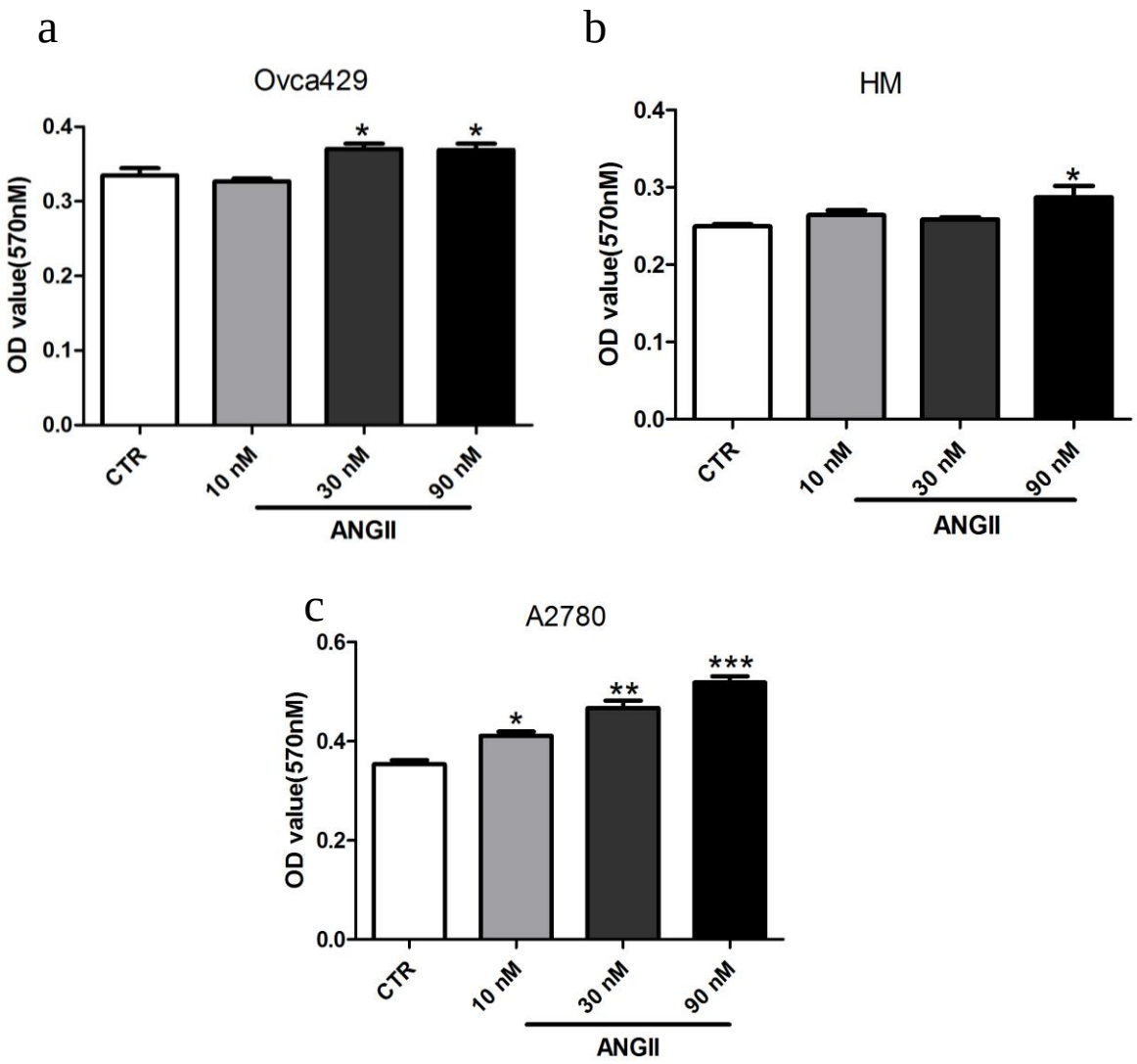
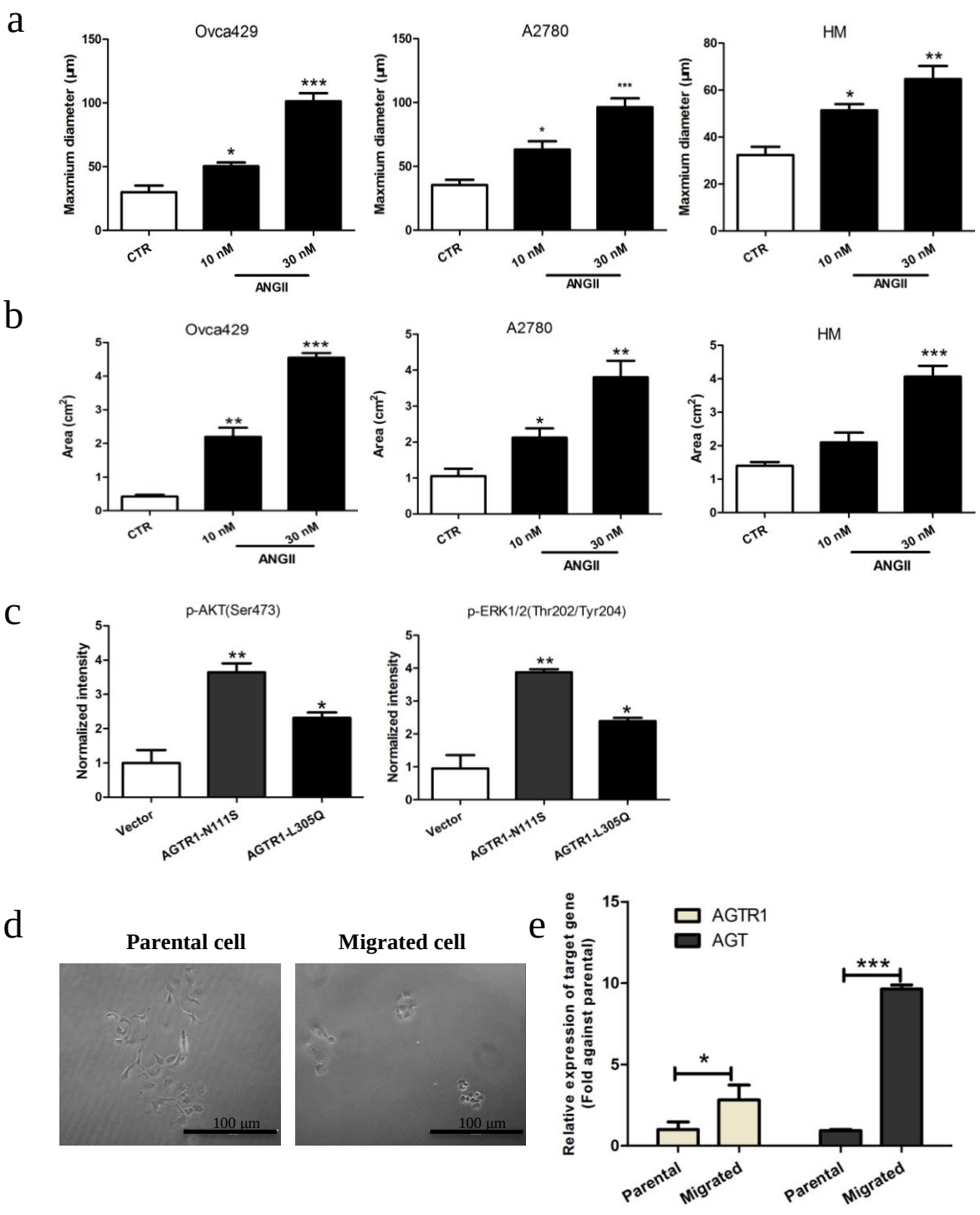
Supplementary Figure 1



