Supplementary Figures

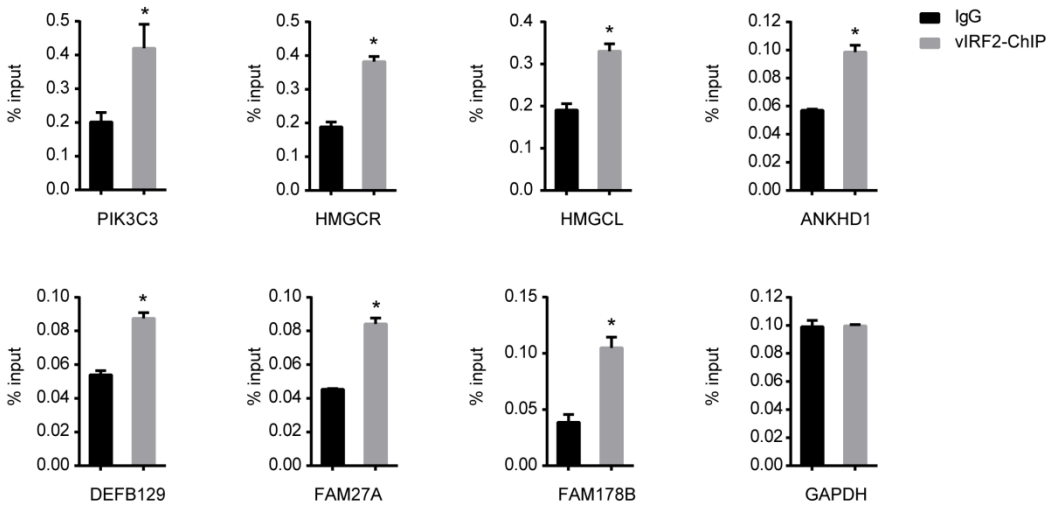

Fig. S1. Binding of vIRF2 to its target promoters were validated by ChIP-qPCR. Data were shown as mean ± s.e.m., n=3. * $p<0.05$

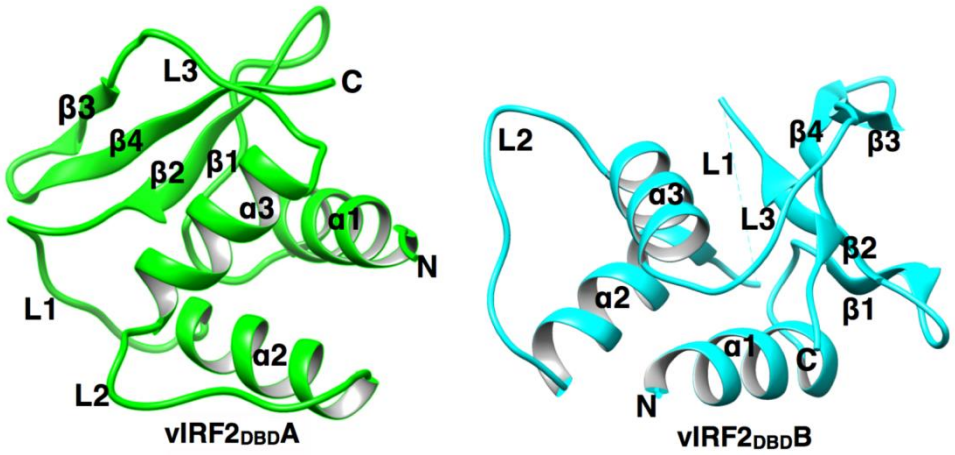

Fig. S2. Interaction of vIRF2$_{DBD}$ with consensus vIRF2-binding motifs *in vitro*. (A) Validation of direct vIRF2-DNA binding by EMSA using vIRF2$_{DBD}$ and the DNA probe1 (representing LANA-binding motif 1), probe 2 (representing vIRF2-binding motif 1) and probe 3 (vIRF2-binding motif 2). vIRF2$_{DBD}$ induced significant gel shift of probe 2 or probe 3 in a dose dependent manner, but not of probe 1. (B) Validation of direct vIRF2-DNA binding by EMSA using vIRF2$_{DBD}$ and the DNA probe representing vIRF2-binding motif 2. Wt vIRF2$_{DBD}$ induced significant gel shift of the probe in a dose dependent manner, however R82A, R85A or R82A/ R85A mutant induced much weaker gel shifts compared to wild type vIRF2$_{DBD}$.

