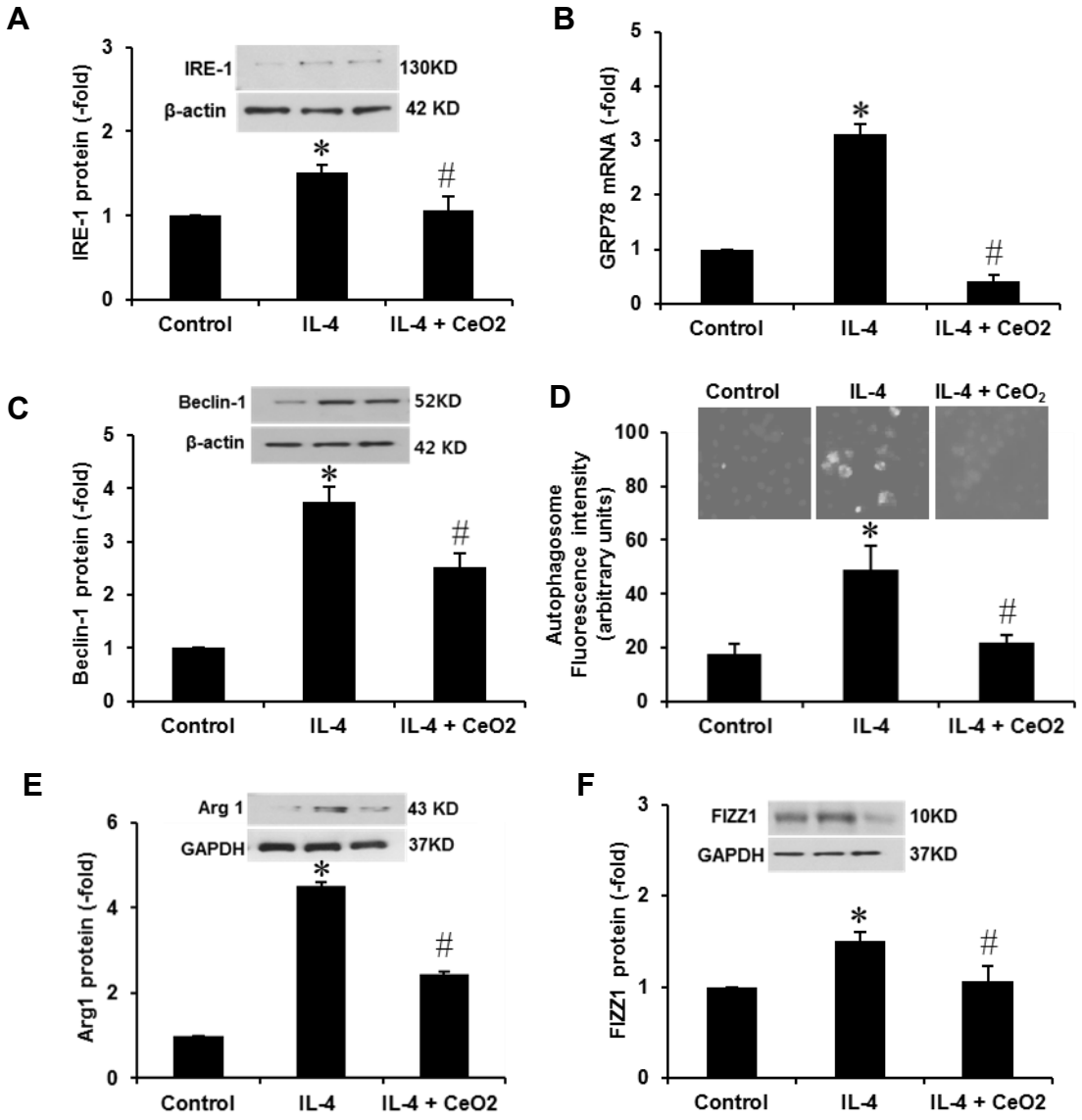

Figure S1. Inhibition of oxidative stress with CeO_2 nanoparticles inhibited ER stress, autophagy and M2 polarization. Mouse macrophages were preincubated with 1μm CeO_2 nanoparticles for 6 hr, and then treated with 20ng/ml IL-4 for 4 hr. Blockage of IL-4-induced ROS production by antioxidant CeO_2 resulted in suppression of IL-4-mediated ER stress as measured by expression of IRE-1 (**A**) and GRP78 (**B**), autophagy as measured by expression of Beclin-1 (**C**) and autophagosome staining (**D**). Furthermore, blockage of IL-4-induced ROS production by CeO_2 resulted in suppression of expression of M2 markers Arg1 (**E**) and FIZZ1 (**F**), $^{*}P < 0.05$ *vs* Control, $^{\#}P < 0.05$ *vs* IL-4 treatment. Experiments were repeated 3 times. Six different fields containing at least 200 cells were analyzed for autophagosome staining.

