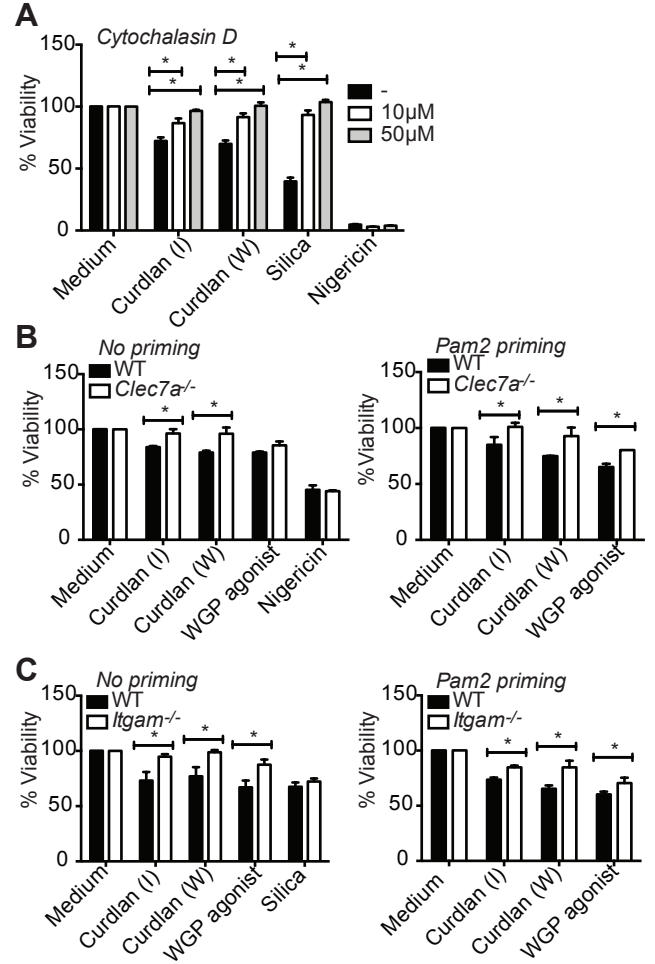

Figure S1. Phagocytosis of β-glucans and the receptors dectin-1 and CR3 trigger β-glucan induced cell death. Unprimed or Pam2CSK4 primed BMDC from C57BL/6 (A-C), *Clec7a*$^{-/-}$ (B) or *Itgam*$^{-/-}$ (C) mice were stimulated with curdlan (I or W), WGP agonist, silica for 6h or nigericin for 1h. Cell viability was assessed by cell titer-glo assay. In panel A, Pam2 primed cells were pretreated with the indicated concentrations of cytochalasin D prior to stimulating them with the indicated ligands. Statistical analysis was performed on results in all panels as described in the Materials and Methods section.