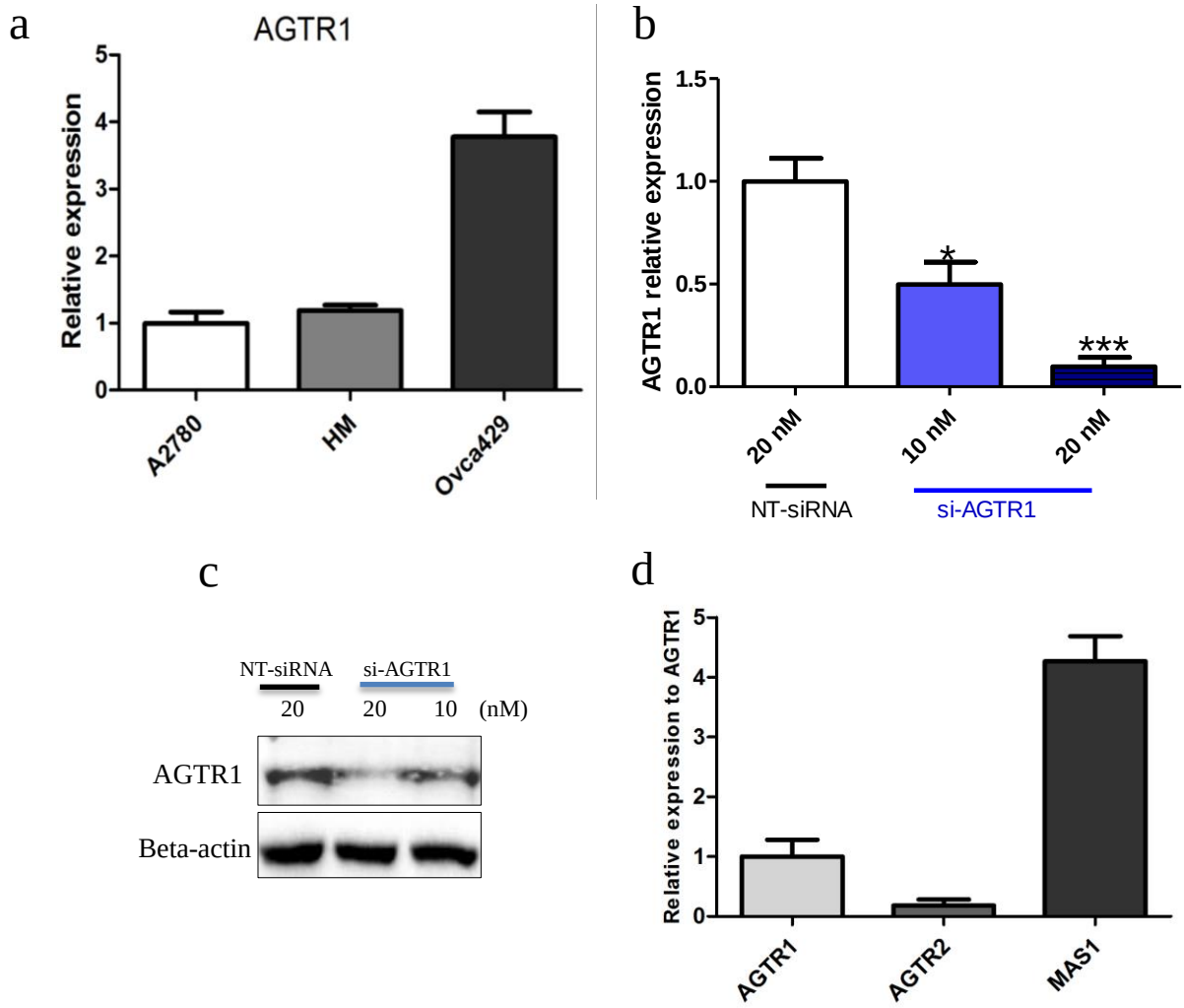
Supplementary Figure 1| ANGII effect on ovarian cancer cell viability. (**a**) The ANGII effect on proliferation of Ovca429 cell were measured by MTT assay. The data is presented as means ± SEM and the significant difference were indicated (*p<0.05 against control). (**b**) The ANGII effect on proliferation of HM cell were measured by MTT assay. The data is presented as means ± SEM and the significant difference were indicated (*p<0.05 against control). (**c**) The ANGII effect on proliferation of A2780 cell were measured by MTT assay. The data is presented as means ± SEM and the significant difference were indicated (*p<0.05,**p<0.01,***p<0.001 against control).

