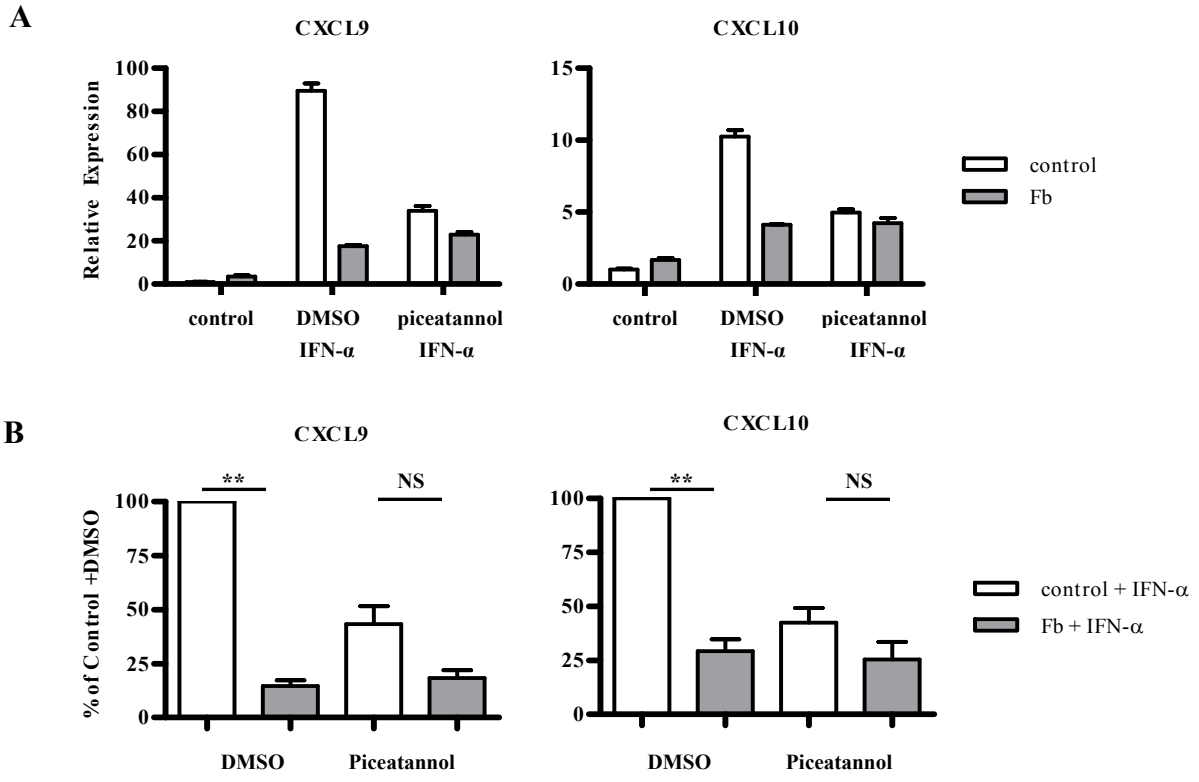

Figure S1. β2 integrin mediated inhibition of type I IFN signaling is Syk-dependent.

Primary human macrophages were pretreated with either the vehicle control DMSO or Syk inhibitor piceatannol (80 μM) for 30 min. Macrophages were then plated onto control wells or fibrinogen (Fb) coated wells for 2 h, and IFN-α (1000 U/mL) was added for an additional 2 h to assess induction of IFN-α response genes.

A, One representative donor is shown. mRNA expression was measured by qPCR, and results are presented as mean ± SD of triplicate wells normalized relative to GAPDH mRNA.

B, Cumulative data from 3 donors is presented as mean ± SEM. Transcript expression from cells plated onto control wells and given IFN-α in the DMSO condition was set as 100%, and expression in all other conditions were set relative to that control. In the control DMSO condition, inhibition of CXCL9 by Fb was statistically significant ($p = 0.0010$, paired Student t test); in the piceatannol condition however, difference between the control and Fb conditions was no longer significant ($p = 0.1237$, paired Student t test). Similarly, inhibition of CXCL10 by Fb in the control DMSO condition was statistically significant ($p = 0.0062$, paired Student t test); in the piceatannol condition however, difference between the control and Fb conditions was no longer significant ($p = 0.2029$, paired Student t test).

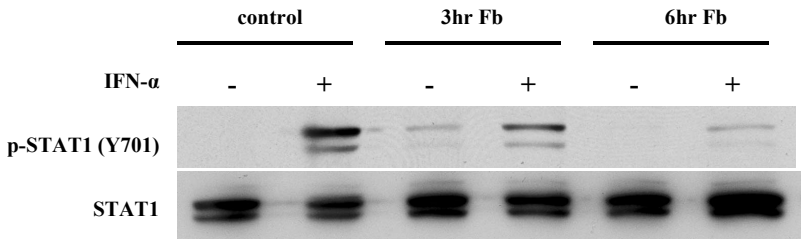

Figure S2. β2 integrin mediated inhibition of type I IFN signaling is time dependent.