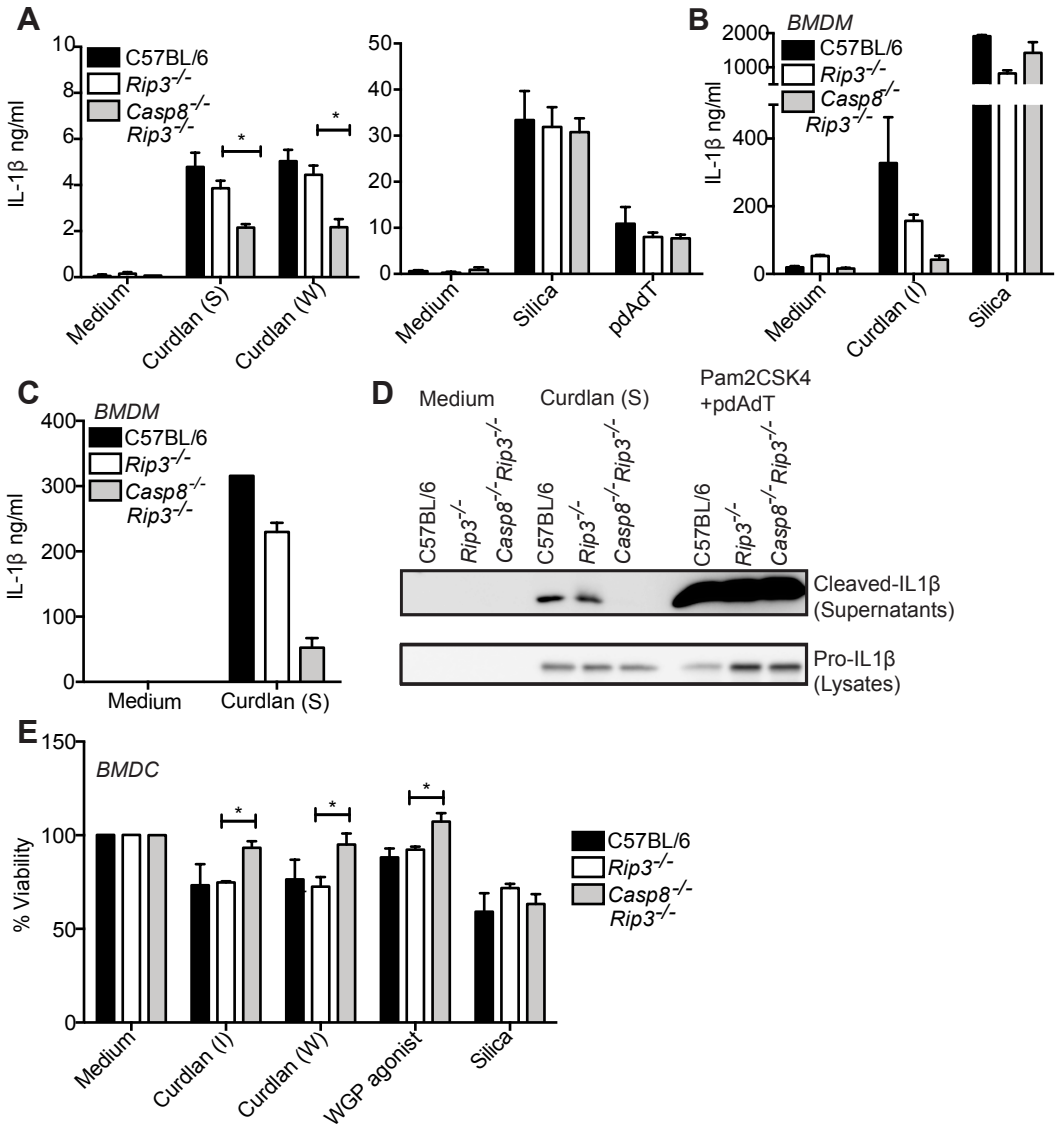

Figure S2. Caspase-8 mediates IL-1β production and cell death in response to curdlan from different sources. Pam2CSK4 primed (A-B, E) or unprimed (C-D) BMDC or BMDM from C57BL/6, *Rip3*$^{-/-}$, *Casp8*$^{-/-}$*Rip3*$^{-/-}$ mice were stimulated with curdlan (I,W or S) for 6h (BMDC) or 24h (BMDM) and silica or pdAdT for 6h. IL-1β release in the supernatants was measured by ELISA (A-C). Precipitated supernatants and cell lysates were probed for IL-1β (D). Cell viability was measured by cell-titer glo assay (E). Statistical analysis was performed on results in panels A and E as described in the Materials and Methods section.

