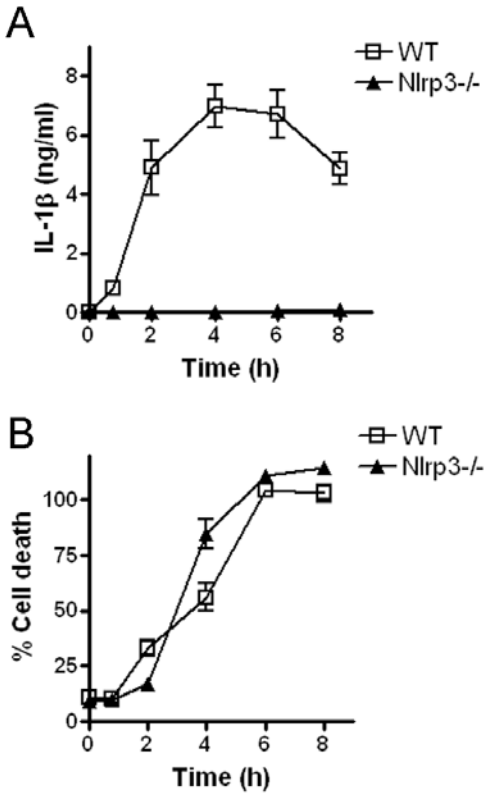
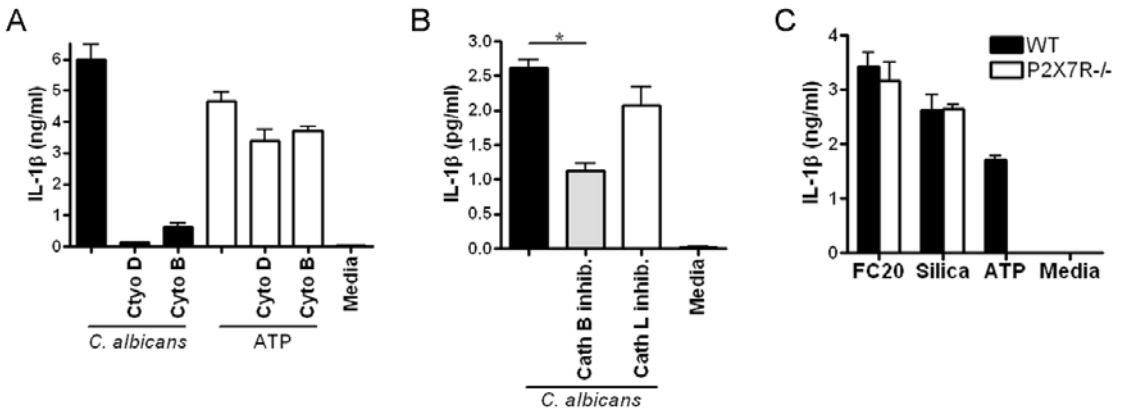
Joly et al. Supplemental Figure 1



SUPPLEMENTAL FIGURE 1. Mϕ IL-1β secretion but not cytotoxicity induced by *C. albicans* is dependent on the Nlrp3 inflammasome. LPS-primed Mϕ from WT or Nlrp3-deficient mice were stimulated for 6 h with *C. albicans* strain FC20 at an MOI of 1:10. Culture supernatants were collected and IL-1β secretion quantified by ELISA (*A*). Cytotoxicity was measured by LDH release and expressed as a percentage of LDH release by Triton X-100 detergent (*B*). Determinations were performed in triplicate and expressed as the mean ± SEM. Results are representative of two separate experiments.

Joly et al. Supplemental Figure 2



SUPPLEMENTAL FIGURE 2. Nlrp3 inflammasome activation is dependent on *C. albicans* internalization and cathepsin B activity. WT LPS-primed Mϕ treated with cytochalasin D (5 μM), cytochalasin B (10 μM) (*A*), cathepsin B inhibitor CA-074-Me (10 μM) or cathepsin L inhibitor V (10 μM) (*B*) were infected with *C. albicans* strain FC20 at an MOI of 1:10 or challenged with 5 mM ATP. 30 min after ATP challenge media was replaced with fresh media containing the indicated inhibitor. IL-1β release into culture supernatants 6 h after challenge was measured by ELISA. *C*, WT or P2X7R-deficient LPS-primed Mϕ were infected with *C. albicans* strain FC20 at an MOI of 1:10 or challenged with 5 mM ATP or silica (50 $\mu g/cm^2$). IL-1β release into culture supernatants 6 h after challenge was measured by ELISA. Determinations were performed in triplicate and presented as the mean ± SEM. Results are representative of two separate experiments. * $p = 0.001$ by two-tailed unpaired Student's t test.