Supplementary Figure 2



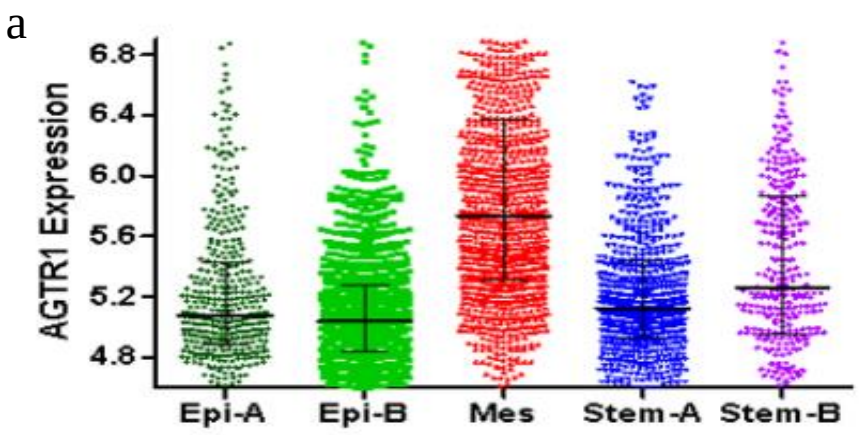
Supplementary Figure 2| ANGII promotes ovarian cancer MCS formation and migration. (**a**) ANGII significantly increased the maximum diameter of the MCS. The diameters of the spheroids (at least 10 spheroids counted) in the Matrigel were measured by ImageJ software. (**b**) The cell growth of the ovarian cancer spheroids was measured by crystal violet staining. The growth areas were quantified by ImageJ software. (**c**) The western blot band intensity was determined by the gel imaging system (ChemiDoc™ XRS+ Imaging Systems, Bio-Rad) and data are shown as means ± SEM; * $p < 0.05$, ** $p < 0.01$. (**d**) Bright field images of the cell morphology of the parental cells and migrated cells after the Transwell assay. Scale bar, 100 μm. (**e**) Total RNA were extracted from the parental cells and the migrated cells. The expression of AGTR1 and AGT were determined by RT-qPCR. The relative expression levels of AGTR1 and AGT were calculated by the -2ddCt method. The data are presented as means ± SEM. Significant differences between parental and migrated cells are indicated (* $p < 0.05$, *** $p < 0.001$).

