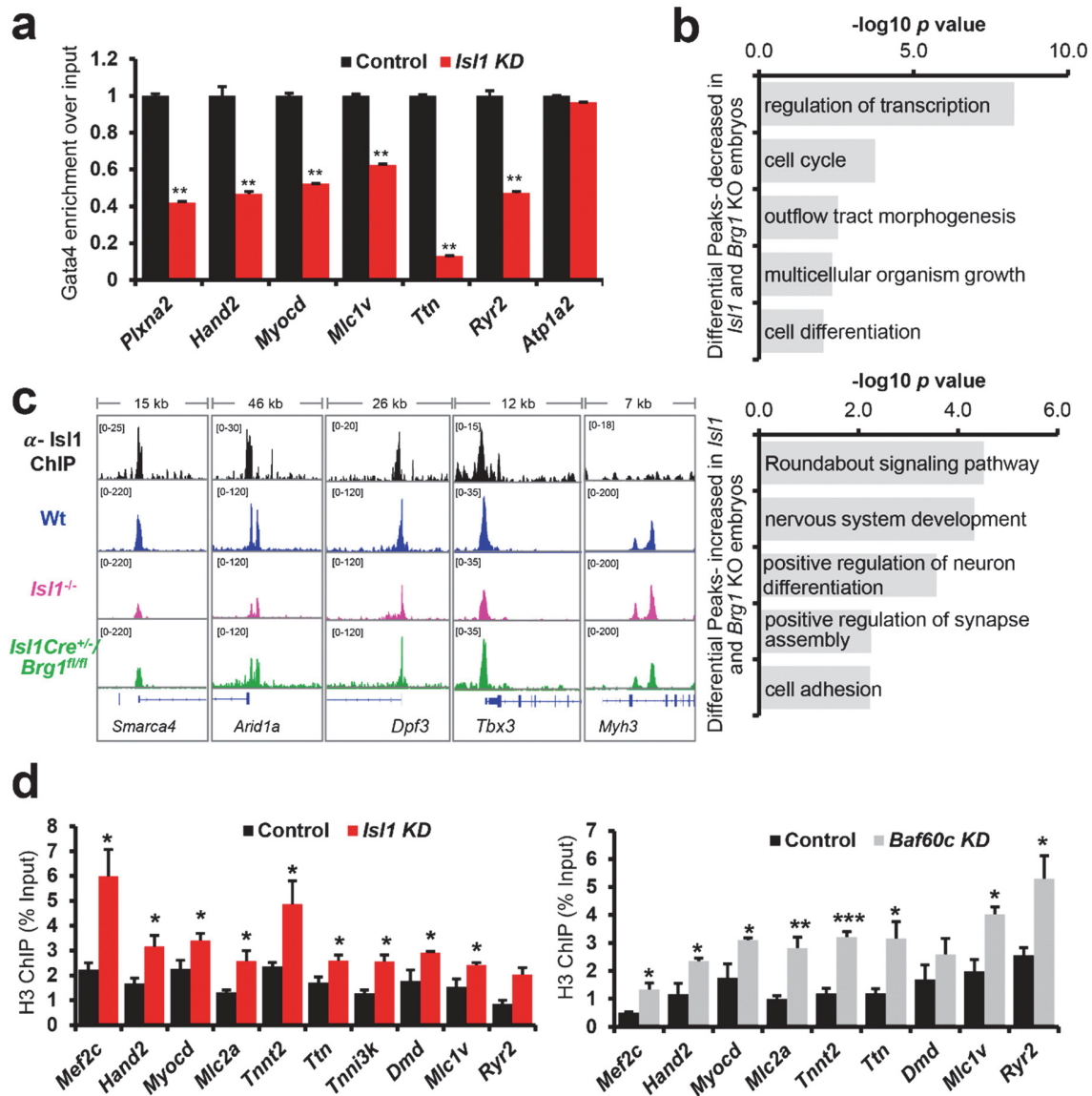




Supplementary information, Figure S7 | *Isl1* and the *Brg1*-*Baf60c* complex induce chromatin reorganization in CPCs. (a) ChIP-qPCR to analyze GATA4 occupancy at *Isl1*- bound sequences in control and *Isl1* KD CPCs. *Atp1a2*, a gene not bound by *Isl1*, serves as a negative control. **(b)** GO terms enriched in ATAC-Seq peaks decreased (top panel) or increased (lower panel) in both *Isl1*^{-/-} and *Isl1*-*Cre*^{+/-}*Brg1*^{fl/fl} embryos versus control embryos (n=3). **(c)** Examples of genes showing decreased ATAC-Seq signals at *Isl1* binding sites in both *Isl1*^{-/-} and *Isl1*-*Cre*^{+/-}*Brg1*^{fl/fl} embryos (*Smarca4*, *Arid1a*), genes bound by *Isl1* with decreased chromatin accessibility

only in *Isl1*^{-/-} knockout embryos (*Tbx3*, *Dpf3*) and genes not bound by Isl1 showing no change in ATAC signal in both *Isl1*^{-/-} and *Isl1-Cre*^{+/-}*Brg1*^{fl/fl} embryos (*Myh3*). Genome tracks of Isl1 ChIP-Seq and ATAC-Seq signals of wild-type, *Isl1*^{-/-} and *Isl1-Cre*^{+/-}*Brg1*^{fl/fl} embryos are presented. **(d)** ChIP-qPCR analysis of relative H3 occupancy at selected Isl1 target genes in control and *Isl1* knockdown CPCs (left) and control and *Baf60c* knockdown CPCs (right). Data are mean $\pm$ SEM, n=3.