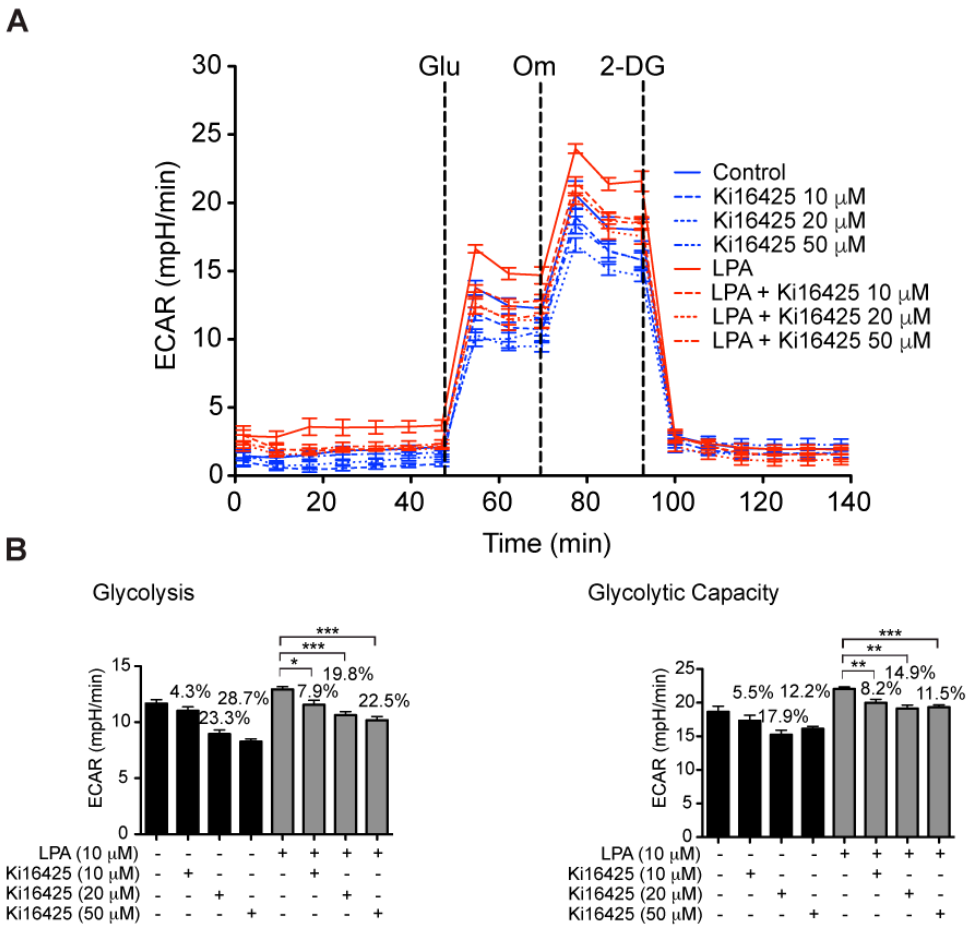

Supplemental Figure S1. LPA-induced glycolysis is mediated through its cognate receptors. TOV112D cells were pretreated with different concentration of Ki16425 for 1 hr, following which the cells were stimulated with LPA for 6 hrs. ECAR response to Glucose (Glu, 10 mM), Oligomycin (Om, 1 μM), and 2-DG (50 mM) was determined using XFe96 Seahorse analyzer. ECAR flux was plotted (*A*). Glycolytic rate and glycolytic capacity derived from the results are presented as bar graphs. Percentile change over the basal level is denoted over the bars of the chart (*B*). The experiment was repeated at least thrice and the results are from a representative experiment. Mean and SEM (n =11 to 12 parallel determinations) are shown along with the statistical significance, determined by Student's t test (*P<0.05, **P<0.005, ***P<0.0005). Results indicate that the glycolytic response to LPA is through the cell surface LPA-receptors (LPARs) since Ki16425, an antagonist for LPAR1, LPAR2, and LPAR3 inhibits LPA-induced glycolytic rate (*B, Left Panel*) and glycolytic capacity (*B, Right Panel*) of TOV112D ovarian cancer cells. Percentile changes over the basal levels of glycolysis and glycolytic capacity are denoted above the bars of the chart.