Fig. S1

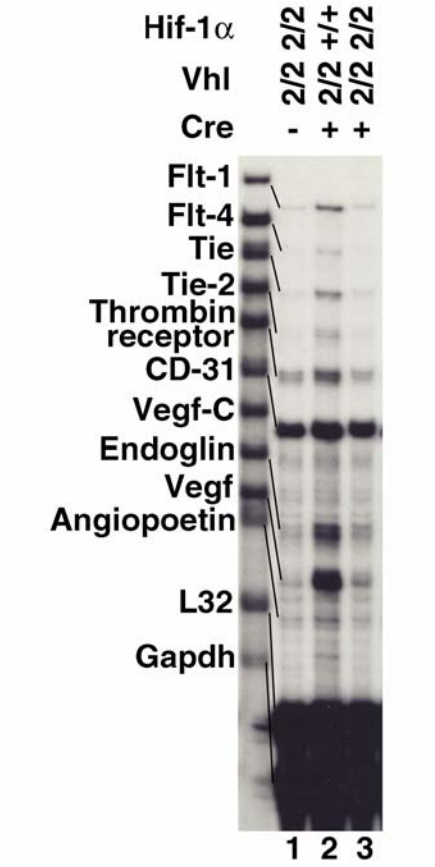

Figure S1: Hif-1 dependence of angiogenic gene expression in *Vhlh* deficient thymi. Expression analysis of *Vegf-A, Vegf* receptors *Flt-1* and *-4* and endothelial cell markers *CD-31* and *endoglin* by RPA using total RNA.

Fig. S2

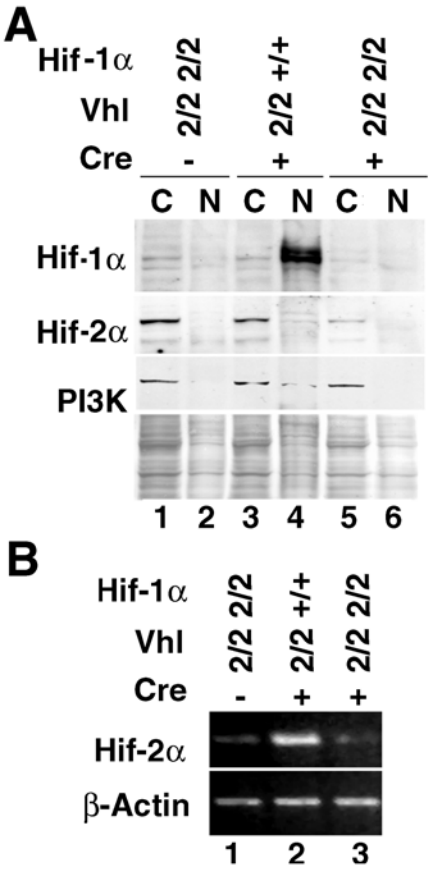

Figure S2: Hif-2α is non-detectable in nuclear fraction from *Vhlh*$^{-/-}$ thymocytes. (A) Cytoplasmic trapping of Hif-2α in *Vhlh* deficient thymocytes. Shown are protein levels of Hif-1α and Hif-2α in cytoplasmic (C) and nuclear fractions (N) using Western blot analysis. Staining with Ponceau S is shown to demonstrate equal loading. Cytoplasmic PI3 kinase was used to assess nuclear fraction purity. **(B)** *Hif-2α* mRNA levels are induced in *Vhlh*$^{-/-}$ thymocytes in a Hif-1 dependent manner. Shown are results from semi-quantitative RT-PCR studies in FACS sorted DP thymocytes. β-actin is used to normalize mRNA.

Fig. S3

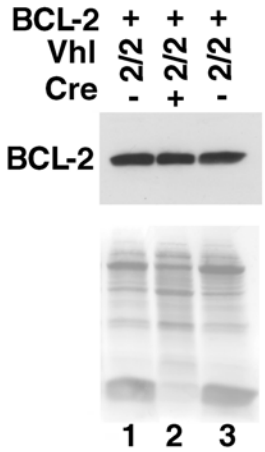

Figure S3: *BCL-2* transgene expression in *Vhlh*$^{-/-}$ thymocytes. Human BCL-2 transgene expression in mutant and control mice by Western blot using a species-specific anti-human BCL-2 antibody.

Fig. S4

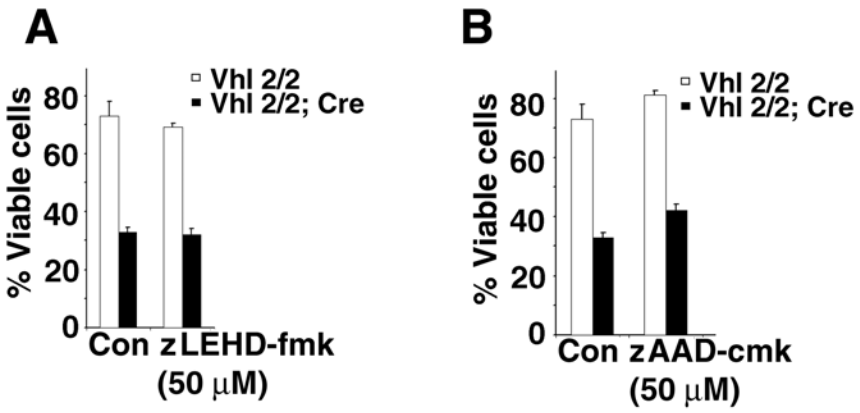

Figure S4: Inhibition of caspase-9 or granzyme-B does not prevent thymocyte death *in vitro*. Shown is the percentage of viable cells in cultures of control and *Vhlh* deficient thymocytes treated for 24hr with **(A)** caspase-9 inhibitor zLEHD-fmk and **(B)** Granzyme B inhibitor zAAD-cmk. Viability was assessed with 7-AAD staining. DMSO treated cells were used as control (Con). Shown are the mean values $\pm$ SD for n= 3 mice, individual experiments were carried out in triplicates.