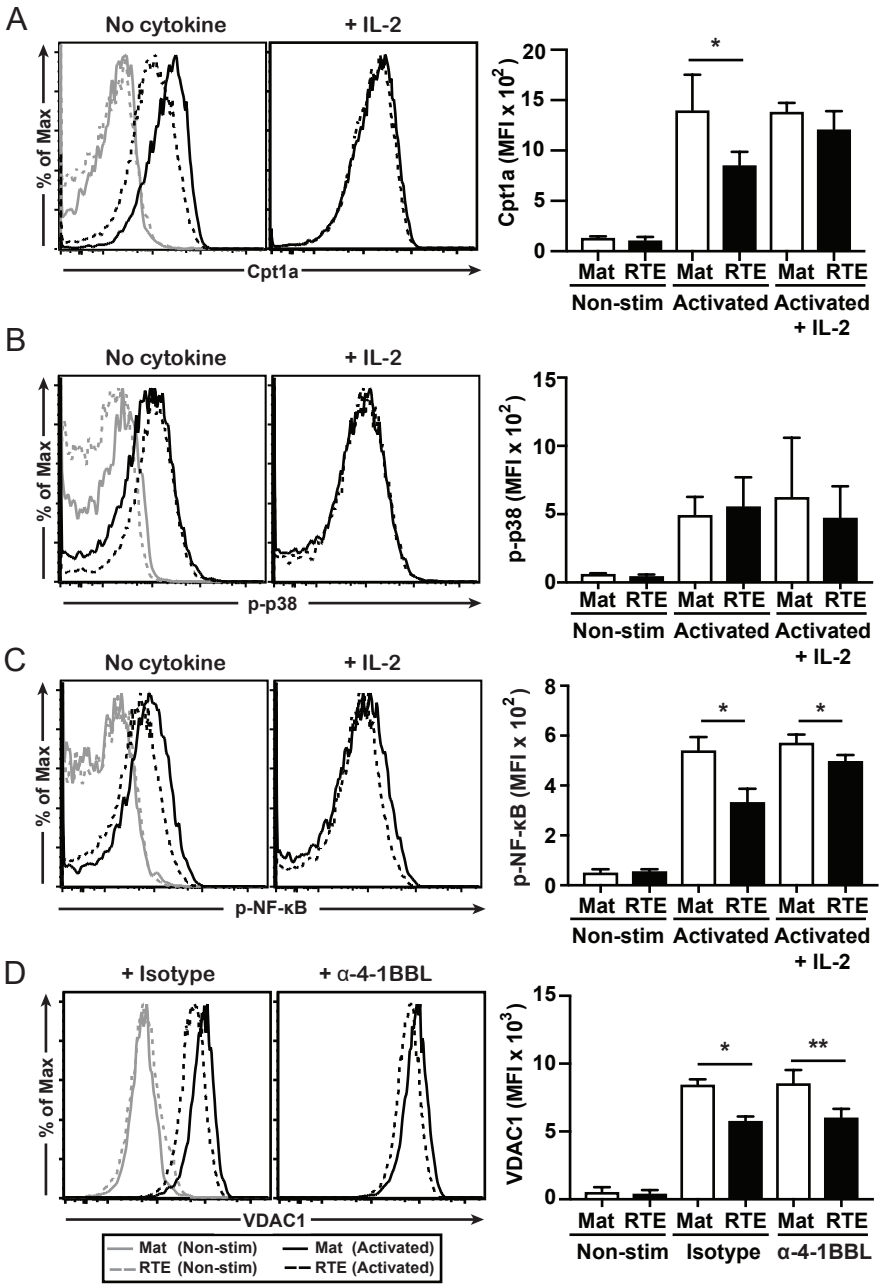
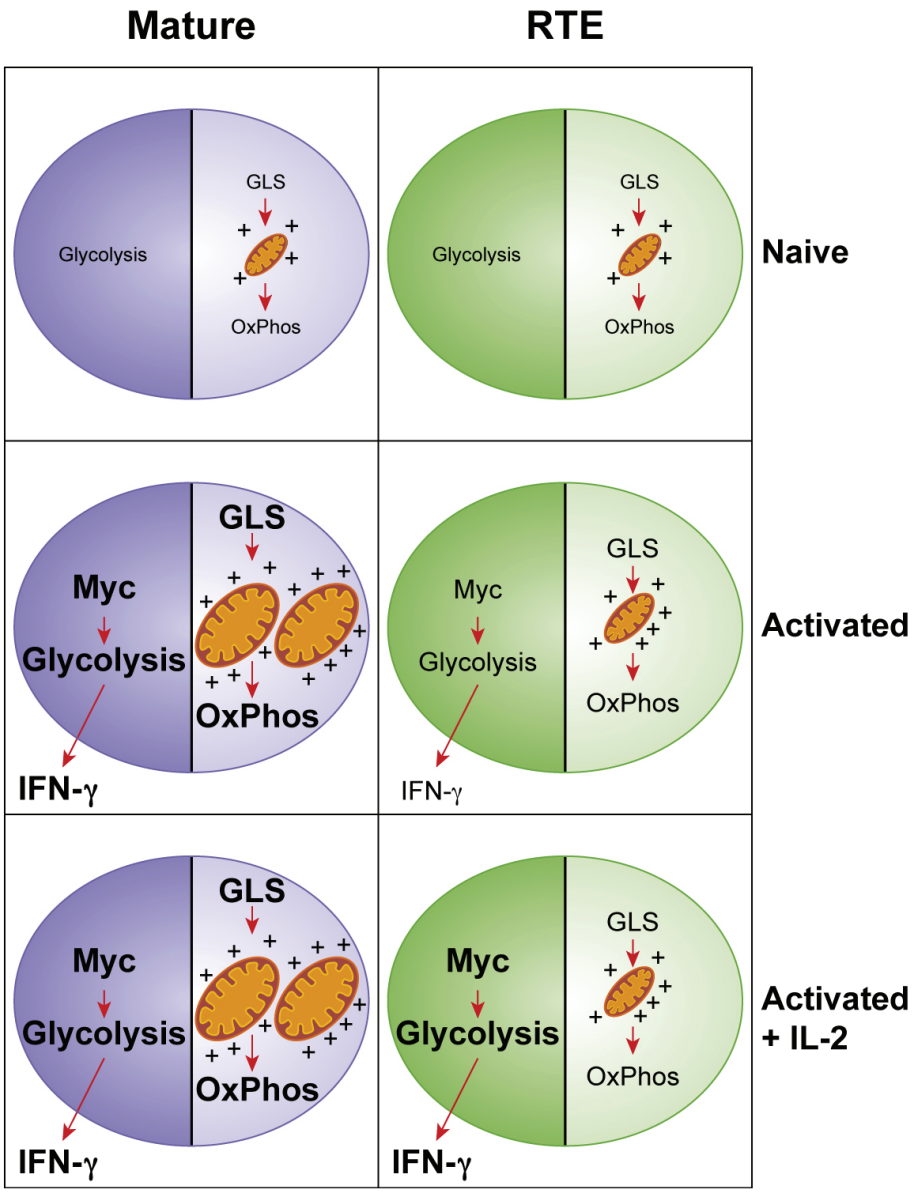
**Supplemental Figure 1**




