Figure S1

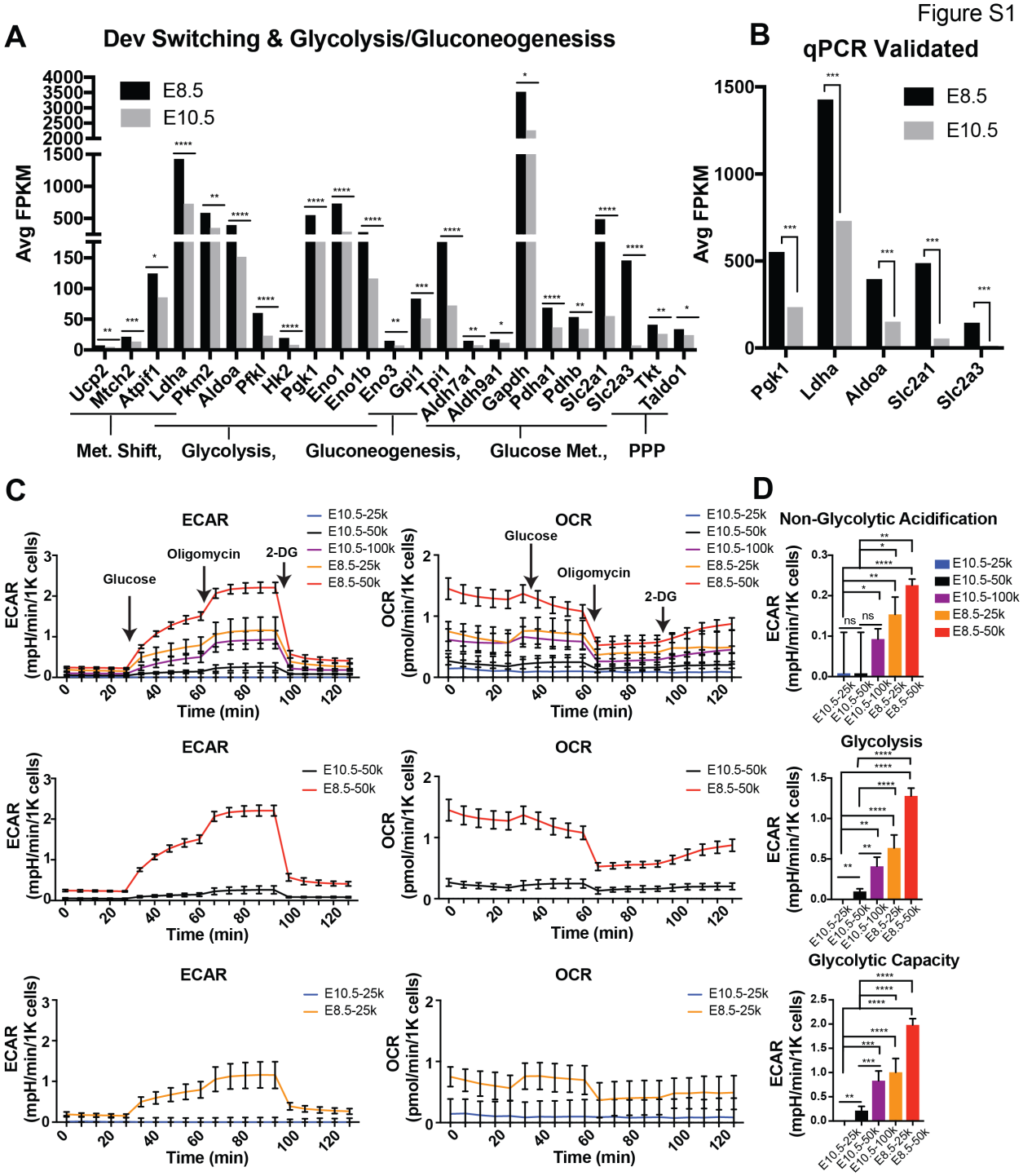

Figure S1. Gene and protein expression suggest metabolic switch away from glycolysis during E8.5 to E10.5 transition and glucose usage shifts in forebrain progenitors to decrease in glycolytic activity from E8.5 to E10.5. (**A**) Average FPKM values from mRNA sequencing data for genes associated with glycolysis, oxidative phosphorylation, and nucleotide biosynthesis through the pentose phosphate pathway (PPP). These pathways were enriched in the E8.5 neuroepithelium (N=2 biological replicates, each a pooled litter) with respect to the E10.5 neuroepithelium (N=2 biological replicates, each a pooled litter) (log_2fold change >0) cuffdiff v2 with q value: *, q < 0.1; **, q < 0.001; ***, q < 0.0001; ****, q < 0.00001; see Chau, et al 2018. (**B**) Average FPKM values for the individual components of these pathways (*pgk1*, *ldha*, *aldoa*, *slc2a1* (glut1), and *slc2a3* (glut3) that were validated by qPCR to be downregulated in E10.5 neuroepithelium. (**C**) Extracellular acidification (ECAR) and oxygen consumption rate (OCR) traces from an experiment comparing the effect of cellular density in the Seahorse Glycolysis Stress test (N=10 wells for experiments at 25k cells/ well; N=5 wells for experiments at 50k cells/ well and 100k cells, well); multiple t-tests ***, p<0.005; ****, p<0.001, error bars = SEM. (**C**) Glycolytic metrics from the different cellular density in Seahorse Glycolysis Stress test (N=10 wells for experiments at 25k cells/ well; N=5 wells for experiments at 50k cells/ well and 100k cells, well);multiple t-tests ***, p<0.005; ****, p<0.001, error bars = SEM. (**D**) This transcriptional enrichment was also reflected in protein levels. Scale bar (E) 10 μm.

