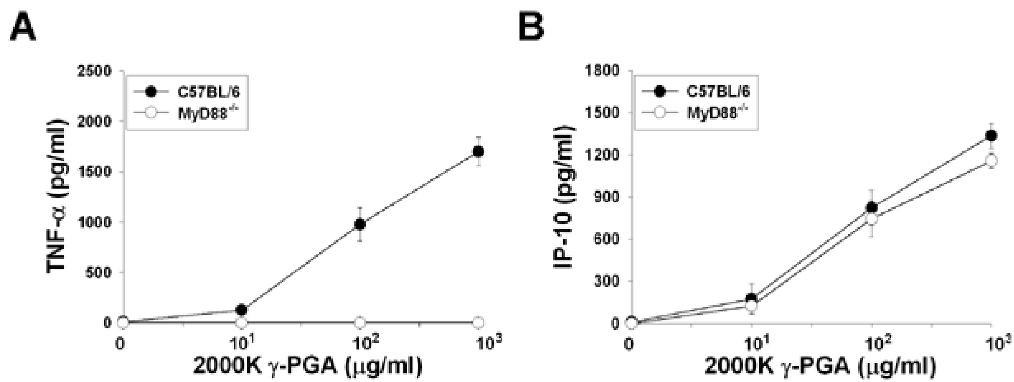
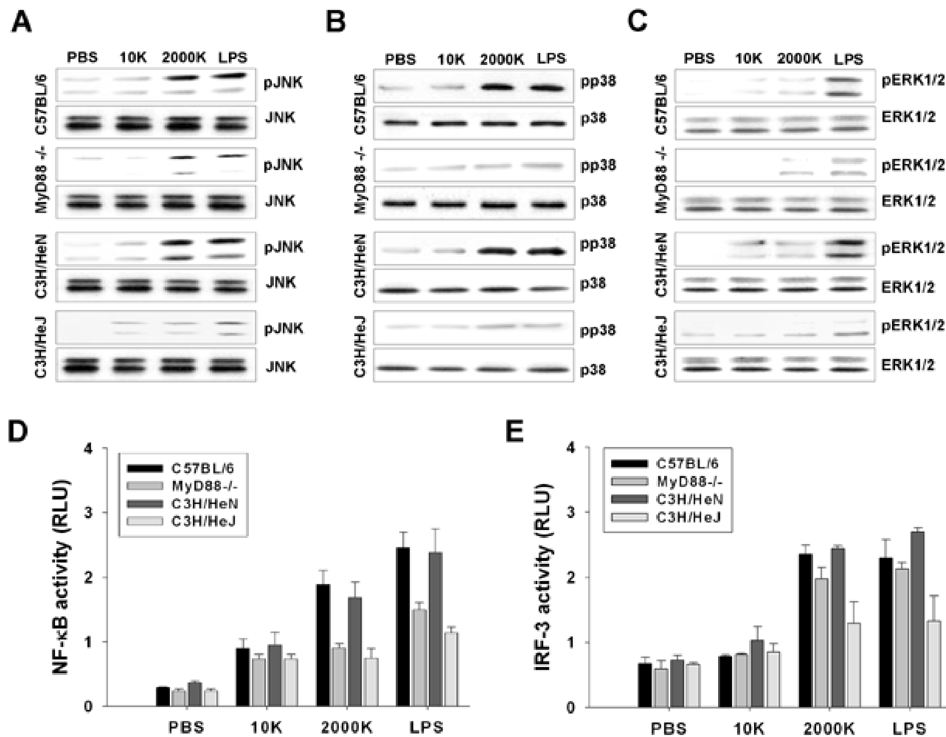
Suppl. Figure 1. Lee et al.



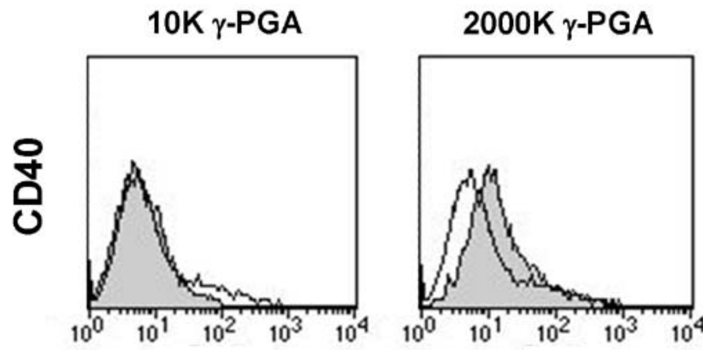
Supplementary figure 1. γ-PGA induced the activation of macrophage dose-dependently by MyD88-dependent and -independent pathways. BMDMs (1×10^6 cells/ml) isolated from wild-type or $MyD88^{-/-}$ mice were stimulated with γ-PGA for 24 h. Concentration of TNF-α **(a)** and IP-10 **(b)** in the culture supernatants were measured by ELISA. The results are expressed as means ± SE of three independent experiments.

Suppl. Figure 2. Lee et al.



Supplementary figure 2. Intracellular signaling induced by γ-PGA was MyD88 and TLR4-dependent. BMDCs isolated from C57BL/6, $MyD88^{-/-}$, C3H/HeN, or C3H/HeJ mice were treated with 100 ng/ml LPS and 1 mg/ml γ-PGA for 30 min. The expression level of phosphorylated JNK (pJNK) (**a**), p38 kinase (pp38) (**b**), and ERK1/2 (pERK1/2) (**c**) were determined by Western blot analysis. BMDCs isolated from C57BL/6, $MyD88^{-/-}$, C3H/HeN, or C3H/HeJ mice were transiently transfected with pNF-κB-Luc and IRF-3-Luc reporter gene plasmids and then cultured in complete medium for 24 h. At 24 hours after transfection, cells were treated with 100 ng/ml LPS for 1 h and 1 mg/ml γ-PGA for 2 h and then NF-κB activity (**d**), IRF-3 activity (**e**) were determined by luciferase reporter assay. The data presented in this figure are representative of triplicate experiments. All data are representative of at least three experiments.

Suppl. Figure 3. Lee et al.



Supplementary figure 3. Oral administration of γ-PGA induce dendritic cell activation *in vivo*. C57BL/6 mice (three mice per group) were treated with daily oral treatment of 400 μg γ-PGA (10 kDa or 2,000 kDa) and PBS. Intestinal DCs were collected 1 week after feeding PBS or γ-PGA. The level of CD40 expression on intestinal DCs was examined by flow cytometry. Filled histogram and solid line indicated γ-PGA fed mice and PBS control, respectively.