## **SUPPLEMENTARY FIGURES**



Supplementary Figure S1: Autophagy defect induces mesenchymal-like morphologies in gastric cancer cells. SGC-shBECN1 and MGC-shBECN1 cells were more spindle-like and scattered than control cells. Scale bar, 100 µm.

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Supplementary Figure S2: Activation of HIF-1 $\alpha$  is necessary for autophagy defect induced EMT. A. HIF-2 $\alpha$  expression in autophagy-deficient cells and control cells were determined by Western blot. B & C. After transfection with HIF-1 $\alpha$  siRNA, the expression of EMT markers in SGC-shBECN1 cells were analyzed by Western blot.



Supplementary Figure S3: Autophagy defect increases HIF-1a expression via ROS-NF- $\kappa$ B-HIF-1a pathway. A. Intracellular and mitochondrial ROS levels were determined by FCM. B. After treating MGC-shBECN1 cells with 3 or 10 mM NAC for 48 h, the expression of HIF-1a and NF- $\kappa$ B p65 in nuclei, and the expression of Phospho-I $\kappa$ Ba, HIF-1a, E-cadherin, Twist-1 in whole cells were determined by Western blot. C & D. After transfection with NF- $\kappa$ B p65 siRNA, the HIF-1a expression in SGC-shBECN1 cells were analyzed by Western blot. E. After incubation with or without 10 mM NAC for 48 h, the EMSA was performed to observe the binding activity between nuclear extracts and specific probe (containing NF- $\kappa$ B binding site in HIF-1a promoter).



**Supplementary Figure S4: Autophagy inhibition induced glycolysis has no effect on EMT.** After incubation with 5 mM 2-DG for 12 h, the EMT markers in SGC-shBECN1 and MGC-shBECN1 cells were determined by Western blot.



Supplementary Figure S5: Immunohistochemical analysis of consecutive sections from human gastric cancer tissues. The expression of HIF-1 $\alpha$ , E-cadherin and Glut1 were determined in positive BECN1 expression case (case 77) and negative case (case 28) respectively. HIF-1 $\alpha$  was mainly localized in nucleus, while Glut1 was largely distributed in cytoplasm and cytomembrane. Scale bar, 100 µm.