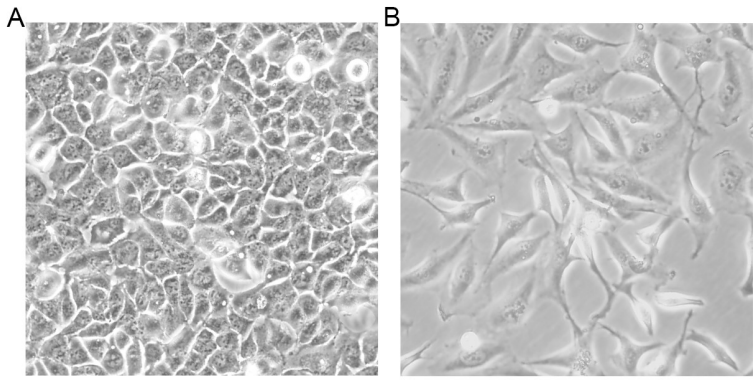
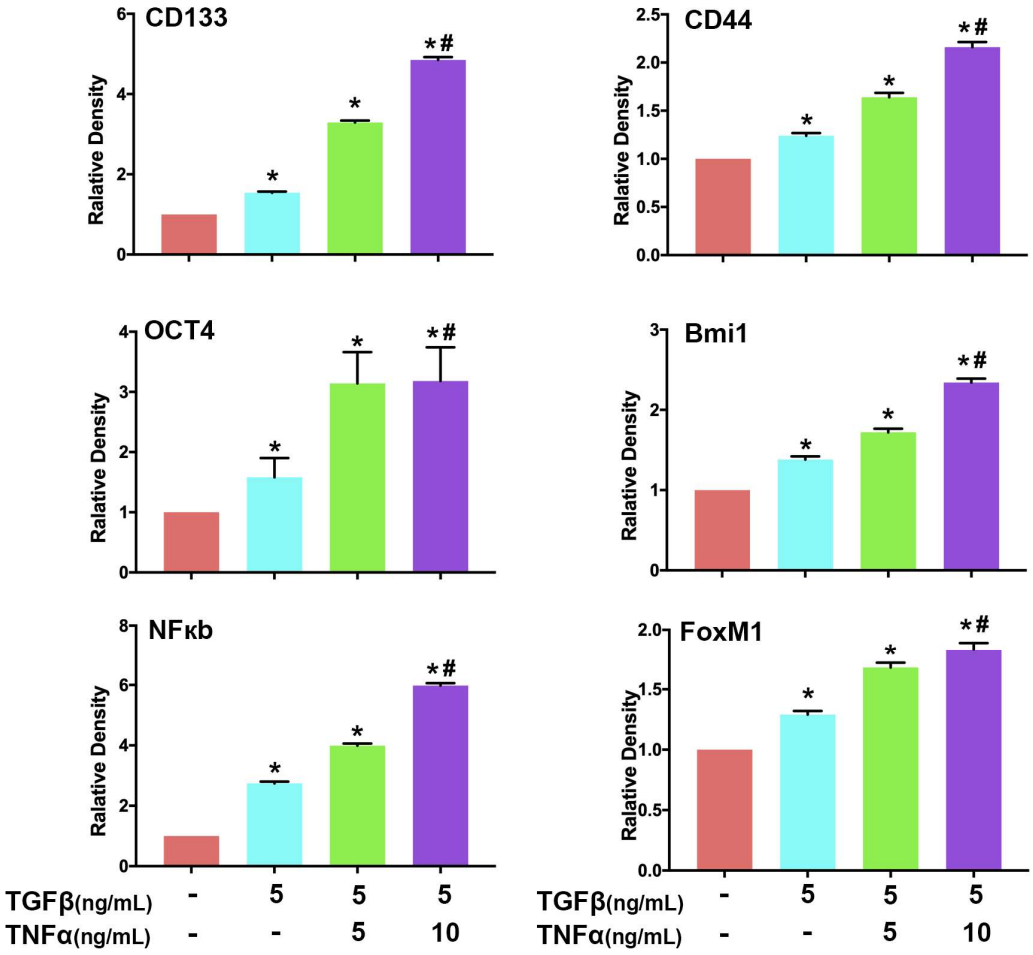
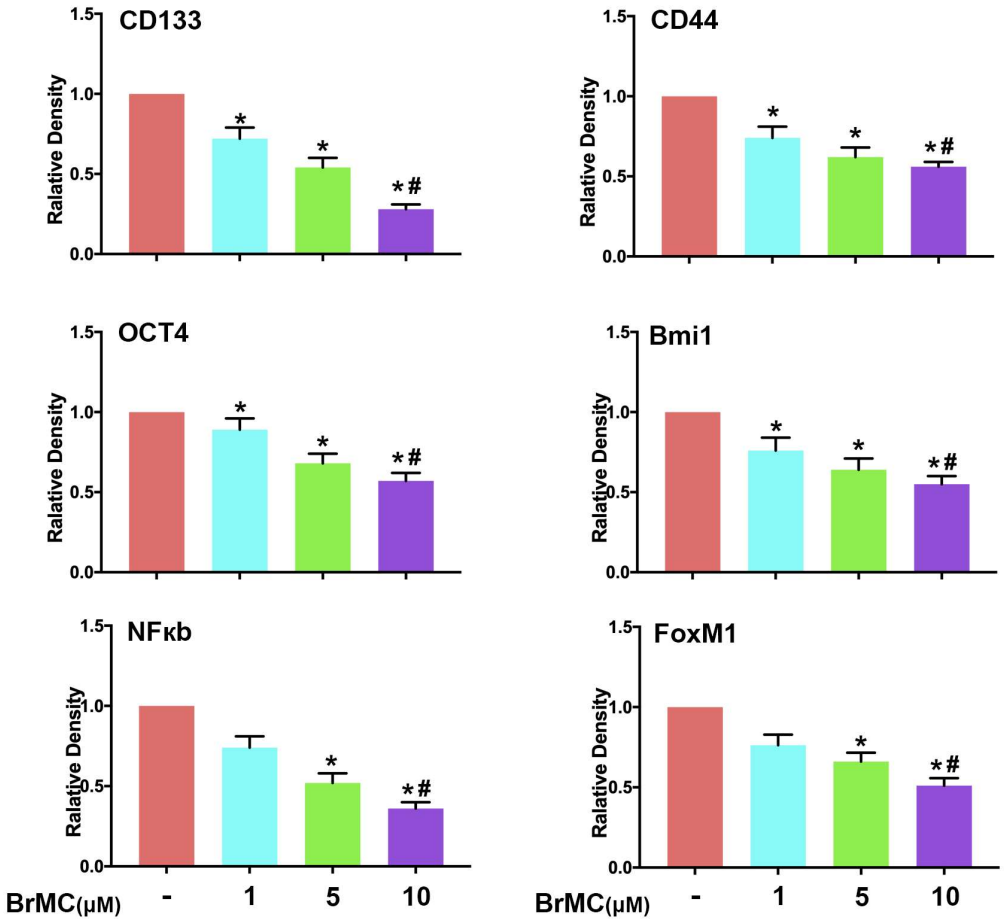
Supplementary figures



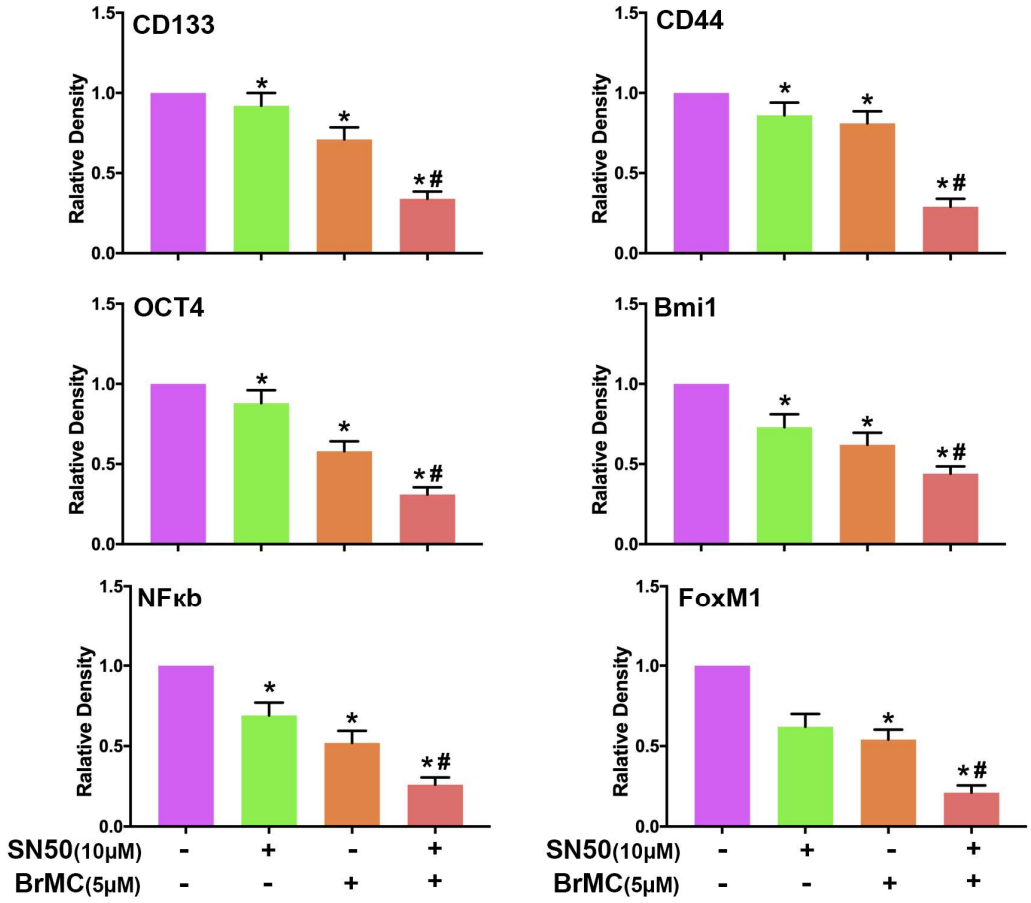
sFig.1 The morphological changes of NSCLC H460 cells induced by