

## SUPPLEMENTARY FIGURE LEGENDS

FIG. S1. **Autophagy was critical for IFN- $\gamma$ -induced anti-proliferation and viral replication inhibition.** *A*, the number of cells was counted 72 h after IFN- $\gamma$  (10 ng/ml) treatment in WT and *Atg5*<sup>-/-</sup> MEFs. Data, obtained from triplicate cultures, are means  $\pm$  S.D. \*,  $p < 0.05$ . Bar, 100  $\mu$ m. *B*, levels of HSV-1 titers 7 days post-infection in IFN- $\gamma$  (10 ng/ml)-treated WT and *Atg5*<sup>-/-</sup> MEFs. pfu, plaque-forming units. Data, obtained from triplicate cultures, are means  $\pm$  S.D. \*,  $p < 0.05$ .

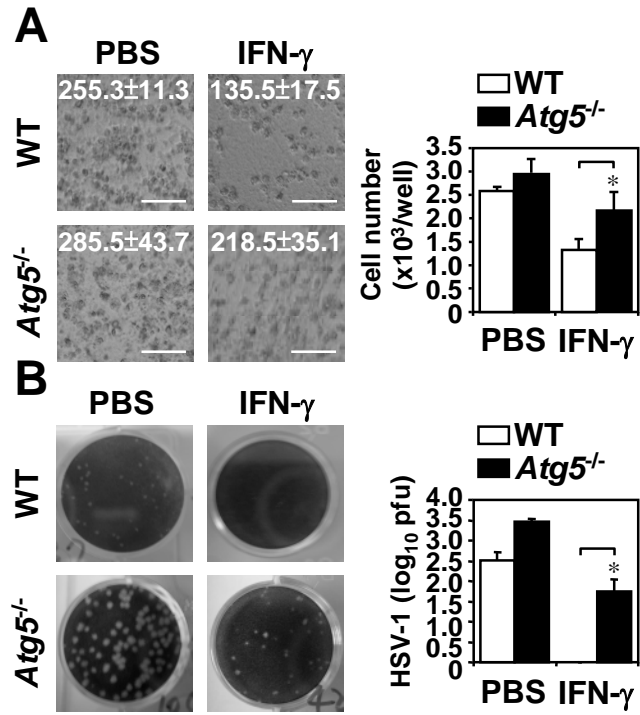
FIG. S2. **STAT1 expression decreased in the absence of autophagy.** *A*, flow cytometry was used to detect the expression of STAT1 $\alpha/\beta$ . The percentages of positive cells and mean fluorescence intensity (*MFI*) are shown. *B*, Western blotting was used to determine the expression of STAT1 $\alpha/\beta$  after pretreatment with (+) and without (-) MG132 (25  $\mu$ M).  $\beta$ -actin was the internal control. Data are representative of three individual experiments.

FIG. S3. **SHP2 mediated the inhibition of IFN- $\gamma$ -induced nitrite generation in the absence of autophagy.** Griess reagent was used to detect nitrite generation 48 h after IFN- $\gamma$  (10 ng/ml) treatment with (+) and without (-) 0.5 h of SHP2 inhibitor NSC-87877 (5  $\mu$ M) pretreatment in WT and *Atg5*<sup>-/-</sup> MEFs. Data, obtained from triplicate cultures, are means  $\pm$  S.D. \*,  $p < 0.05$ .

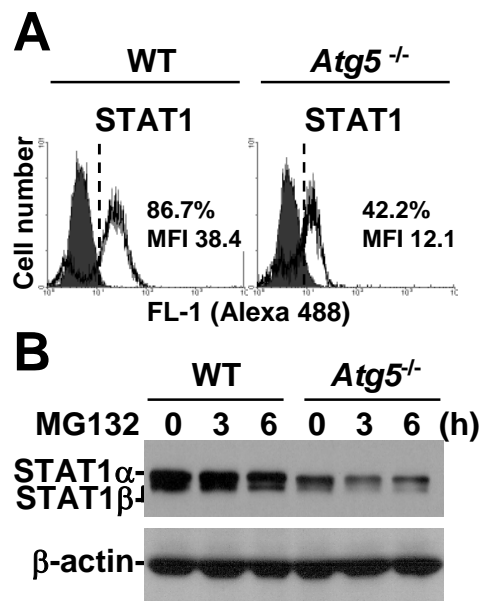
FIG. S4. **Autophagic stimuli and SHP2 inhibition facilitated IFN- $\gamma$ -induced STAT1 activation in K562 cells.** *A*, Western blotting was used to determine the time kinetic phosphorylation of STAT1 $\alpha/\beta$  (Y701) and LC3 conversion in IFN- $\gamma$  (10 ng/ml)-treated U937 and K562 cells. *B* and *C*, with (+) and without (-) 0.5 h of rapamycin (5  $\mu$ M) pretreatment or shSHP2-2 transfection, Western blotting was used to determine the phosphorylation of STAT1 $\alpha/\beta$  (Y701) and LC3 conversion in IFN- $\gamma$  (10 ng/ml)-treated K562 cells for 0.25 h.  $\beta$ -actin was the internal control. Data are representative of three individual experiments.