Figure S2

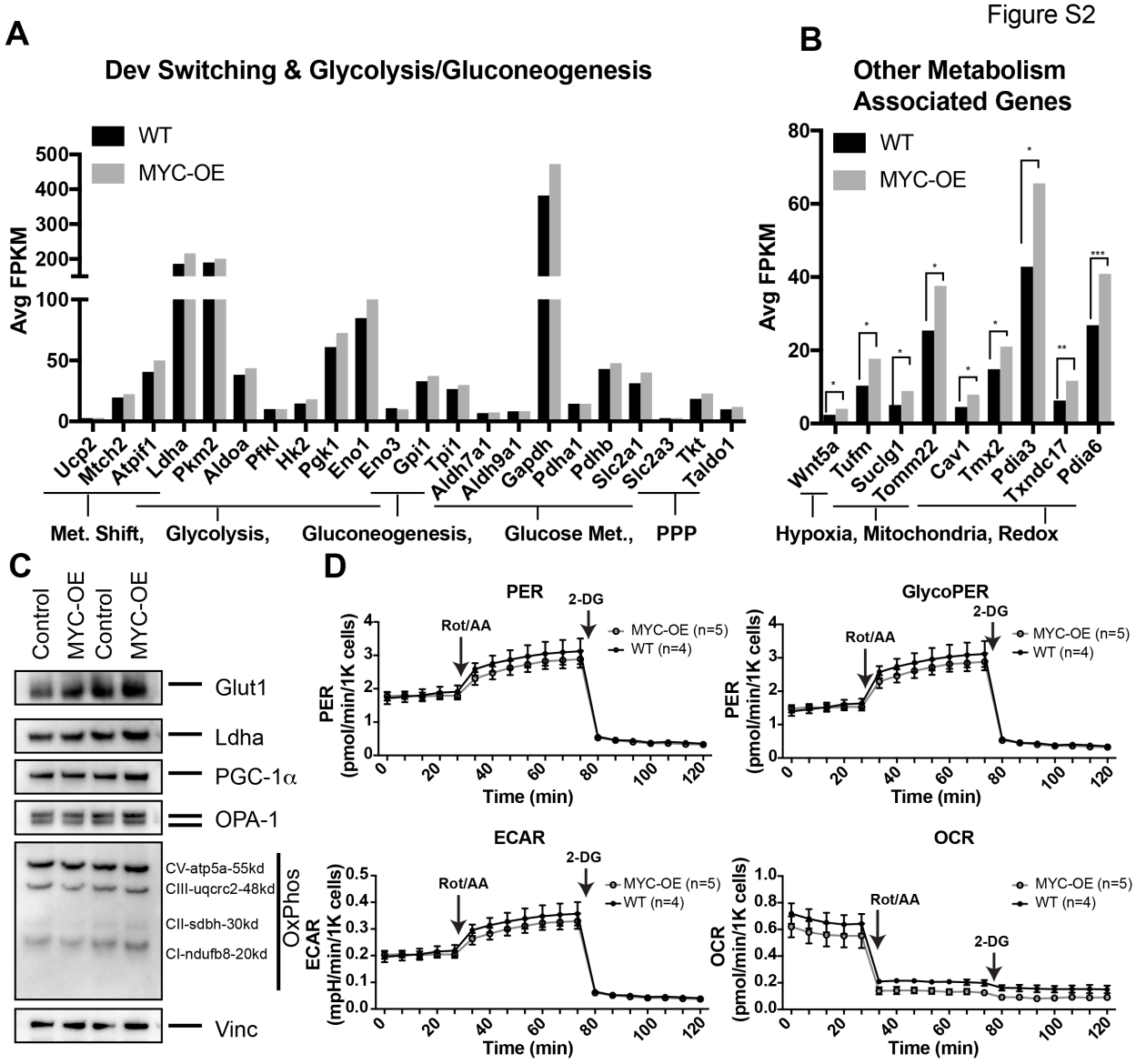

Figure S2. Mitochondrial morphology in the neuroepithelium, but not glycolysis, is dependent on developmental downregulation of MYC. (**A**) Average FPKM values from mRNA sequencing data for genes associated with glycolysis, oxidative phosphorylation, and nucleotide biosynthesis through the pentose phosphate pathway (PPP) were unchanged after forced c-MYC expression in the E12.5 neuroepithelium (N=3 biological replicates) with respect to the control E12.5 neuroepithelium (N=3 biological replicates) (log_2fold change >0) cuffdiff v2, ns: $q > 0.1$; see Chau, et al 2018. (**B**) Average FPKM values for genes associated with metabolism that were affected by c-MYC overexpression in the E12.5 neuroepithelium (log_2fold change >0) cuffdiff v2 with q value: *, $q < 0.1$. (**C**) Immunoblotting for canonical mitochondrial morphology component proteins PGC-1α and OPA1, and OxPhos complex components. Vinculin was used as a loading control. (**D**) Traces of proton efflux rate (PER), PER from glycolysis (GlycoPER); extracellular acidification rate (ECAR), and oxygen consumption rate (OCR) for the Seahorse Glycolytic Rate Assay in neuroepithelial cells after prolonged c-MYC expression; N=2 experiments, N=4 control animals and N=5 MYC-OE animals, error bars = SEM.