

SUPPLEMENTAL MATERIAL

SUPPLEMENTAL LEGENDS

SUPPLEMENTAL FIGURE S1. ATRA induced KLF4 and RAR α expressions in a concentration-dependent manner in VSMCs. **A**, VSMCs were treated with different concentrations of ATRA (10^{-8} ~ 10^{-4} M) for 24 h. Crude proteins were extracted from the treated cells and then subjected to Western blotting with antibodies against KLF4 or RAR α . β -Actin was used as a control for equal protein loading. **B**, Densitometry. Results were normalized to β -actin. Bars represent the mean \pm SEM from 3 independent experiments. * P < 0.05 vs. ATRA-free group.

SUPPLEMENTAL FIGURE S2. Partial peptide sequences of rat YB1. Numbering was from the initiation methionine of YB1. The peptide sequences *a* to *c* obtained by peptide mass fingerprinting were shown.

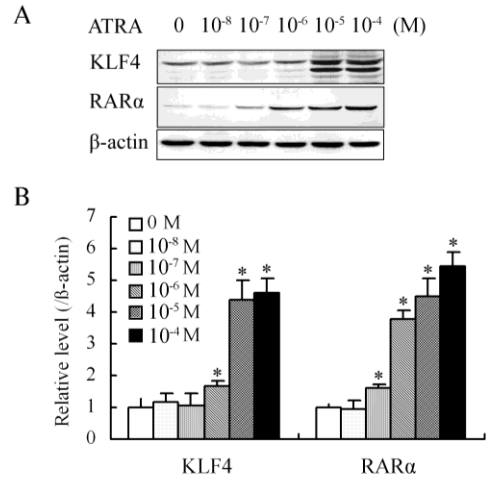
SUPPLEMENTAL FIGURE S3. The association of KLF4, Sp1 and YB1 with RAR β or RAR γ . GST, GST-KLF4, GST-Sp1, GST-YB1 and GST-MEF2C fusion proteins were purified from IPTG-induced BL21 *E. coli* using glutathione-Sepharose 4B beads. CHO-K1 cells were transfected for 24 h to express GFP-RAR β (**A**) or GFP-RAR γ (**B**) protein and then treated with 10 μ M of ATRA for one hour. The whole-cell lysates were used to perform GST pull-down assay. The recombinant GST, GST-KLF4, GST-Sp1, GST-YB1 and GST-MEF2C proteins on the glutathione beads were incubated with the cell lysates, respectively, overnight at 4 $^{\circ}$ C, followed by extensive washing. Proteins on the beads were eluted and detected by Western blot with anti-GFP and anti-GST antibodies.

SUPPLEMENTAL FIGURE S4. KLF4, Sp1 and YB1 cooperatively transactivated the *Klf4* promoter. **A**, CHO-K1 cells were co-transfected with a constant amount of pEGFP-KLF4 and increasing amounts of pEGFP-YB1, along with *Klf4* promoter-reporter construct pGL3-*Klf4*-luc. Cell lysates were subjected to luciferase activity assays using the dual-luciferase reporter assay system, and the luciferase activity was normalized to pRL-TK activity. The bars represent the mean \pm SEM from 3 independent experiments. * P < 0.05 vs. pEGFP-YB1-untransfected-group (*Bar 1*). Expression level of GFP-KLF4 and GFP-YB1 was assessed by Western blot analysis. β -Actin was used as a control for equal protein loading. **B-D**, CHO-K1 cells were co-transfected with the indicated constructs and then luciferase

activity of *Klf4* promoter-reporter construct were analyzed as described above. **P* < 0.05 vs. the control group (*Bar 1*). Expression level of GFP-YB1, GFP-KLF4 and Sp1 was assessed by Western blot analysis. β -Actin was used as a control for equal protein loading.

SUPPLEMENTAL FIGURE S5. ATRA induced SM22 α , p21 and p53 expression in VSMCs. **A**, VSMCs were treated with 10 μ M of ATRA for 0, 6, 12, or 24 h. Crude proteins were extracted from the treated cells and then subjected to Western blot with antibodies against SM22 α , p21 or p53. β -Actin was used as a control for equal protein loading. **B**, Densitometry; results were normalized to β -actin. Bars represent the mean \pm SEM from 3 independent experiments. **P* < 0.05 vs. ATRA-free group.

Supplemental Figure S1

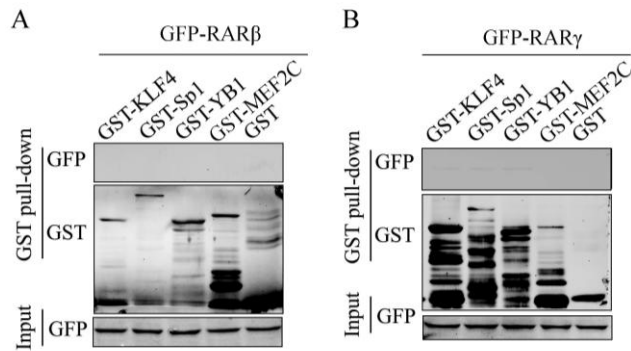


Supplemental Figure S2

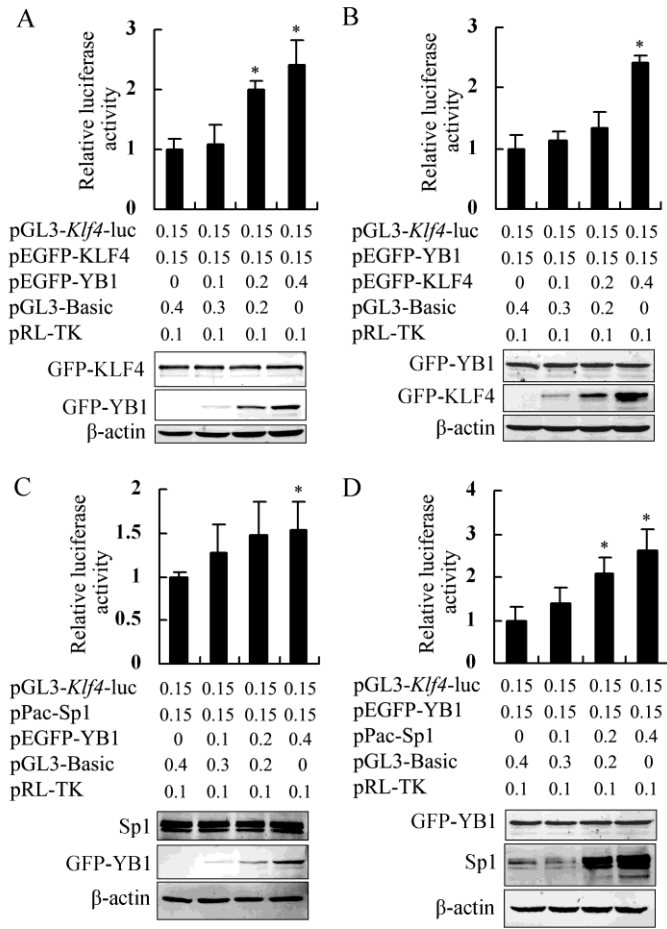
YB1 91 KNNPRKYLRSVGDGETVEFDVVEGEKGAEEANVTGPGGVPVQGSKYAADR 140
 (a) RSVGDGETVEFDVVEGEK
 (b) KGAEANVTGPGGVPVQGSKY

YB1 221 NQGAGEQGRPVQRQNMRYRPRFRGPPRQRQPREDGNEEDKENQGDDET 269
 (c) RQNMRYRPRF

Supplemental Figure S3



Supplemental Figure S4



Supplemental Figure S5