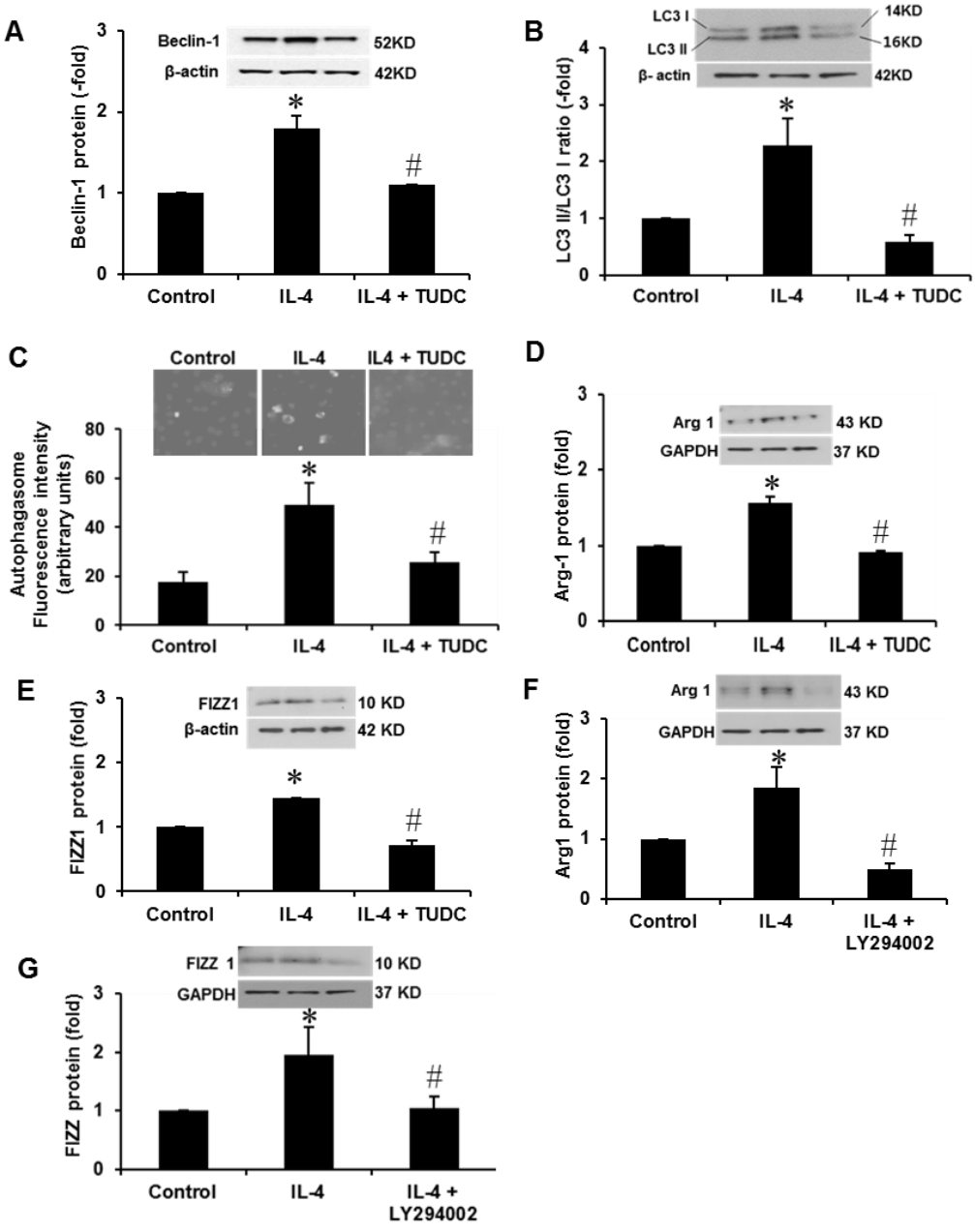

Figure S2: Inhibition of ER stress and autophagy inhibited M2 polarization. Mouse macrophages were preincubated with 100μm TUDC or LY294002 for 6 hr, and then treated with 20ng/ml IL-4 for 4 hr. Inhibition of IL-4-induced ER stress by TUDC resulted in inhibition of IL-4-induced autophagy as measured for Beclin-1(**A**), LC3II:LC3I ratio (**B**) by immunoblots and autophagosome formation (**C**), and expression of M2 markers Arg1 (**D**) and FIZZ1 (**E**) assayed by immunoblot. Inhibition of autophagy by LY294002 resulted in inhibition of IL-4 induced expression of M2 markers Arg1 (**F**) and FIZZ1 (**G**) assayed by immunoblots. $*P < 0.005$ *vs* Control. $^{\#}P < 0.05$ *vs* IL-4 treatment. Experiments were repeated 3 times. Six different fields containing at least 200 cells were analyzed for autophagosome staining.