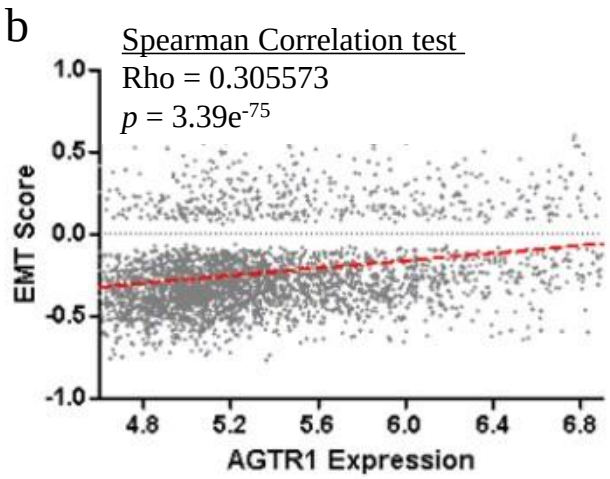
Supplementary Figure 3

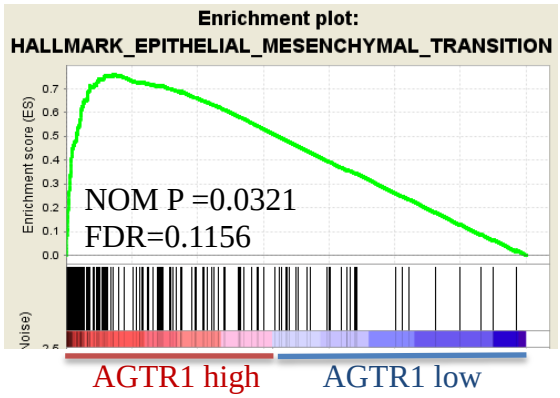


Supplementary Figure 3| AGTR1 gene expression in ovarian cancer cell line. (**a**) AGTR1 gene relative expression level in A2780, HM and Ovca429 cell were quantified by RT-qPCR. The result is presented as means ± SEM. (**b**) The silencing efficiency of siRNA-AGTR1 on suppressing of AGTR1 mRNA expression level. The result is presented as means ± SEM and the significant difference were indicated (*p<0.05,***p<0.001 against NT-siRNA). (**c**) The silencing efficiency of siRNA-AGTR1 was confirmed by Western blotting. (**d**) Three receptor AGTR1, AGTR2 and MAS1 expression level in Ovca429 cell were quantified by RT-qPCR. The result is presented as means ± SEM.

