Inactivation of HMGCL promotes proliferation and metastasis of nasopharyngeal carcinoma by suppressing oxidative stress

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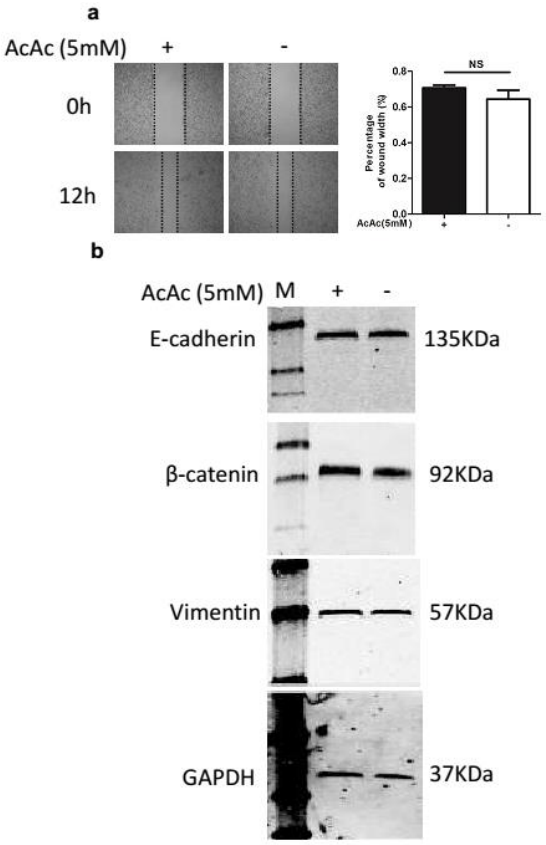
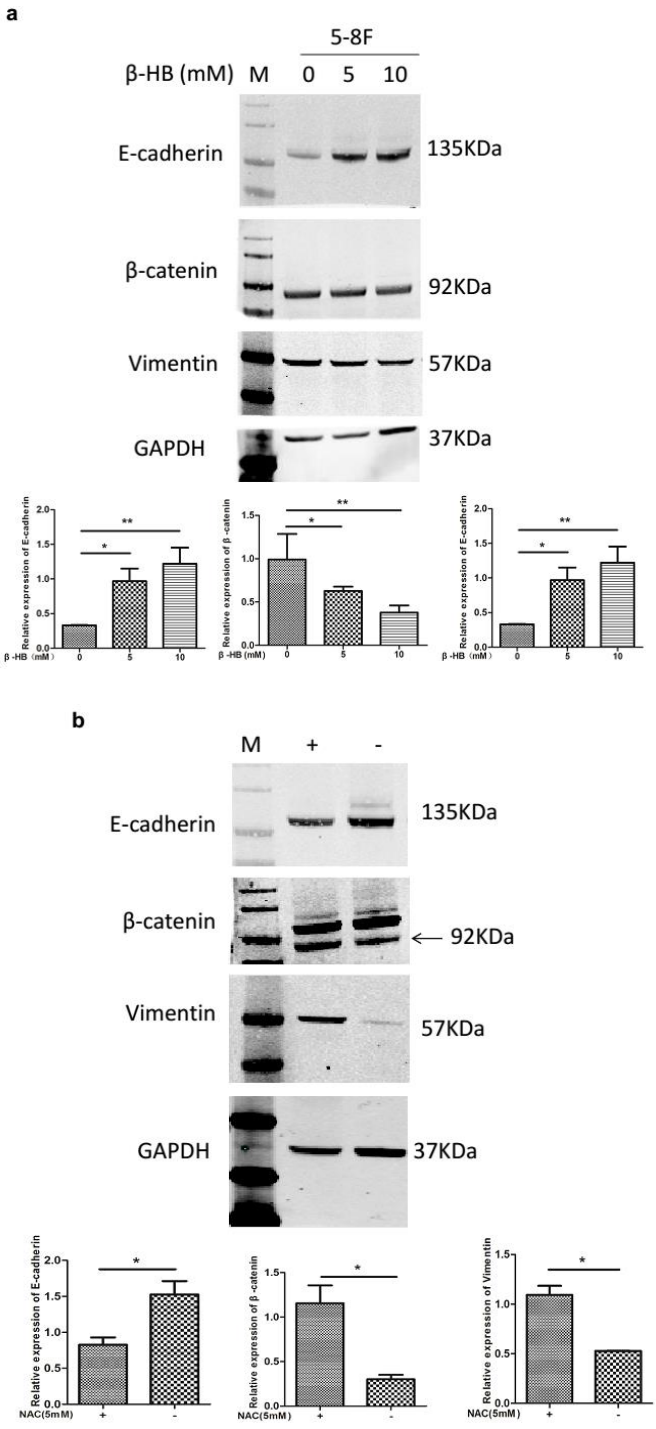
Supplementary Figure 1



Supplementary Figure Legend 1:

The motility of 5-8F cells does not affected by acetoacetate (AcAc). (a) The migration of 5-8F cells upon the treatment of 5mM lithium acetoacetate (Li-AcAc, Sigma Aldrich, # A8509) was analyzed by wound healing assay. (b) Western blot analysis of the expression of three key molecules involved in EMT, E-cadherin, β-catenin and Vimentin. GAPDH was an internal control. Data are mean ±SD (n=3).

Supplementary Figure 2



Supplementary Figure Legend 2:

Exogenous β-HB treatment promotes the mesenchymal-epithelial transition (MET) in 5-8F cells. NAC reversed the MET functioned by HMGCL in HMGCL-5-8F cells. (a)Western blot analysis of the expression of three key molecules involved in E-cadherin, β-catenin and Vimentin in 5-8F treated by β-HB (0mM, 5mM and 10mM) for

24 h. (b) EMT markers were analyzed by western blot in HMGCL-5-8F cells treated with NAC (0mM, 5mM) for 24 h. GAPDH was an internal control. Data are mean ±SD (n=3). (*p<0.05; **p<0.01)