TNF-α and TGF-β before (A) and after (B) incubation The epithelial cells got transition to mesenchymal cell shape in the presence of TNF-α and TGF-β. (Magnification: 20x).



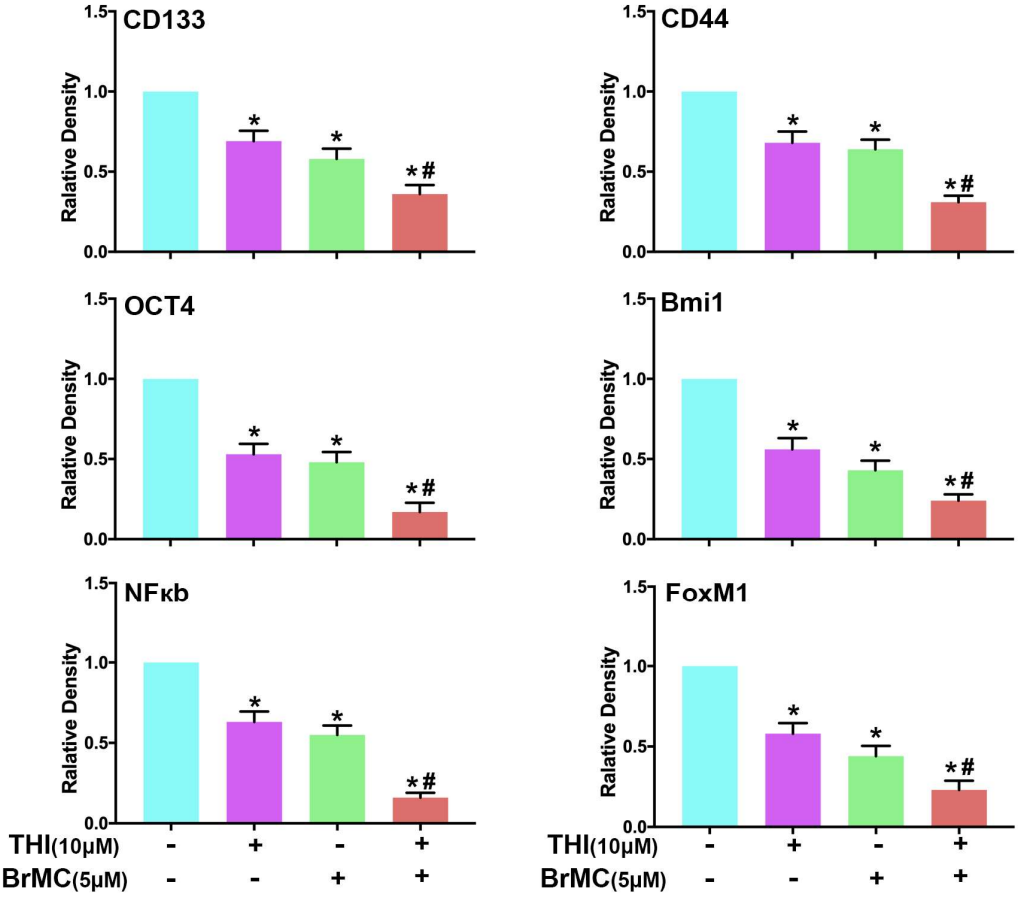
sFig.2 incubation of TNF-α and TGF-β promote expression of stem cell marker (CD133, CD44, OCT4, Bmi1), NFκB and FoxM1 in non-small cell lung cancer cells H460. The protein expression level was normalized by β-actin. * P<0.05, compared to untreated cells. # P<0.05, compared to TGFβ alone.