Supplementary Figure 4

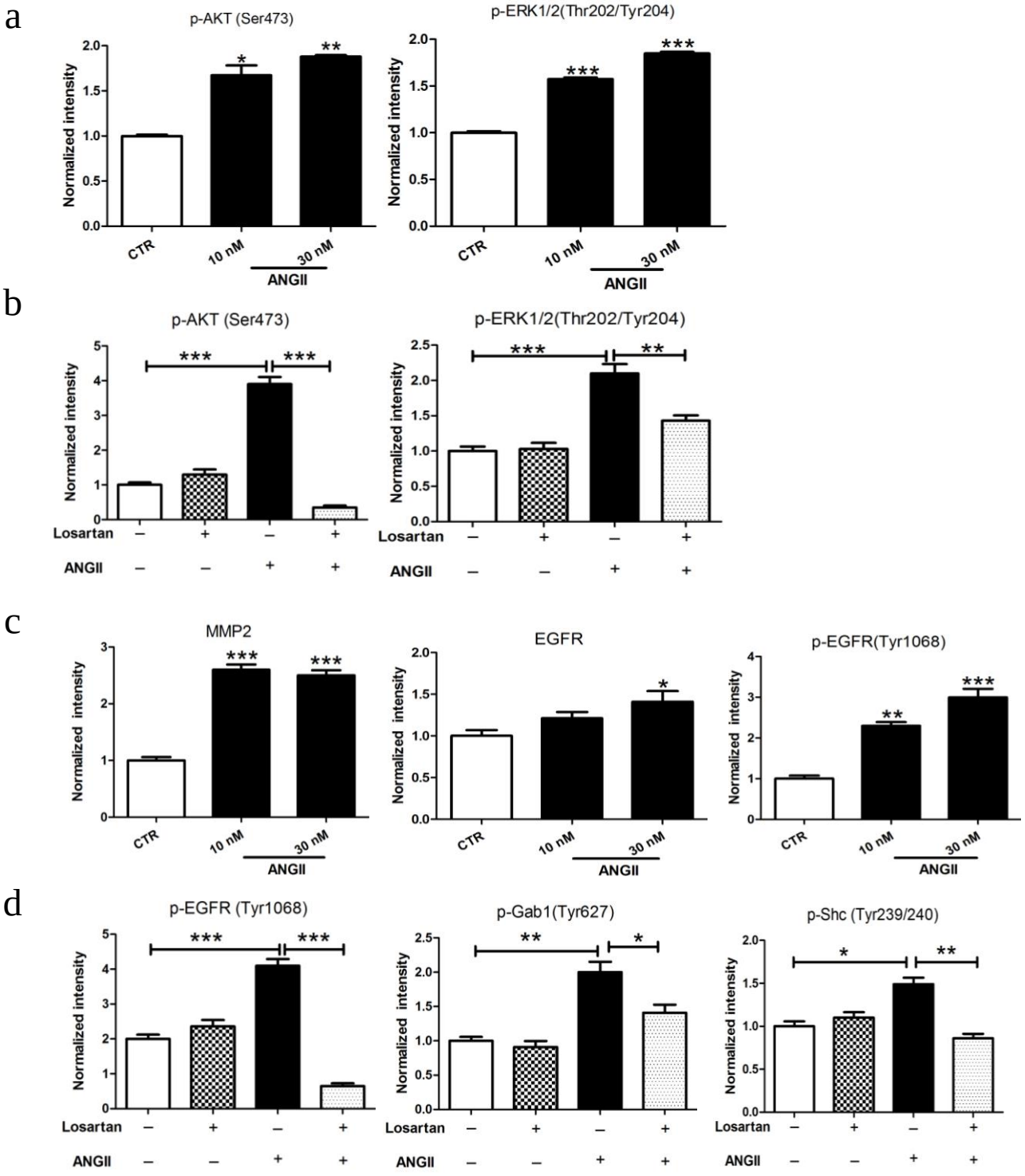






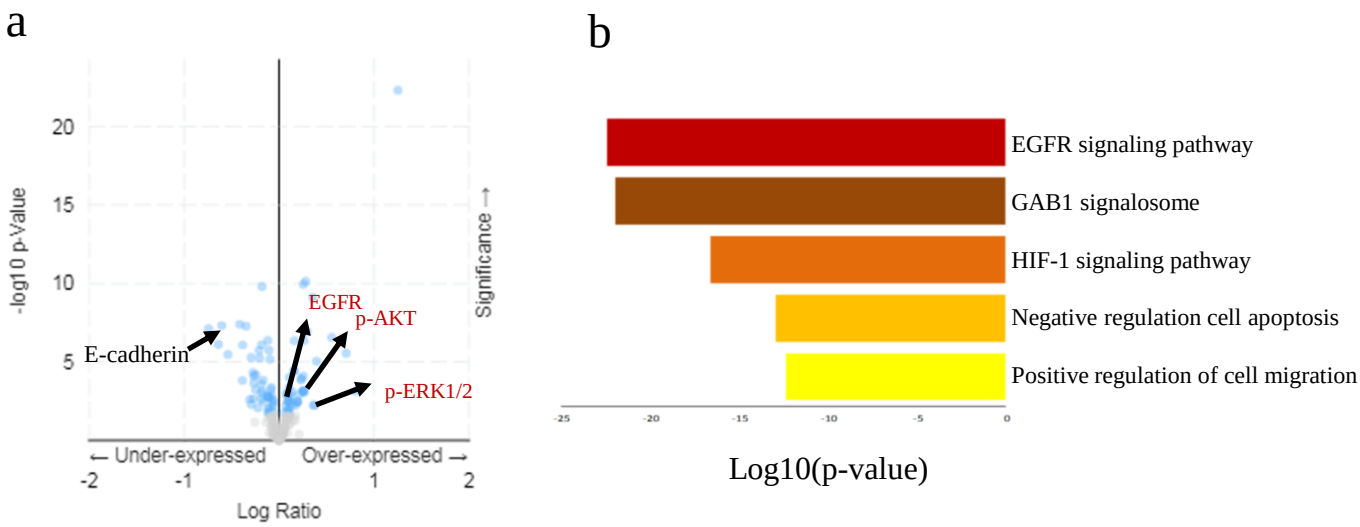
Supplementary Figure 4| AGTR1 gene expression predicates high metastasis of ovarian cancer cell. (**a**) AGTR1 upregulated in metastatic subtype of ovarian cancer patients. (**b**) The AGTR1 gene expression is significantly positively correlated with EMT markers gene expression (spearman correlation test, p-value =3.39e^{-75}). (**c**) GSEA enrichment analysis show the EMT gene set were activated in AGTR1 high expression patients (NES=1.77, NOM p=0.032, FDR=0.115). Abbreviation: Epi-A, epithelial-A; Epi-B, epithelial-B; Mes, mesenchymal; Stem-A, stem-like-A; Stem-B, stem-like-B.