Primary human monocyte-derived macrophages were plated onto control wells or fibrinogen (Fb) coated wells for 3 or 6 h, and then IFN-α (1000 U/mL) was added for an additional 15 min to assess phosphorylation of STAT1. Whole cell lysates were immunoblotted with Abs against phospho-STAT1 (Y701) and STAT1. Data are representative of 2 independent experiments.

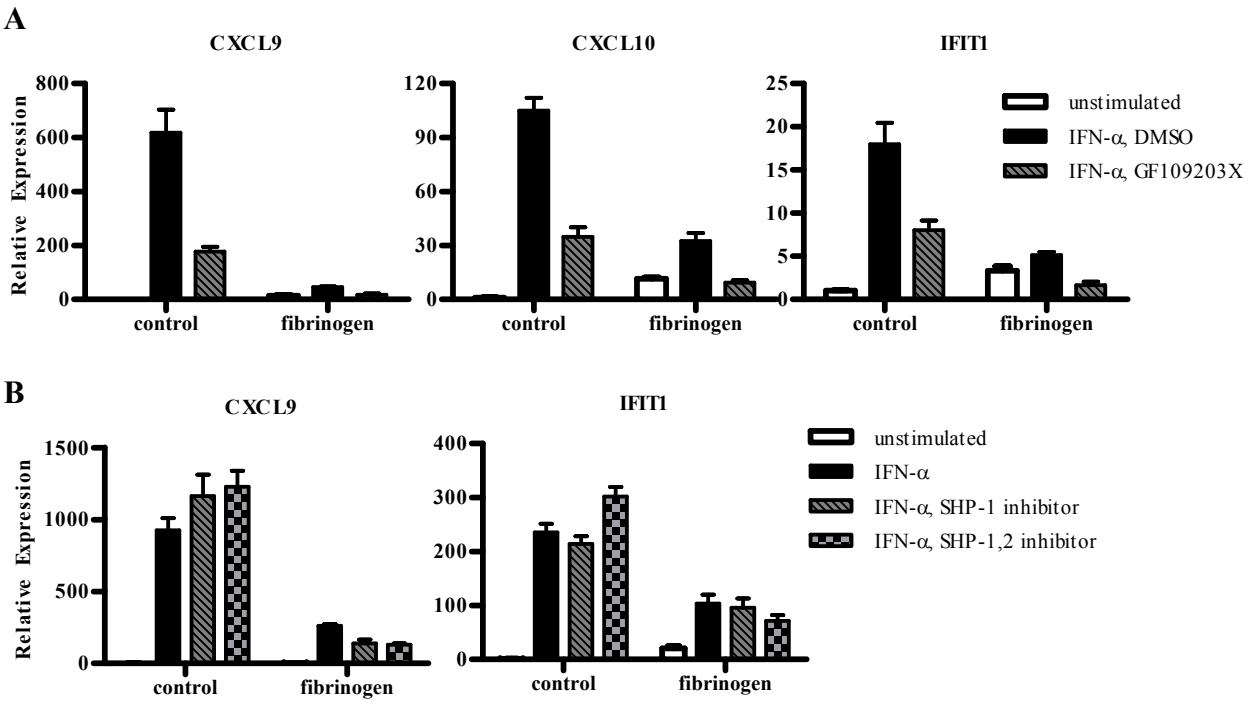

Figure S3. β2 integrin mediated inhibition of type I IFN signaling is independent of PKC and SHP-1 and SHP-2.

Primary human monocyte-derived macrophages were pretreated with (A) the vehicle control DMSO or PKC inhibitor GF109203X (10 μM) for 30 min or (B) SHP-1 inhibitor sodium stibogluconate (10 μg/mL) for 30 min. Sodium stibogluconate was also used at 100 μg/mL to inhibit both SHP-1 and SHP-2. Macrophages were then plated onto control wells or fibrinogen (Fb) coated wells, and IFN-α (1000 U/mL) was added for an additional 2 h to assess induction of IFN-α response genes. mRNA amounts were measured by qPCR, and results are presented as mean ± SD of triplicate wells normalized relative to GAPDH mRNA. Data are representative of 2 independent experiments.