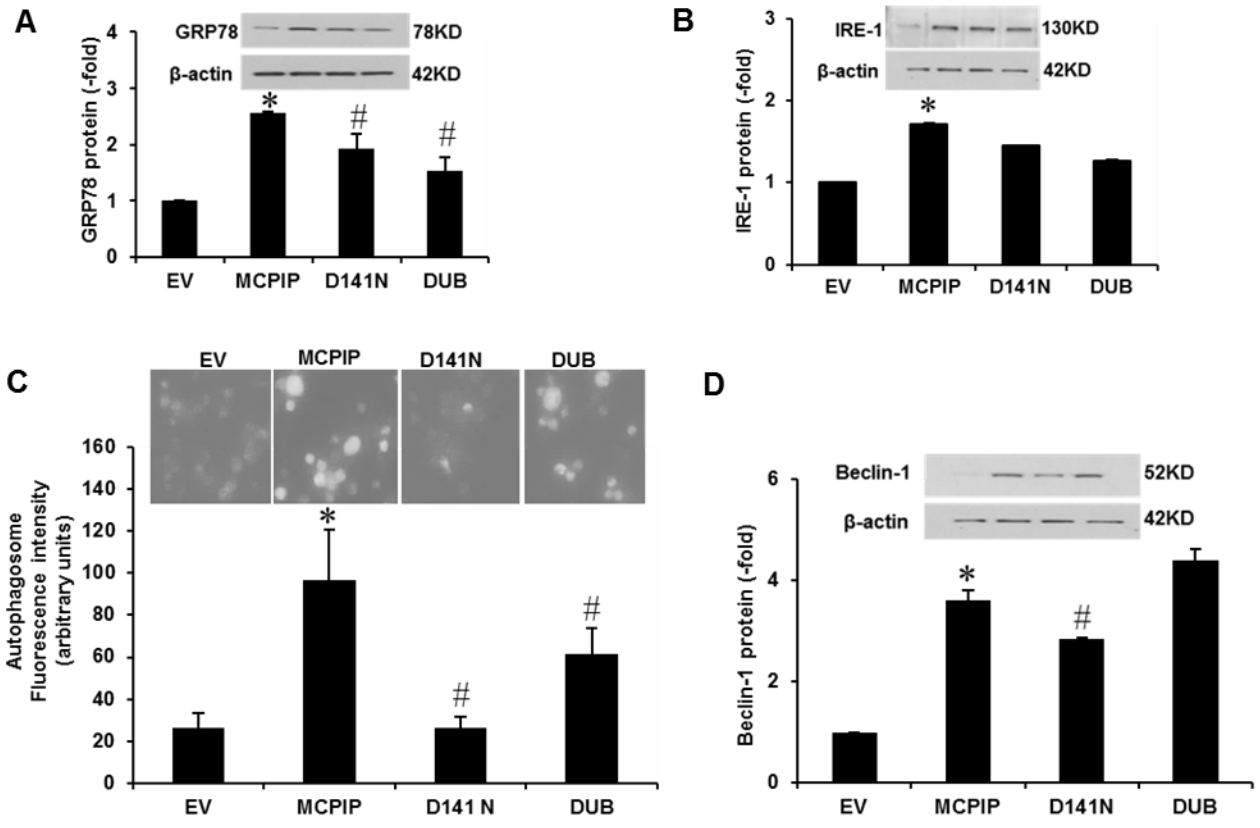

Figure S3: Dual activities of MCPIP are required for M2 polarization. Mouse macrophages were transfected with either empty vector (EV), or MCPIP expression plasmid, or expression vectors for D141N mutant, or DUB mutant for 48 hr. Induction of ER stress as measured by expression of GRP78 (**A**) and IRE-1 (**B**) protein levels and autophagy as measured by autophagosome formation (**C**) and Beclin-1 protein levels (**D**) by MCPIP showed that loss of either RNase or dequbiquitinase activity of MCPIP inhibited autophagosome formation but DUB mutation did not affect Beclin-1 or IRE-1 protein levels. *$P < 0.05$ *vs* EV; $^{\#}P < 0.05$ *vs* MCPIP. Six different fields containing at least 200 cells were analyzed for autophagosome staining.