Supplementary Figure 5



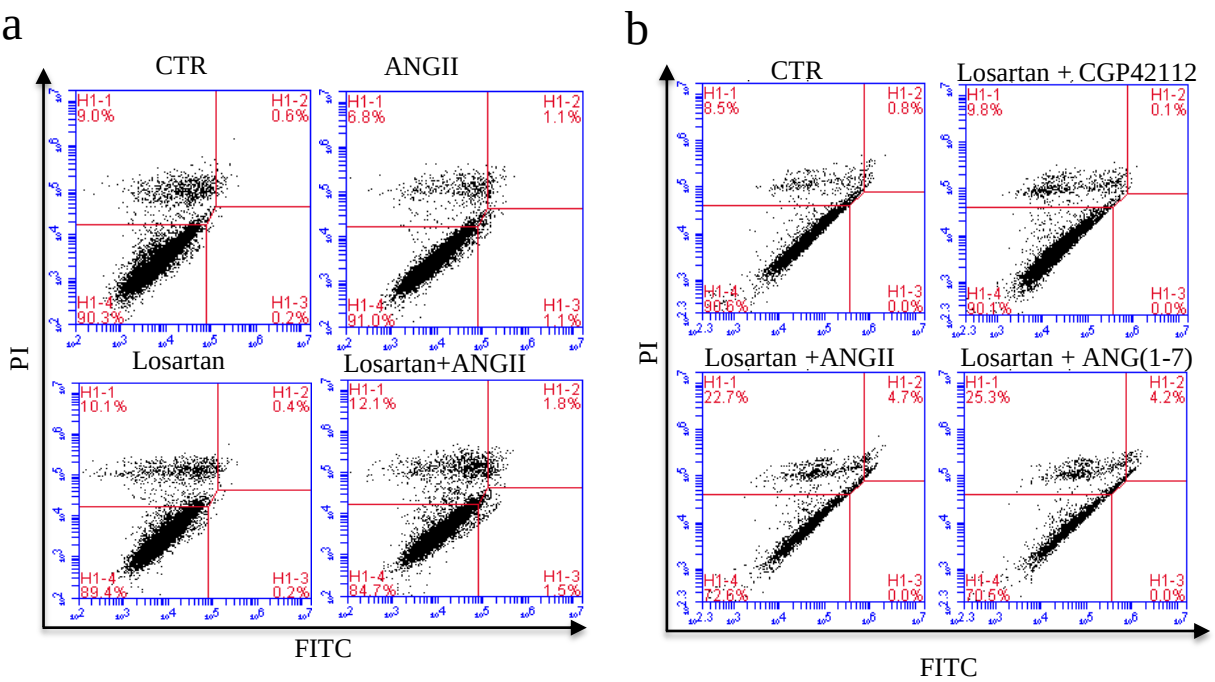
Supplementary Figure 5| ANGII triggered classical AGTR1 signaling and the transactivation of EGFR in ovarian cancer cells. (a) p-AKT and p-ERK protein level in ovarian cancer cell after ANGII treatment were measured by Western blot and normalized using GAPDH as a loading control. (**b**) p-AKT and p-ERK protein level in ovarian cancer cell under ANGII with/without losartan treatment were measured by Western blot and normalized using GAPDH as a control.(**c**) MMP2, EGFR, p-EGFR protein level in ovarian cancer cell under ANGII treatment were measured by Western blot and normalized using GAPDH as a loading control. (**d**) p-EGFR, p-Gab1 and p-Shc protein level in ovarian cancer under ANGII with/without losartan treatment were measured by Western blot and normalized using GAPDH as a loading control All data are presented as means ± SEM from at least three experiments; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ against the no treatment control or the samples with ANGII treatment.