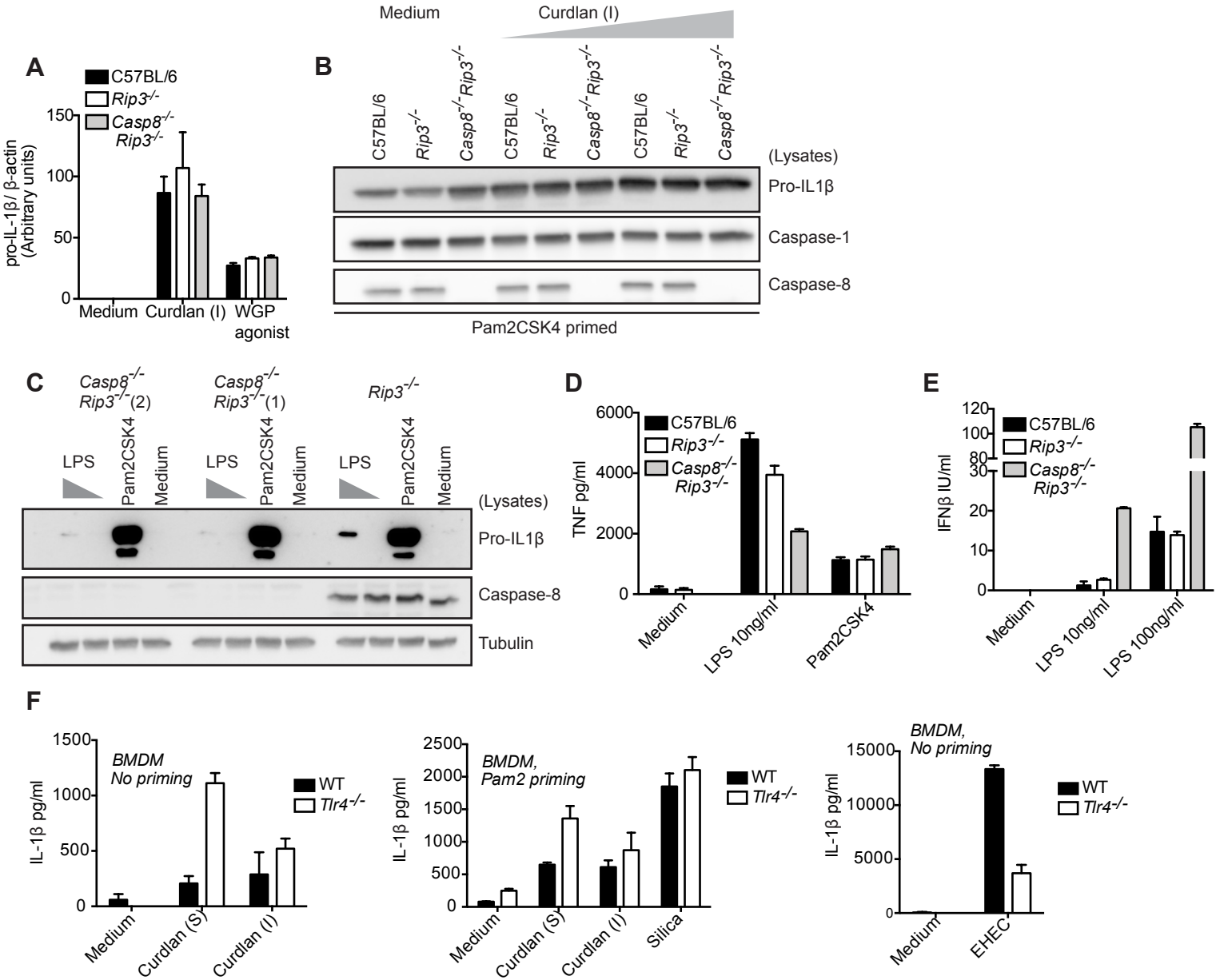

Figure S3. Role of caspase-8 in TLR signaling does not affect β-glucan induced IL-1β. Unprimed BMDC from C57BL/6, *Rip3*$^{-/-}$, *Casp8*$^{-/-}$*Rip3*$^{-/-}$ mice were stimulated with β-glucan ligands for 4h and pro-IL1β mRNA levels was measured by qRT-PCR (A). Pam2CSK4 primed cells were stimulated with the indicated ligands for 6h and cell lysates were probed for IL-1β, caspase-1 and caspase-8 (B). Unprimed BMDC were stimulated with LPS, Pam2CSK4 for 6h, pro-IL-1β, caspase-8 and tubulin were detected in the cell lysates by western blotting (C), TNF and IFNβ released in the supernatants was measured by ELISA (D, E). Unprimed or Pam2CSK4 primed BMDM from WT or *Tlr4*$^{-/-}$ mice were stimulated with the indicated ligands for 24h and IL-1β levels were measured in the supernatants by ELISA (F).