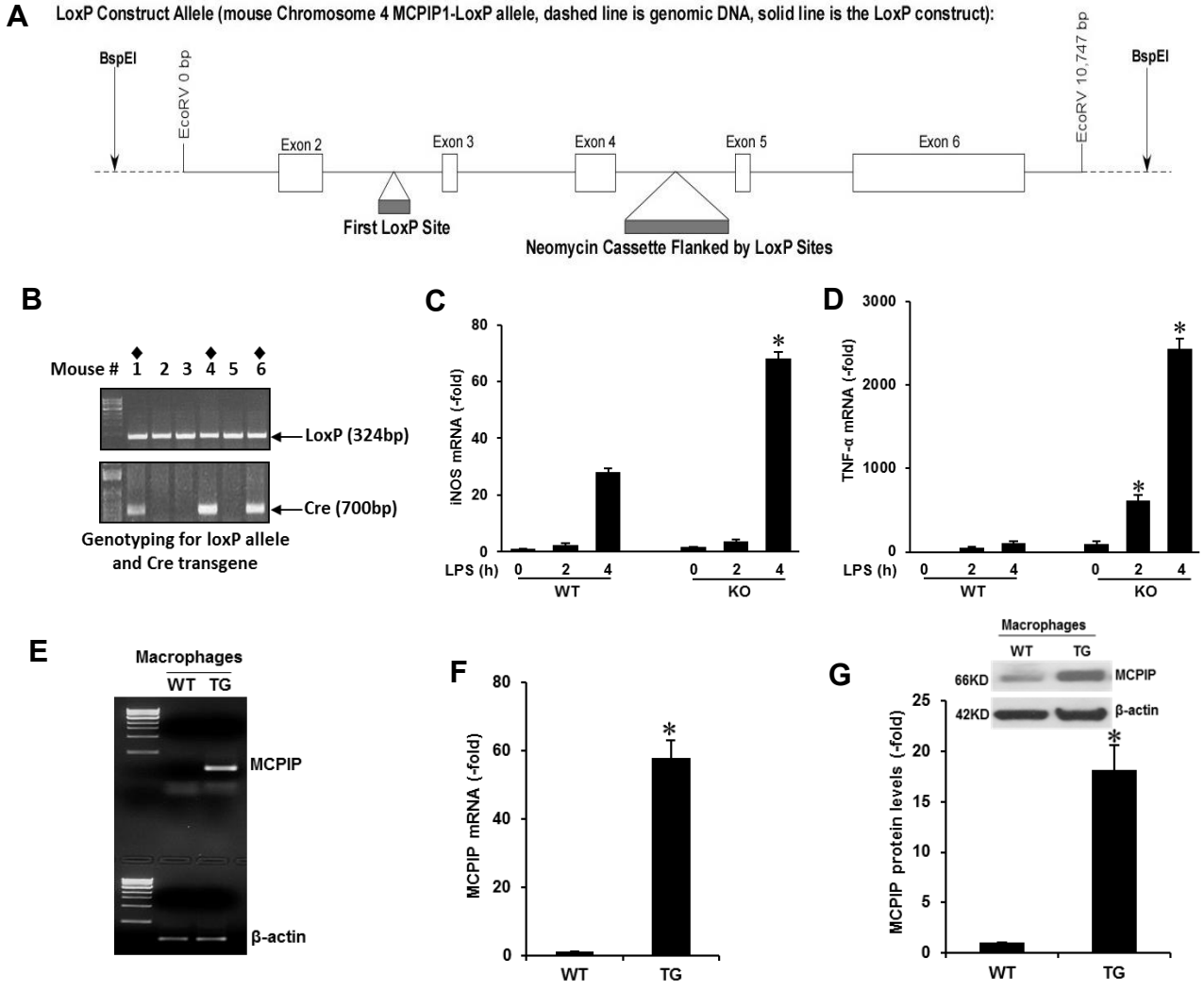

Figure S4: Generation of mice with macrophage-specific deletion or expression of MCPIP. The construct for generation of MCPIP-LoxP mice (**A**). The mice homozygotes for the loxP allele and positive for the Cre transgene were identified by PCR genotyping (**B**). Macrophages from myelo-KO and WT mice were treated with 100ng of LPS for the indicated periods and expression of M1 markers iNOS and TNF-α was measured by qRT-PCR (**C**, **D**). Macrophages from WT and myelo-MCPIP (TG) mice were analyzed for MCPIP transcript by RT-PCR (**E**) and qRT-PCR (**F**), and for MCPIP protein level by immunoblot (**G**). **P* < 0.05 *vs* WT (n = 3 per each genotype).