Supplementary Figure 6



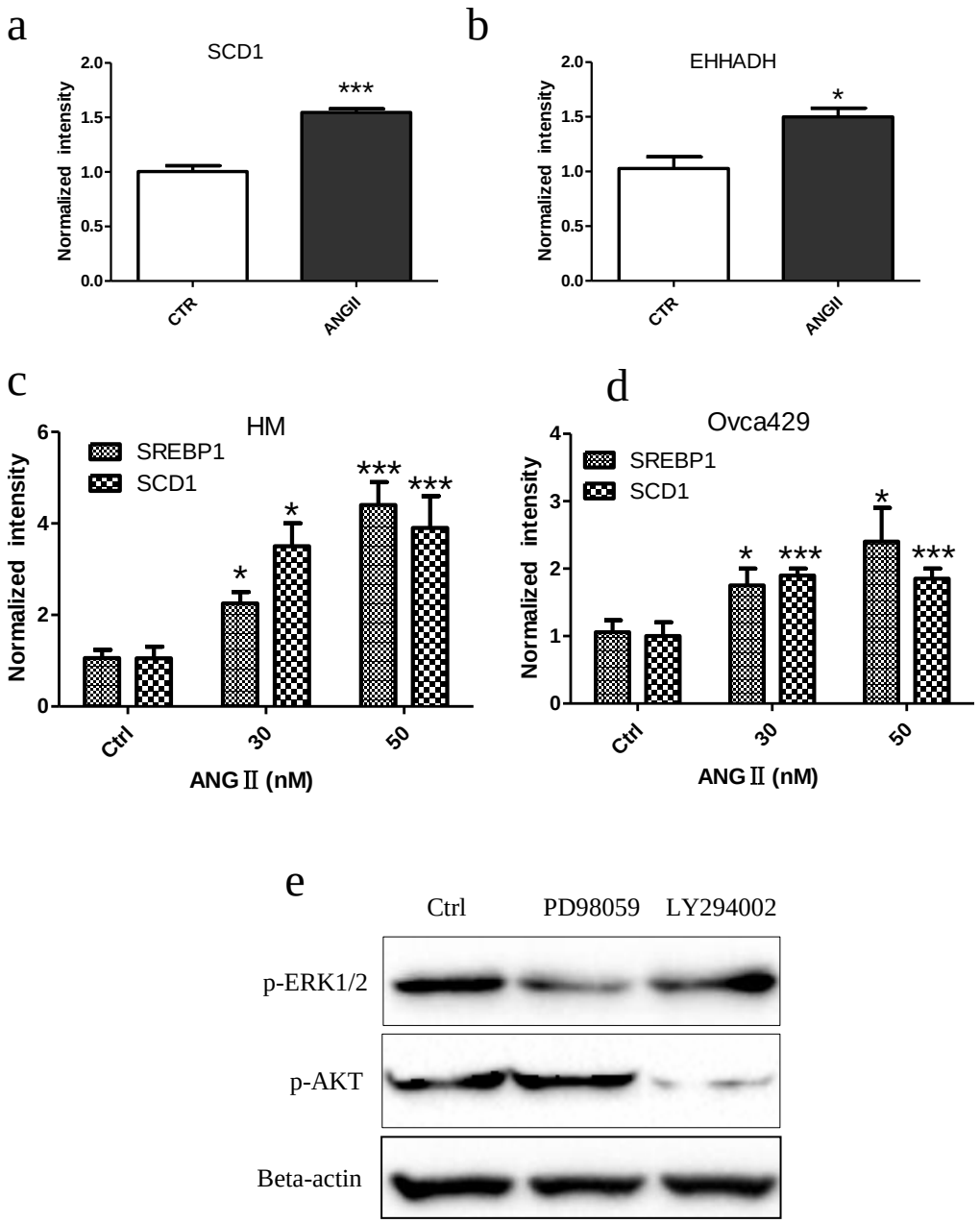
Supplementary Figure 6| AGTR1 high expression predicates transactivation of EGFR signaling pathway. (**a**) Volcano plot show the proteins upregulated/ downregulated in AGTR1 high expression patients tumor tissues compared with AGTR1 low expression patients tumor tissues. (**b**) The proteins upregulated were analyzed using GO enrichment analysis.