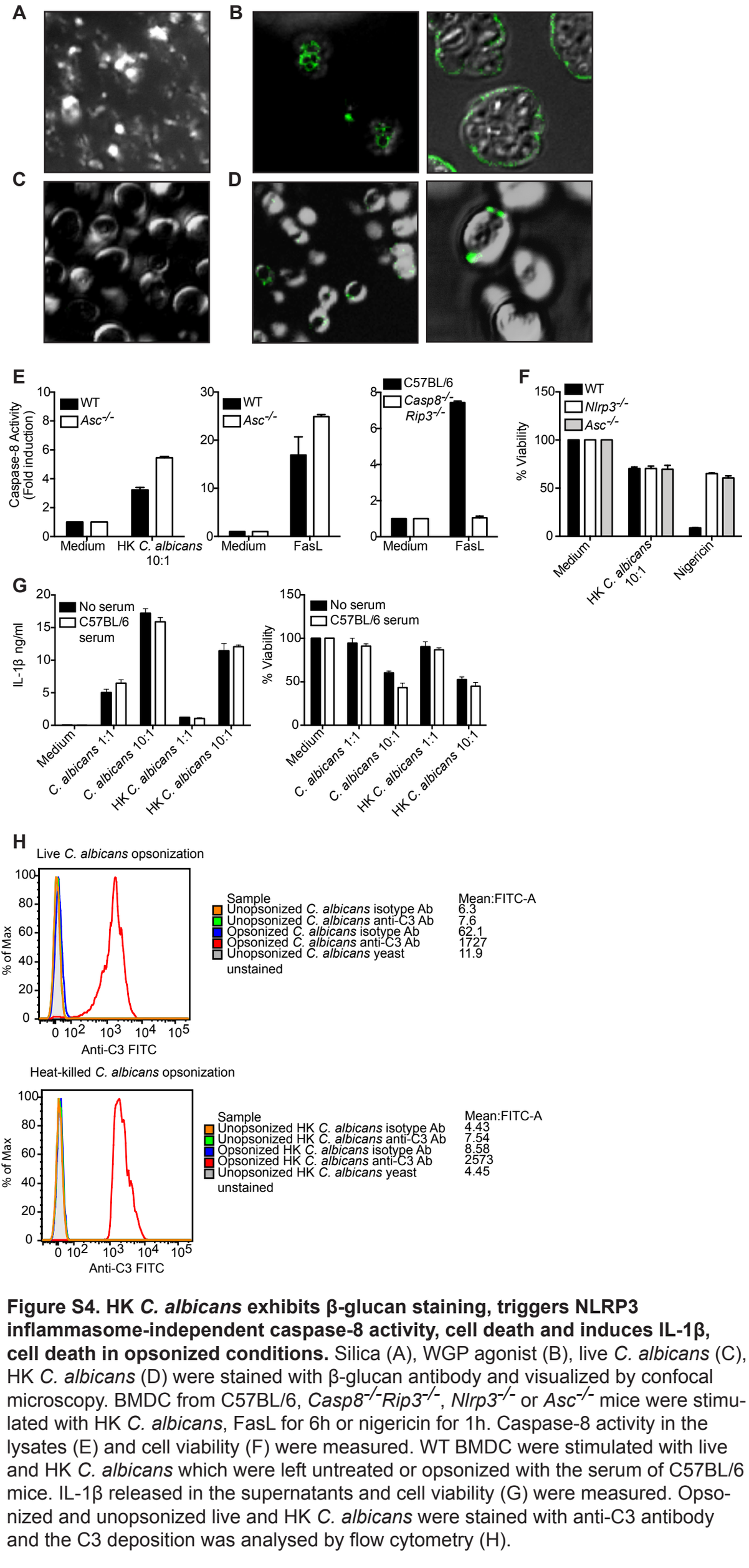

Figure S4. HK *C. albicans* exhibits β-glucan staining, triggers NLRP3 inflammasome-independent caspase-8 activity, cell death and induces IL-1β, cell death in opsonized conditions. Silica (A), WGP agonist (B), live *C. albicans* (C), HK *C. albicans* (D) were stained with β-glucan antibody and visualized by confocal microscopy. BMDC from C57BL/6, *Casp8*$^{-/-}$*Rip3*$^{-/-}$, *Nlrp3*$^{-/-}$ or *Asc*$^{-/-}$ mice were stimulated with HK *C. albicans*, FasL for 6h or nigericin for 1h. Caspase-8 activity in the lysates (E) and cell viability (F) were measured. WT BMDC were stimulated with live and HK *C. albicans* which were left untreated or opsonized with the serum of C57BL/6 mice. IL-1β released in the supernatants and cell viability (G) were measured. Opsonized and unopsonized live and HK *C. albicans* were stained with anti-C3 antibody and the C3 deposition was analysed by flow cytometry (H).