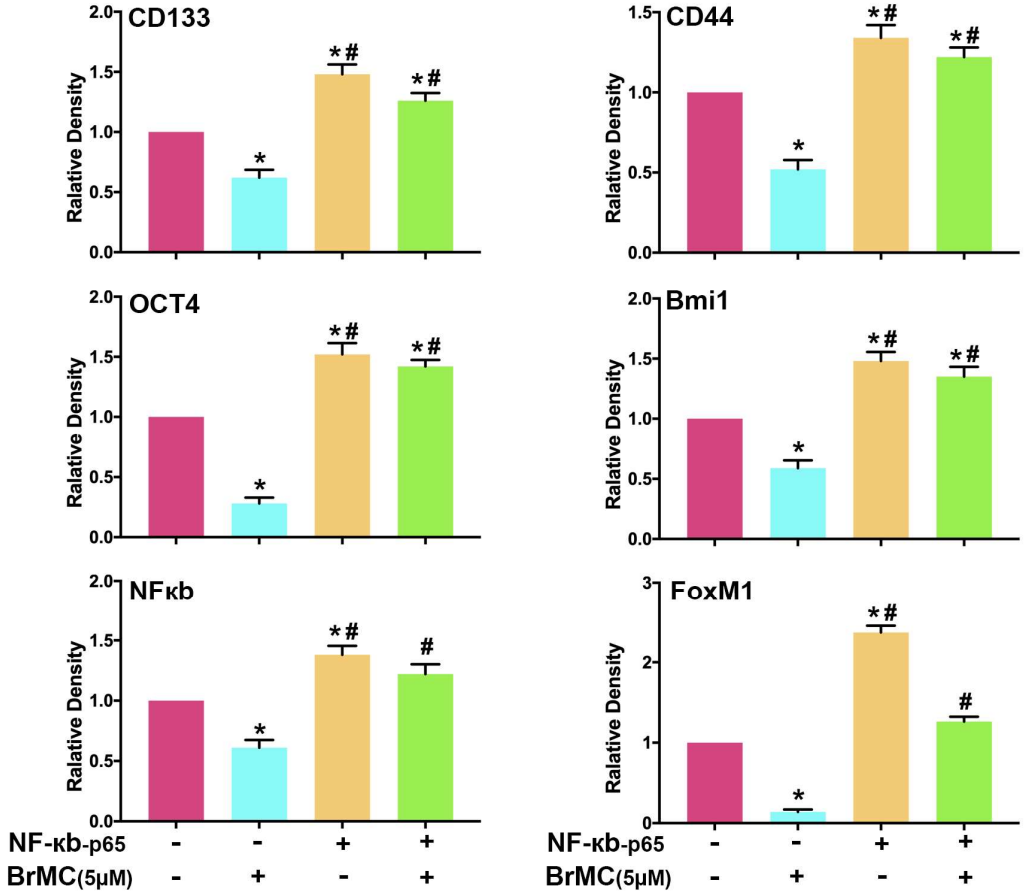
sFig.3 BrMC inhibit stem cell marker (CD133, CD44, OCT4, Bmi1), NFκB and FoxM1 in H460 stem cells induced by TNF-α and TGF-β. The protein expression level was normalized by β-actin. * P<0.05, compared to untreated cells. # P<0.05, compared to BrMC (1.0 μmol/L) treatment.