Supplementary Figure 7



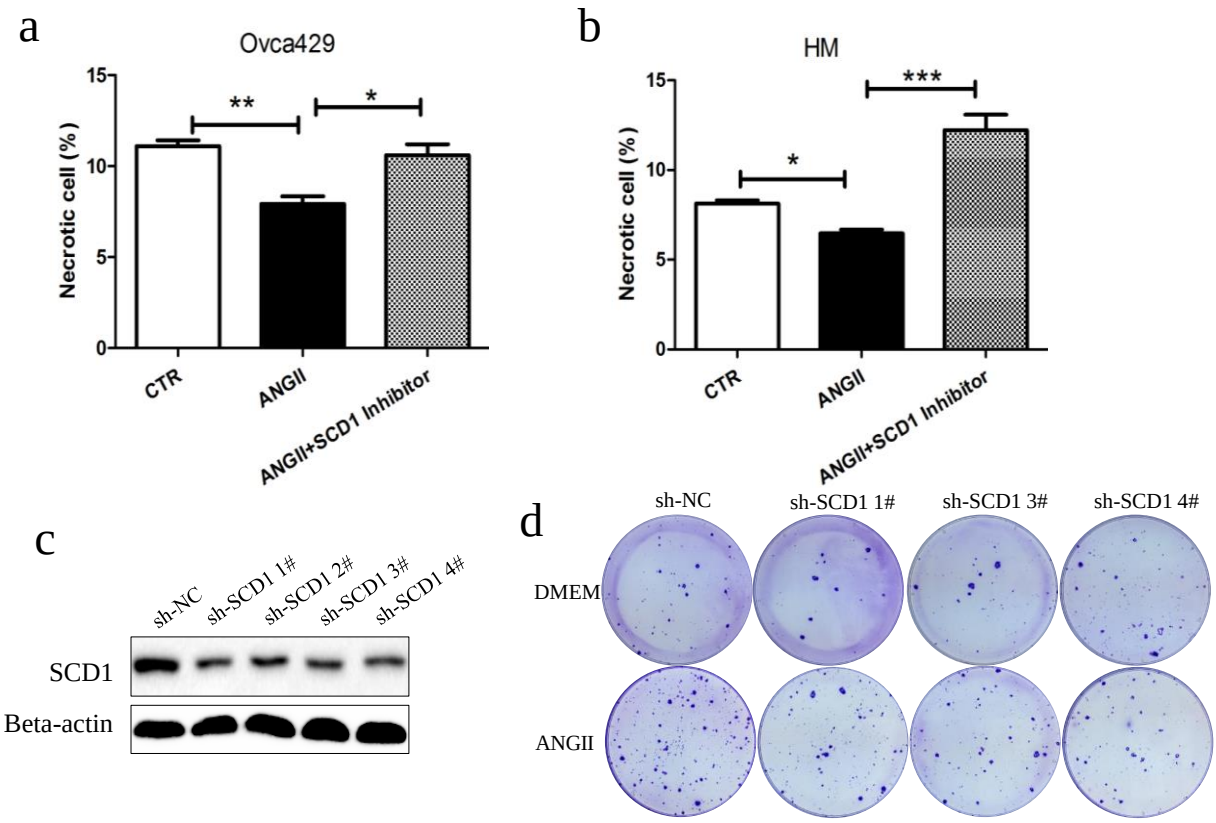
Supplementary Figure 7| ANGII enhances the MCS formation by reducing the cell necrosis (a) Cell death of MCS was assessed by Annexin V-FITC and PI assay by flow cytometry after treatment with ANGII (100 nM) and/or losartan (10 μM). Necrotic cells in each group were quantified. The data are presented as means ± SEM from at least three experiments; * $p < 0.05$, *** $p < 0.001$ against the control group. **(b)** Cell death inside MCS were detected by flow cytometry with different combinations of treatment: ANGII (100 nM), losartan (10 μM), CGP42112 (50 nM) and/or ANG(1-7) (100 nM). Necrotic cells in each group were quantified accordingly. The data are presented as means ± SEM from at least three experiments; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ against control.

Supplementary Figure 8



Supplementary Figure 8| ANGII induced SCD1 expression by upregulation of transcriptional factor SREBP1. (a & b) SCD1 and EHHADH protein level in ovarian cancer cell after ANGII treatment were measured by Western blot and normalized using beta-actin as a loading control. (**c** & **d**) SREBP1 and SCD1 protein level in HM cell and Ovca429 cell under ANGII were measured by Western blot and normalized using beta-actin as a control. (**e**) p-ERK1/2, p-AKT protein level in ovarian cancer cell under ERK1/2 inhibitor(PD98059,50μM) and PI3K/AKT inhibitor(LY294002, 100μM) treatment were measured by Western blot and normalized using beta-actin as a loading control. All data are presented as means ± SEM from at least three experiments; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$ against the no treatment control or the samples with ANGII treatment.

Supplementary Figure 9



Supplementary Figure 9| Blocking of SCD1 reverse ANGII effect on relieve cancer cell death during spheroid formation. (a & b) SCD1 inhibitor (cat: ab142089, 10 nM) reversed the effect of ANGII on necrosis in spheroids of two ovarian cancer cell lines (a: Ovca429 cells, b: HM cells). The necrotic cells were measured by Annexin V-FITC/PI flow cytometry. The data are presented as means ± SEM from at least three experiments; * $p < 0.05$, *** $p < 0.001$ against the control group. (**c**) The knock-down efficiency of shRNA on SCD1 expression was confirmed by Western blotting. (**d**) Knock-down of SCD1 significantly reduced the ANGII mediated spheroid formation of ovarian cancer cell.