**Supplemental Figure 1. Cpt1a and 4-1BB signaling are dispensable for the mitochondrial phenotype of RTEs (A)** Sort-purified CD8+ RTEs and MN T cells were freshly isolated (non-stim) or stimulated with Ag ± IL-2 in vitro for 3 days and the expression level of intracellular Cpt1a (A), p-p38 (B), and p-NF-κB (C) determined; representative histograms are shown on the left. The bar graphs on the right depict the average MFI ± SD from 3 independent experiments. (D) RTEs and MN T cells left unstimulated or activated in the presence of α-4-1BBL or an isotype control (both at 10 μg/mL) and VDAC1 levels determined; representative histograms are shown on the left. The bar graphs on the right depict the average MFI ± SD from 3 independent experiments. *$p < 0.05$ ** $p < 0.01$ by paired Student *t* test.

**Supplemental Figure 2**




**Supplemental Figure 2. Altered metabolism in antigen-activated RTEs**. Resting RTEs and mature T cells have similar metabolic profiles (top panels). However, upon activation in the absence of exogenous IL-2 (middle panels), RTEs display defective aerobic glycolysis and glutamine-fueled OxPhos relative to mature T cells. When RTEs are activated in the presence of exogenous IL-2 (bottom panels), aerobic glycolysis (and thus IFN-γ production) is restored. However, IL-2 fails to restore GLS expression and mitochondrial mass accumulation in RTEs.