

Figure S1. Expression of SIRT1 in HTR-8/SVneo cells exposed to LPS. (**A**) HTR-8/SVneo cells were treated with the indicated concentration of LPS for 4 h. Cell lysates were immunoblotted with an anti-SIRT1 antibody. α -Tubulin served as the loading control. Band intensities were quantified and normalized to the α -tubulin levels. (**B**) HTR-8/SVneo cells were treated with 200 ng/mL LPS for the indicated time. Cell lysates were immunoblotted with an anti-SIRT1 antibody. β -Actin served as the loading control. Numbers below the immunoblot bands indicate the fold changes normalized to the β -actin bands.

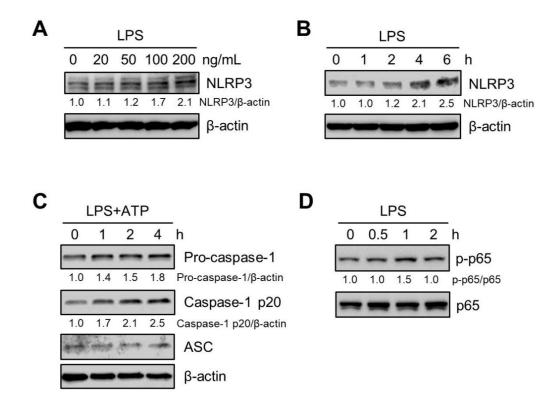


Figure S2. NLRP3 inflammasome activation in HTR-8/SVneo cells exposed to LPS. (A) HTR-8/SVneo cells were treated with the indicated concentration of LPS for 4 h. Cell lysates were immunoblotted with an anti-NLRP3 antibody. β -Actin served as the loading control. Numbers below the immunoblot bands indicate the fold changes normalized to the β -actin bands. (B,D) HTR-8/SVneo cells were treated with 200 ng/mL LPS for the indicated time. Cell lysates were immunoblotted with an anti-NLRP3, anti-p-p65 NF- κ B, and anti-p65 NF- κ B antibodies. (C) HTR-8/SVneo cells were treated with 200 ng/mL LPS for the indicated time and then followed by ATP (5 mM) treatment for 45 min. Cell lysates were immunoblotted with anti-Caspase-1 and anti-ASC antibodies. β -Actin or p65 served as the loading control. Numbers below the immunoblot bands indicate the fold changes normalized to the control bands.

Cells **2020**, 9, 728

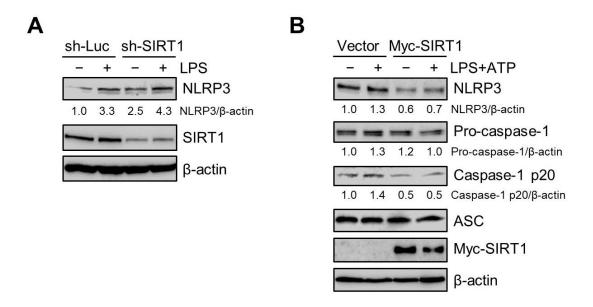


Figure S3. SIRT1 inhibits LPS-induced NLRP3 inflammasome activation (A) HTR-8/SVneo cells were infected with lentiviruses expressing shRNAs targeting luciferase (sh-Luc) or SIRT1 (sh-SIRT1) and then treated with 200 ng/mL LPS for 10 h. Cell lysates were immunoblotted with anti-NLRP3 and anti-SIRT1 antibodies. (B) Sw.71 cells were transfected with pcDNA3-myc-SIRT1 or pcDNA3 (empty vector) and treated with LPS (100 ng/mL) for 24 h and then followed by ATP (5 mM) treatment for 45 min, or were treated with vehicle. Cell lysates were immunoblotted with anti-NLRP3, anti-Caspase-1, anti-ASC, and anti-myc antibodies. β -Actin served as the loading control. Numbers below the immunoblot bands indicate fold changes normalized to the β -actin bands.

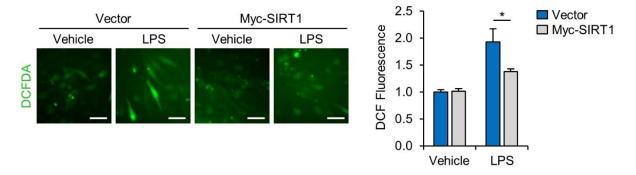


Figure S4. SIRT1 overexpression suppresses LPS-induced oxidative stress in Sw.71 cells. Sw.71 cells were transfected with pcDNA3-myc-SIRT1 or pcDNA3 (empty vector) and then treated with LPS (100 ng/mL) for 6 h. Reactive oxygen species (ROS) levels were analyzed using CM-H₂DCFDA. Scale bars, 50 μ m. Data are indicated as mean \pm SEM. Results are representative of at least three independent experiments. * p < 0.05 (Student's t-test).