Supplementary Table 1

IRF-1	Hs00971965_m1
STAT1	Hs01014002_m1
FGF-1	Hs0026254_m1
FGF-2	Hs00266645_m1
MMP2	Hs00234422_m1
TIMP1	Hs99999139_m1
TIMP2	Hs00234278_m1
VEGF,	Hs00173626_m1
Epo	Hs01071097_m1
iNOS	Hs00167248_m1
HAS1	Hs 00155410_m1
HAS2	Hs00193435_m1
CD44	Hs00174139_m1
E-cadherin	Hs01023894_m1
collagen 1α	Hs00164004_m1
integrin β1	Hs00559595_m1
selectin E	Hs00174057_m1

Supplementary Figure 1 Huh7 and HepG2 cells have the same tendencies as the case with PLC/PRF/5 cells. Expression levels as detected by real-time RT-PCR

analysis. Huh7 and HepG2 cells were treated with 100 μM $CoCl_2$ and/or 1.0 x 10^3 IU/ml of IFNα for 24 hours. Asterisks indicate significant differences (*1, P<.001, *2, P<.01).

Supplementary Figure 2 The inhibitory effect of IFNα on VEGF expression was seen in low O_2 condition. Activation of STAT1 and expression of HIF-1α, STAT1, and VEGF as detected by western blot analysis. PLC/PRF/5 cells were treated with 1.0 x 10^4 IU/ml of IFNα and incubated in 1% O_2 concentration for 24 hours.

Supplementary Figure 3 IFNγ suppressed VEGF expression through inhibiting HRE-promoter activity. (*a*) (*b*) Expression levels as detected by real-time RT-PCR analysis. Cells were treated with 100 μM $CoCl_2$ and/or 100 ng/ml of IFNγ for 24 hours. (*c*) Activation of STAT1 and expression of VEGF as detected by western blot analysis. PLC/PRF/5 cells were transfected with control siRNA or siRNA for STAT1 and subjected to each stimulation for 24 hours. (*d*) Expression levels of HIF-1α and hydroxylated-HIF-1α as detected by western blot analysis. (*e*) Cells were cotransfected of the pGL2TkHRE plasmid or the pGL2Tk plasmid with the pRLtK plasmid. The cells were then stimulated with 100 mM of $CoCl_2$ units/ml and/or 100 ng/ml of IFNγ, or left unstimulated, and subjected to dual luciferase assay. The relative light unit of the unstimulated sample was considered as 1 and the data were expressed as mean ± S.D. Lower panel shows expression levels of STAT1 as detected by western blot analysis. Asterisks indicate significant differences (*1, P<.001, *2, P<.01, *3, P<.05).

Supplementary Figure 4 Cellular proliferation in IFNα-treated cells. PLC/PRF/5 cells were transfected with siRNA for STAT1 or control, and seeded on a

96-well culture plate, and then viable cells were assessed by water-soluble tetrazolium colorimetric assay. These cells were stimulated with 1.0 x 10^3 IU/ml of IFNα for 4 days (left panel) or for 2 days (right panel).

Supplementary Figure 5 Knockdown of STAT1 by siRNA method continued for more than 20 days. Three HCC cell lines were transfected with siRNA for STAT1 and extracted at the indicated times. These cellular lysates were subjected to western blot analysis.

Supplementary Figure 6 Human IFNα strongly activates STAT1 in human cell lines but less activates in mouse liver. Activation of STAT1 as detected by western blot analysis. 5×10^6 of PLC/PRF/5 cells were subcutaneously injected to Balb/c nude mice, and subcutaneous tumor and mouse liver were removed 10 days after transplantation. These mice were treated with 5×10^4 IU/mouse of human IFNα 1 hour before sacrifice.

Supplementary Figure 7 IFNα has almost no effects on E-cadherin, collagen 1α, integrin β1, and selectin E in the absence of STAT1. Expression levels of these molecules as detected by real-time RT-PCR analysis. PLC/PRF/5 cells were transfected with control siRNA or siRNA for STAT1 and stimulated on the next day with 5.0 x 10^3 IU/ml of IFNα for 3 hours or left untreated. The relative expression level of the untreated cells [IFNα (-), STAT1 (+)] was set as 1, and the fold expression level of each type of cell was calculated.

Supplementary Figure 8 Protein levels of VEGF were enhanced in suppressed pSTAT1 group, especially in tumor lesions. Tumor size-matched patients between

control and suppressed pSTAT1 group were selected and their NT and T samples were subjected to western blot analysis. Representative data are presented.