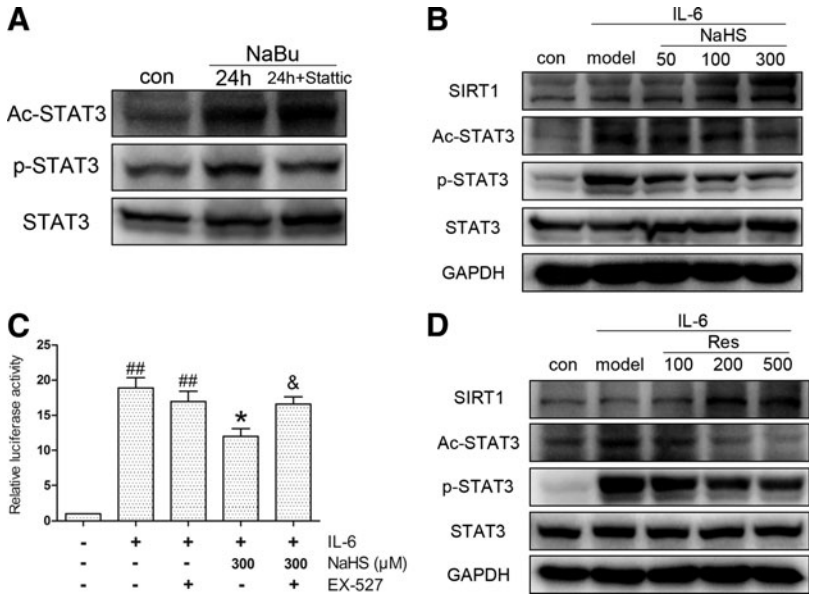

SUPPLEMENTARY FIG. S4. Evaluation of STAT3 acetylation and SIRT1 in the induction of hepcidin. **(A)** NaBu (1 m*M*), an HDAC inhibitor, increased the STAT3 acetylation level and added to STAT3 phosphorylation as well ($n = 3$). The induction in p-STAT3 was abolished by stattic (10 μM). **(B)** NaHS promoted SIRT1 expression and suppressed IL-6-induced STAT3 acetylation and phosphorylation in mouse primary hepatocytes ($n = 3$). **(C)** EX-527 (10 μM) blocked the inhibition of NaHS on STAT3 transcriptional function assessed by SIE reporter ($n = 5$). **(D)** Resveratrol pretreatment, similar to NaHS, promoted SIRT1 and inhibited STAT3 acetylation and phosphorylation irritated by IL-6 ($n = 3$). GAPDH served as the loading control. Representative immunoblots are presented. Data are represented as the mean ± SEM of three individual experiments. $^{\#\#}p < 0.01$ compared with the control group; $^{*}p < 0.05$ compared with the IL-6 group; $^{\&}p < 0.05$ compared with the IL-6 + NaHS group. GAPDH, glyceraldehyde-3-phosphate dehydrogenase; SIRT1, sirtuin 1.