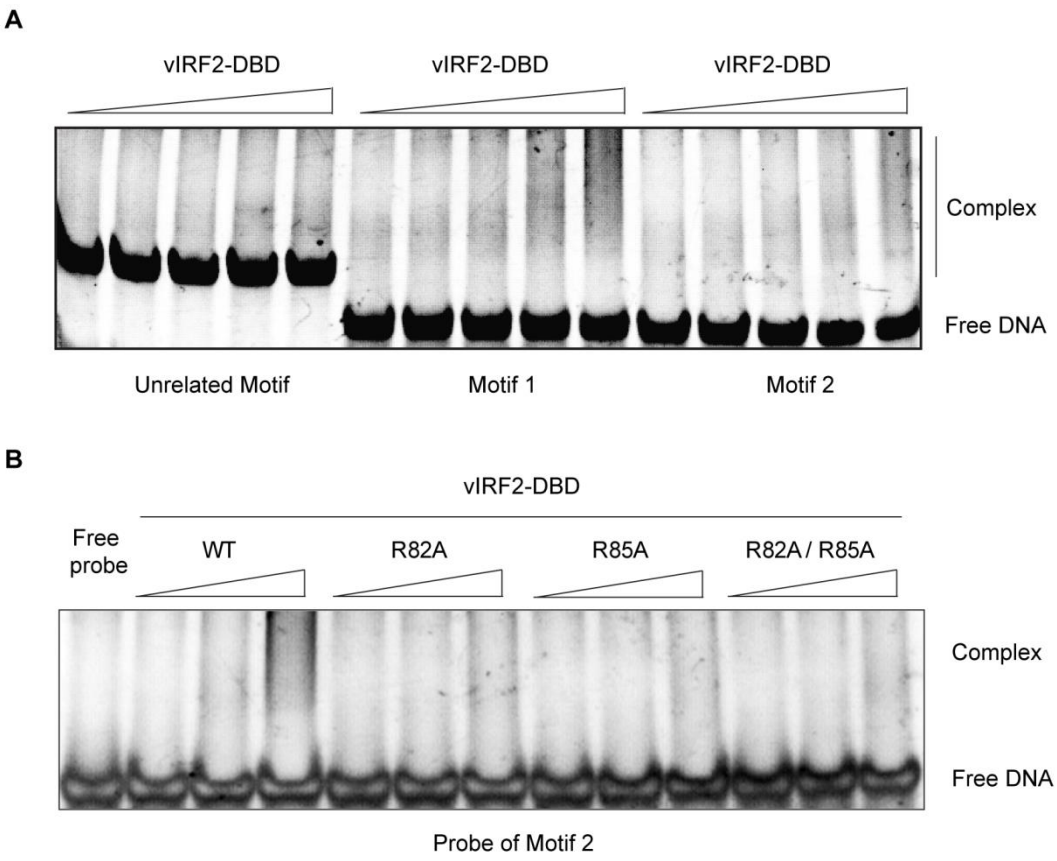

Fig. S3. Overall view of vIRF2$_{DBD}$ structure. There were two molecules in the asymmetric unit, named vIRF2$_{DBD}$A (green) and vIRF2$_{DBD}$B (cyan), respectively. The residues Arg42- Gly50 without electronic density were connected by a dashed line.

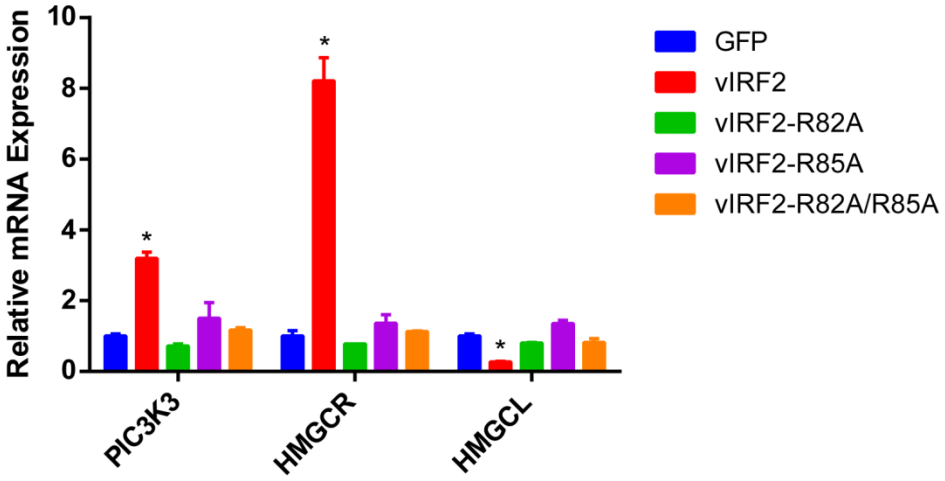

Fig. S4. mRNA level of *PIK3C3* and *HMGCR* were significantly up-regulated, while *HMGCL* was significantly down-regulated by vIRF2 expression, however, R82A, R85A or R82A/ R85A mutants lost their function in transcription regulation of these three genes. The mRNA level was measured by qRT-PCR. Data were shown as mean ± s.e.m., n=3. * $p<0.05$