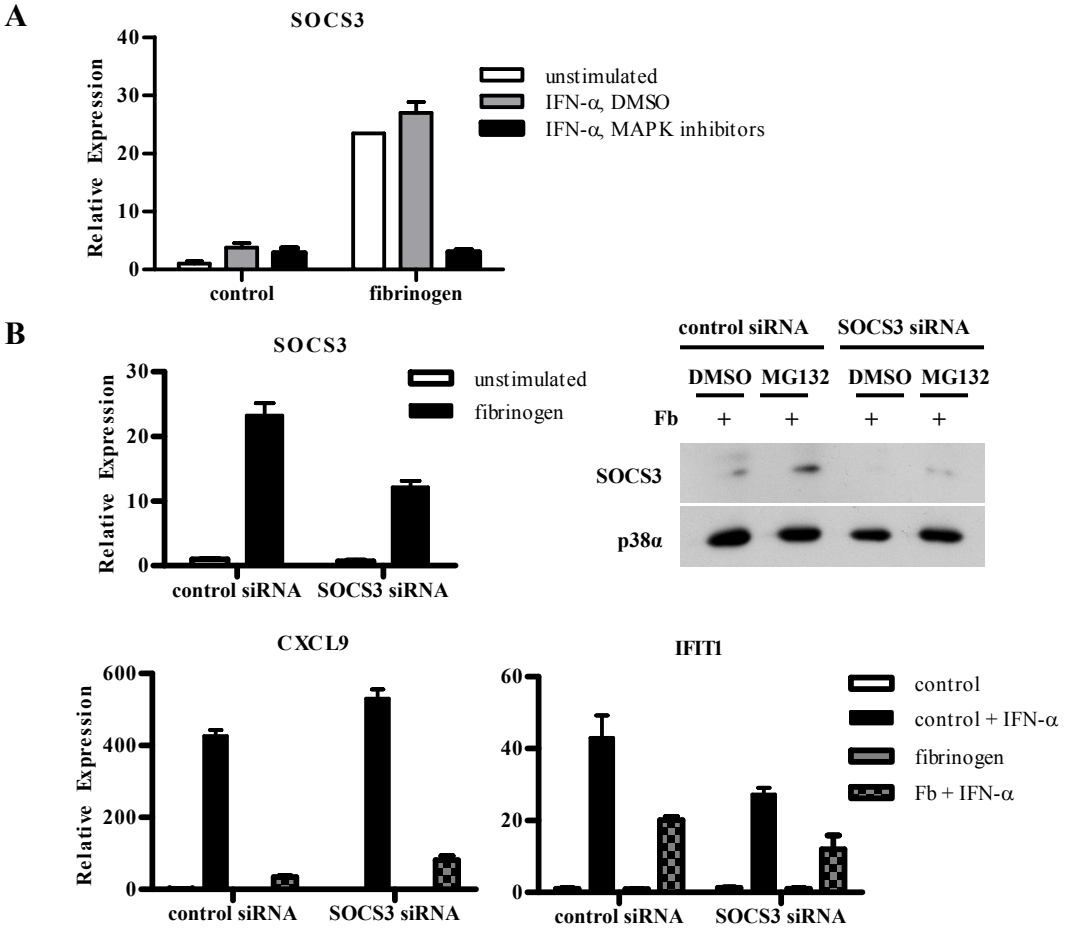

Figure S4. β2 integrin signaling induces SOCS3 expression in a MAPK-dependent manner.

A, Primary human macrophages were pretreated with either the vehicle control DMSO or two MAPK inhibitors: p38 inhibitor SB203580 (15 μM) and MEK inhibitor U0126 (15 μM) for 30 min. Treated macrophages were plated onto control wells or fibrinogen (Fb) coated wells for 2 h, and then IFN-α (1000 U/mL) was added for an additional 2 h. mRNA expression was measured by qPCR, and results are presented as mean ± SD of triplicate wells normalized relative to GAPDH mRNA. Data are representative of at least 3 independent experiments.

B, Macrophages were nucleofected with non-specific (control) or SOCS3-specific short interfering RNAs (siRNA) and were plated onto coated wells 3 days later. The top panels show knockdown efficiency. For SOCS3 mRNA detection, cells were plated onto coated wells for 4 h. For the immunoblot, the proteosome inhibitor MG-132 (20 μM) was used to stabilize and facilitate detection of SOCS3 protein. After 30 min incubation with MG-132, cells were plated onto Fb coated wells for 6 h, and whole cell lysates were immunoblotted with Abs against SOCS3 and p38α. The bottom panels show intact β2 integrin mediated inhibition of type I IFN signaling. Macrophages were plated onto wells for 4 h, and then IFN-α (1000 U/mL) was added for an additional 2 h to assess induction of IFN-α response genes. mRNA expression was measured by qPCR, and results are presented as mean ± SD of triplicate wells normalized relative to GAPDH mRNA. Data are representative of 2 independent experiments.