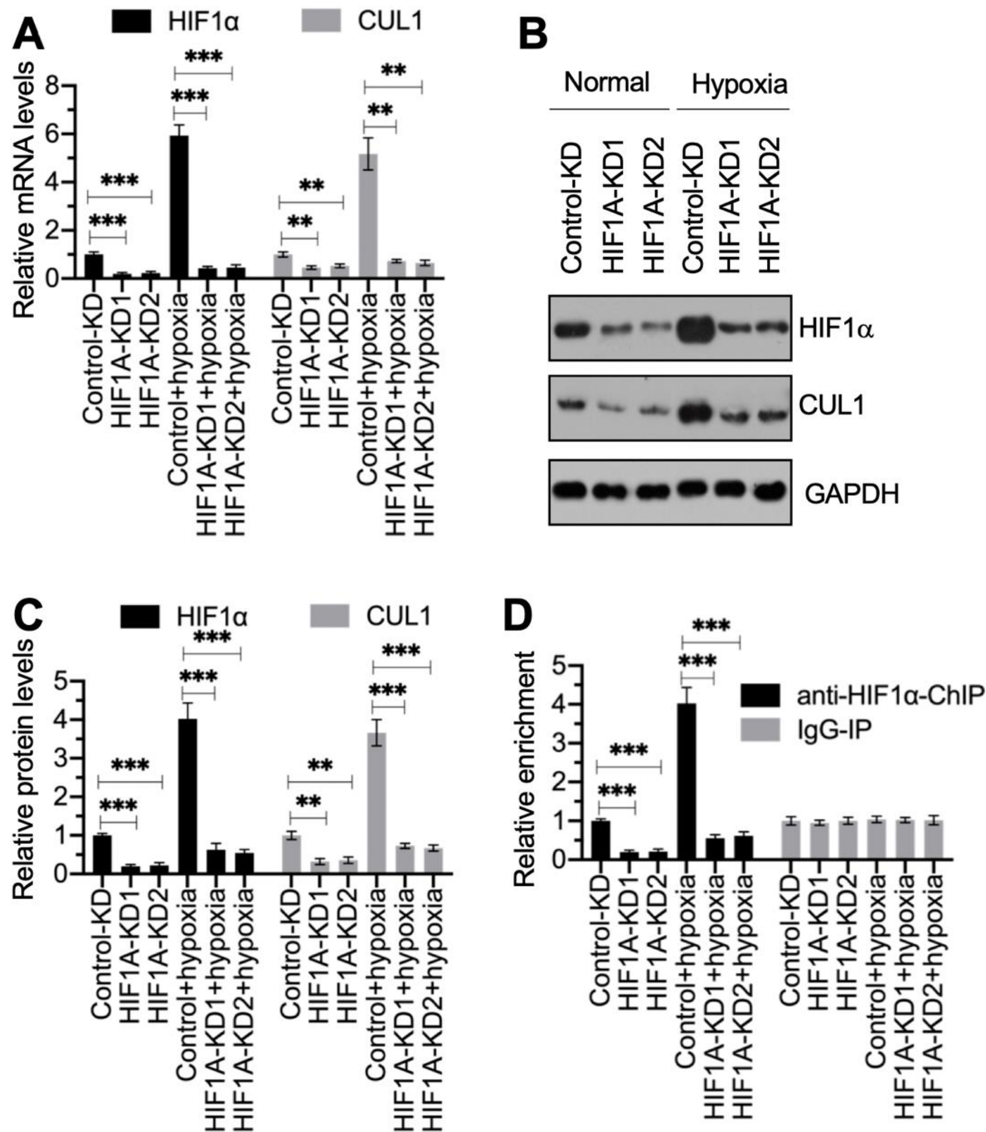

S9 Fig. Hypoxia induced the expression of HIF1α and CUL1. (A) The relative mRNA levels of *HIF1A* and *CUL1*. The Control-KD and HIF1A-KD (#1 and #2) cells in CCD-18Co background were treated with or without hypoxia, followed by quantitative reverse-transcription polymerase chain reaction analyses to detect the mRNA levels of *HIF1A* and *CUL1*. (B, C) The protein levels of HIF1α and CUL1. Total cell extracts from cells in (A) were subjected to immunoblots to examine protein levels of HIF1α, CUL1, and glyceraldehyde 3-phosphate dehydrogenase (GAPDH) (B). The protein signals were quantified and normalized to GAPDH (C). (D) The relative enrichment of HIF1α on the promoter of *CUL1*. Cells in (A) were used for ChIP assays with anti-HIF1α and IgG (negative control). The enriched DNA was subjected to quantitative reverse-transcription polymer-ase chain reaction analyses to detect the enrichment of HIF1α. The relative enrichment of HIF1α and IgG in each sample was normalized to that in the Control-KD cells. **$p < 0.01$, ***$p < 0.001$.