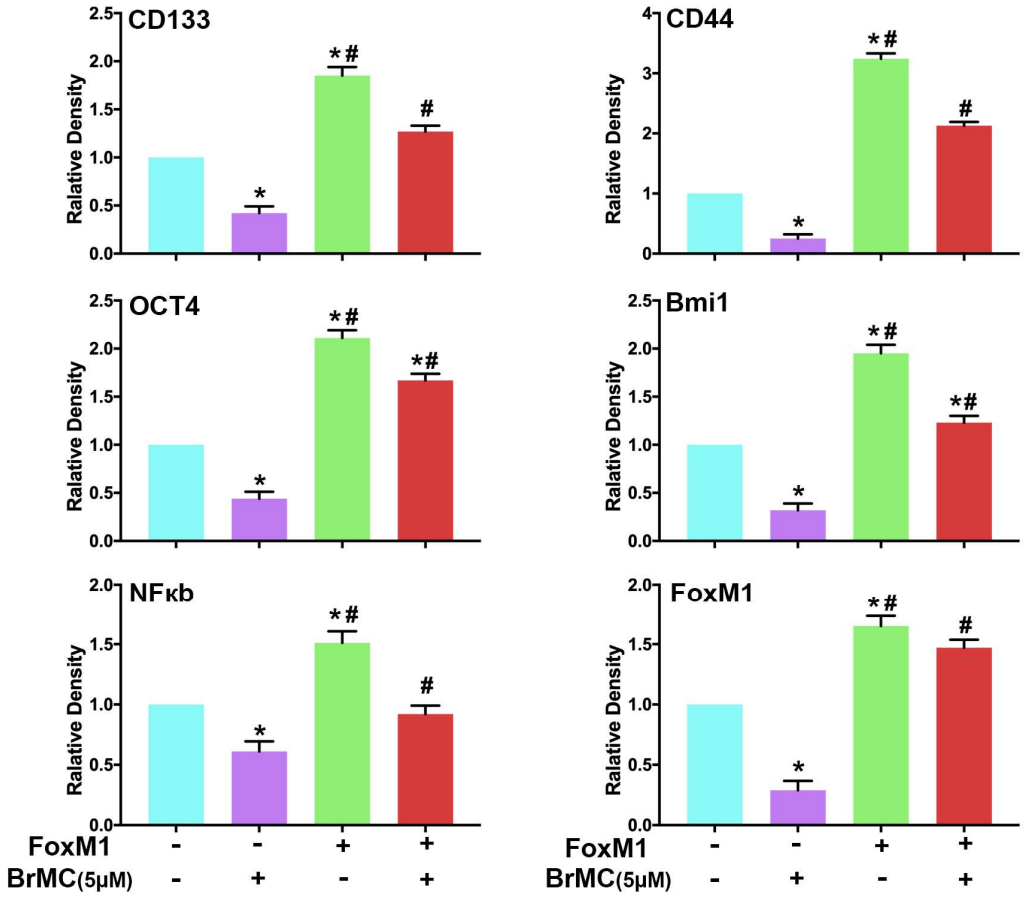
sFig.4 Suppression of NFκB by SN50 can enhance the inhibition of BrMC on non-small lung cancer stem cells. Expression of cancer stem cell marker (CD133, CD44, OCT4, Bmi1), NFκB and FoxM1 were analyzed by western blot, and the protein expression level was normalized by β-actin. * P<0.05, compared to untreated cells. # P<0.05, compared to SN50 (10.0 μmol/L) treatment only.



sFig.5 Suppression of FoxM1 by thiostrepton can enhance the inhibition of BrMC on non-small lung cancer stem cells. Expression of cancer stem cell marker (CD133, CD44, OCT4, Bmi1), NFκB and FoxM1 were analyzed by western blot, and the protein expression level was normalized by β-actin. * $P<0.05$, compared to untreated cells. # $P<0.05$, compared to THI (10.0 μmol/L) treatment only.



sFig.6 Overexpression of NFκB-p65 compromised the inhibition of BrMC on non-small lung cancer stem cells. Expression of cancer stem cell marker (CD133, CD44, OCT4, Bmi1), NFκB and FoxM1 were analyzed by western blot, and the protein expression level was normalized by β-actin. * $P<0.05$, compared to cells with vectors. # $P<0.05$, compared to cells with vectors and treated by BrMC (5.0 μmol/L).



sFig.7 Overexpression of FoxM1 compromised the inhibition of BrMC on non-small lung cancer stem cells. Expression of cancer stem cell marker (CD133, CD44, OCT4, Bmi1), NFκB and FoxM1 were analyzed by western blot, and the protein expression level was normalized by β-actin. * P<0.05, compared to cells with vectors. # P<0.05, compared to cells with vectors and treated by BrMC (5.0 μmol/L).