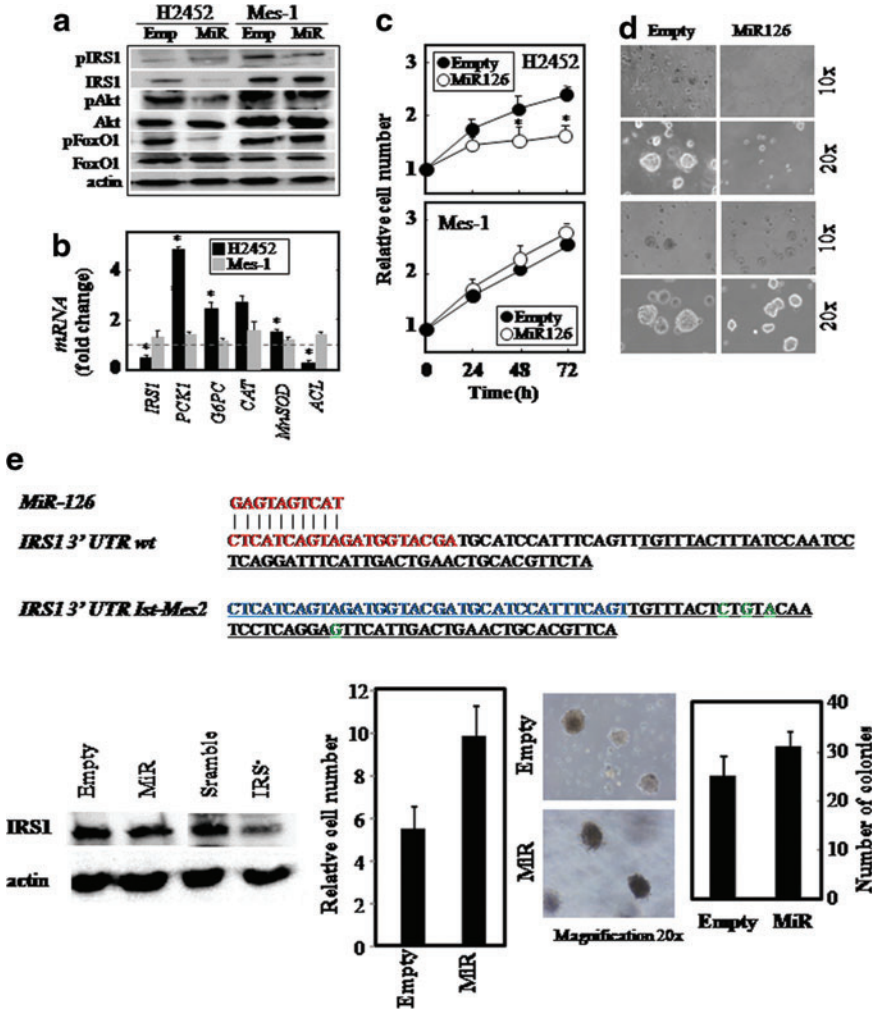

SUPPLEMENTARY FIG. S4. MiR126 suppresses the malignant phenotype in H2452 cells but not in Mes-1 and Ist-Mes2 cells. Empty plasmid- and MiR126-transfected H2452 and Mes-1 cells were evaluated for the level of expression of pIRS1 (S307), IRS1, pAkt, Akt, pFoxO1 (T24) and FoxO1 **(a)**, and the level of the mRNA of IRS1, the gluconeogenesis genes PCK1 and G6PC, and oxidative defense genes CAT and MnSOD, as well as ACL **(b)**. The cells were also assessed for their proliferation rates using the MTT assay **(c)** and the colony-forming activity **(d)**. Ist-Mes2 cells with truncated IRS1 lacking the MiR126-binding site at the 3′-UTR of IRS1 (blue sequence) were transfected with MiR126 or siRNA against IRS1. IRS1 protein levels were determined by western blotting. The empty plasmid-transfected Ist-Mes2 and their MiR126-transfected counterparts were evaluated for cell growth and colony-forming activity **(e)**. The results are the mean ± SD of three experiments performed in duplicate; images are representative of three independent experiments. Comparisons among and between groups were determined by one-way ANOVA with Tukey *post-hoc* analysis, and by the two-tailed Student's *t*-test, respectively. The symbol ''*'' indicates significantly different values with $p < 0.05$. FoxO1, Forkhead box O1; G6PC, glucose-6-phosphatase catalytic; MTT, 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide; PCK1, phosphoenolpyruvate carboxykinase 1; siRNA, small interfering RNA.