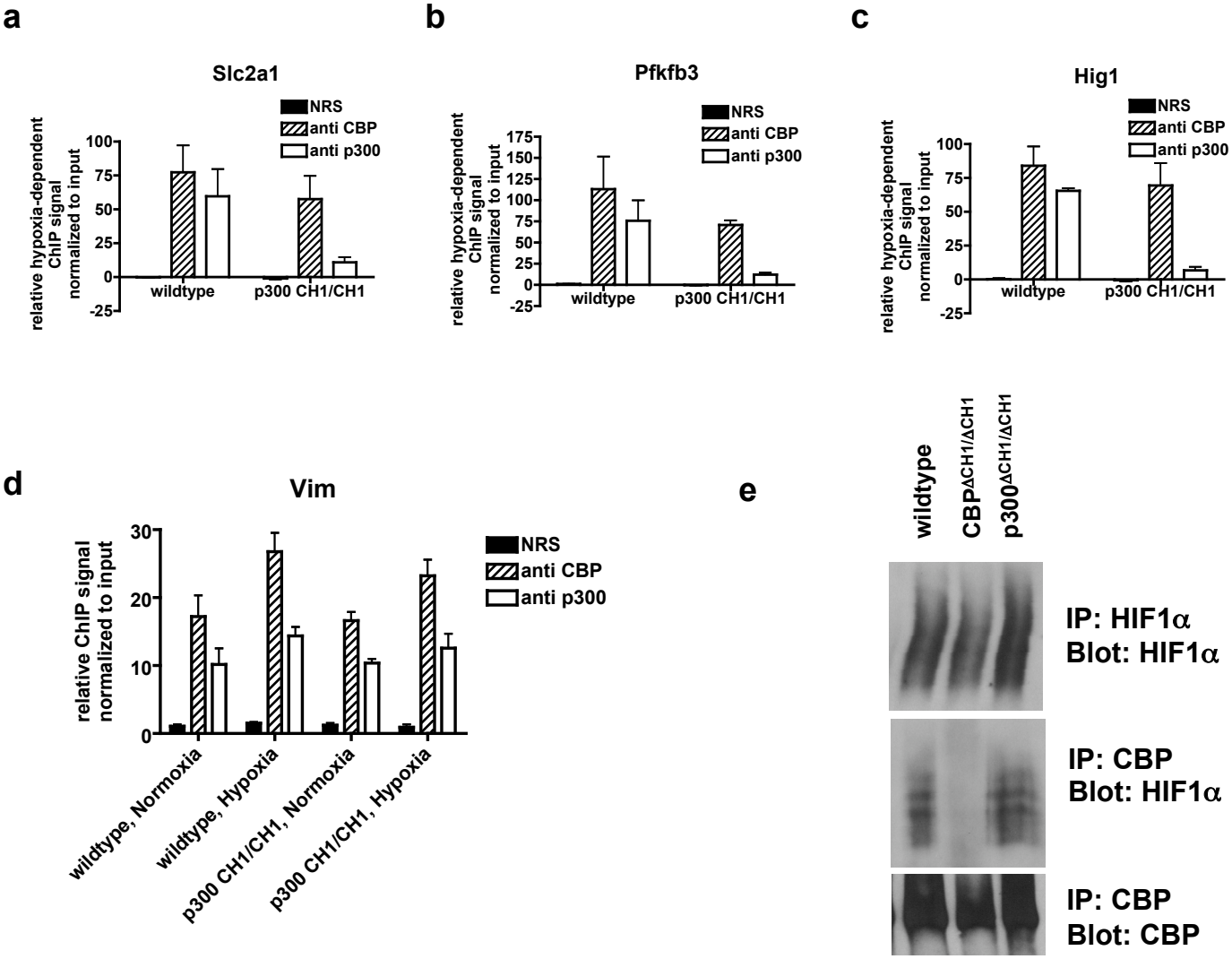
Supplementary Fig. S2 (Kasper)



Supplementary Fig. S2. The ΔCH1 mutation attenuates hypoxia-dependent recruitment of p300 to HIF-binding sites but not to the control gene *Vim*. **a-d,** Quantitative ChIP assays using WT and $p300^{\Delta CH1/\Delta CH1}$ MEFs treated four hours with normoxia or hypoxia (mean ± S.E.M. N=3, 3 independent experiments). Control (NRS) and specific (anti-CBP, anti-p300) IP antisera indicated. Hypoxia-dependent ChIP signal was determined by subtracting the normoxia signal from the hypoxia signal after normalizing to input DNA signal for HIF-target genes (**a-c**). *Vim* is a non-HIF target gene (**d**). **e,** Co-immunoprecipitation of HIF1α with CBP in MEFs treated 2 hrs with 100μM dipyridyl is attenuated by the ΔCH1 mutation. Immunoprecipitations were performed as described in Yang *et al*. (1998) MCB 18:2218-2229, except that buffers for nuclear extracts and IPs were made without EDTA or EGTA using HIF-1α (HI α67) antibody from Novus and CBP A-22 and C-20 antibodies from Santa Cruz.