FIG. S5. **Autophagic stimuli facilitated IFN- $\gamma$ -induced STAT1 activation.** Western blotting was used to determine the phosphorylation of STAT1 $\alpha/\beta$  (Y701) after a 0.5 h-pretreatment with rapamycin (*RAP*, 5  $\mu$ M), starvation (*STA*), or LPS (1  $\mu$ g/ml), and then a 0.25-h treatment with IFN- $\gamma$  (10 ng/ml) in WT MEFs.  $\beta$ -actin was the internal control. Data are representative of three individual experiments.

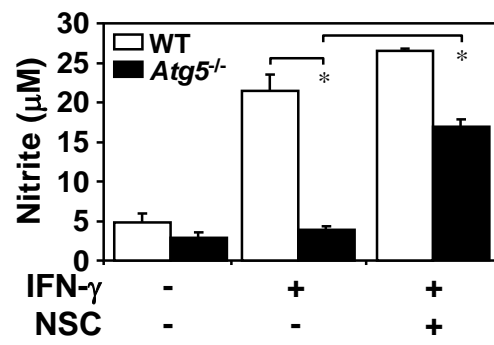
# Chang et al. Figure S1



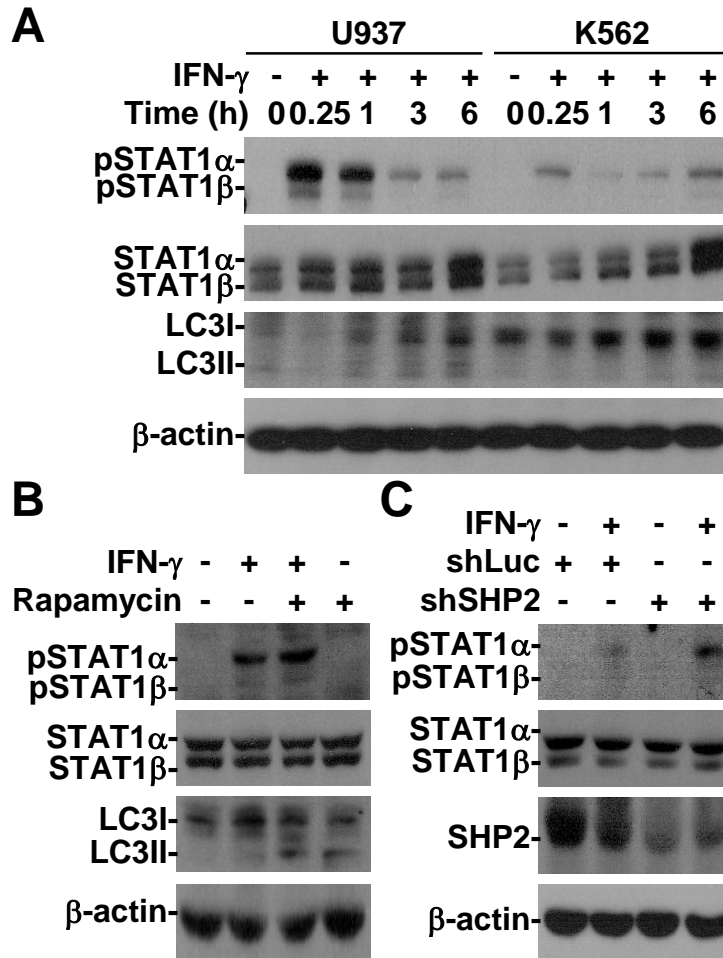
## Chang et al. Figure S2



### Chang et al. Figure S3



## Chang et al. Figure S4



## Chang et al. Figure S5

