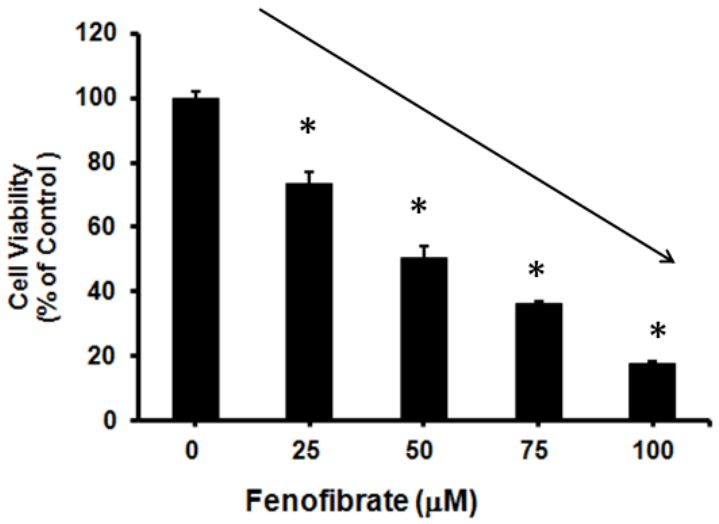
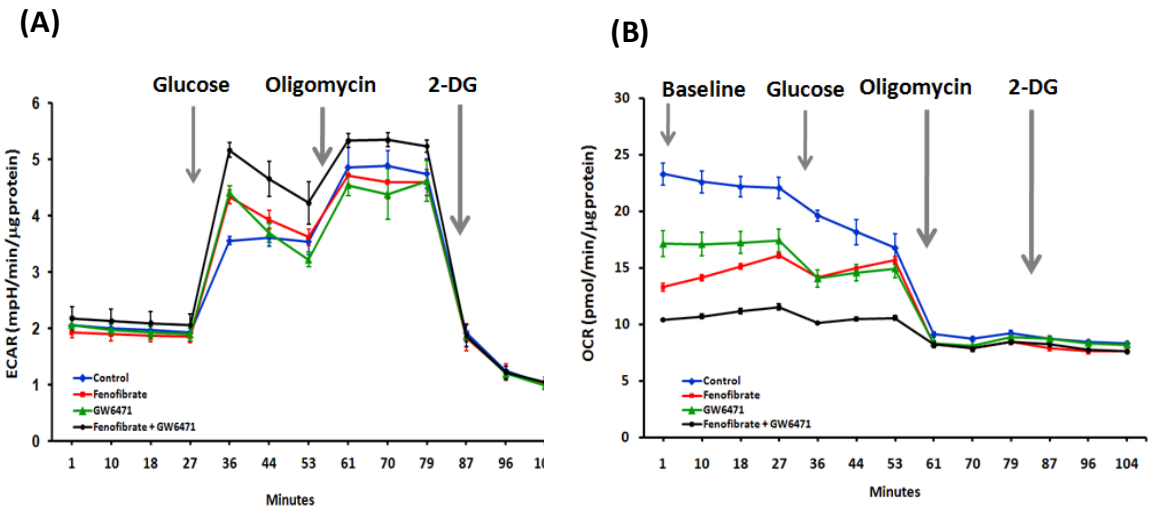


Supplementary Fig. 1. Fenofibrate inhibits cell growth in SAS oral cancer cells. The cells were treated with various concentrations of fenofibrate for 24 hrs, and then, the cell numbers were calculated by the trypan blue exclusion method. Cell viability is expressed as the percentage of the control value at 0 h. All data are shown as the mean ± S.D. from at least three independent experiments. Asterisks represent a significant difference from the control (*P<0.001, one-way ANOVA).



Supplementary Fig. 2. Effects of fenofibrate, GW6471, and fenofibrate+GW6471 on ECAR and OCR in SAS cells through glucose stress test. The SAS cells were pretreated with DMSO (control), fenofibrate (50 μM), GW6471 (20 μM), and fenofibrate (50 μM) + GW6471 (20 μM) for 2 hrs. Assays were initiated by replacing growth medium with XF medium (Seahorse Bioscience), free of sodium bicarbonate and glucose for 32 mins. Then, ECAR and OCR were monitored after sequential injections of glucose (10 mM) for 24 mins, oligomycin (1μ M) for 24 mins, and 2-DG (50 mM) for 24 mins by a Seahorse XFe24 analyzer. Fenofibrate is an agonist of PPARα which transcribes gene expression of β-oxidation related proteins. GW6471, which inhibits the binding of PPARα to DNA, is a specific PPARα inhibitor. Data are shown as mean±SD; n=4 in each group.