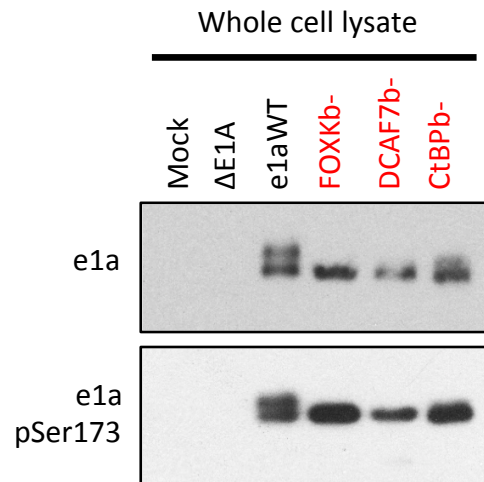
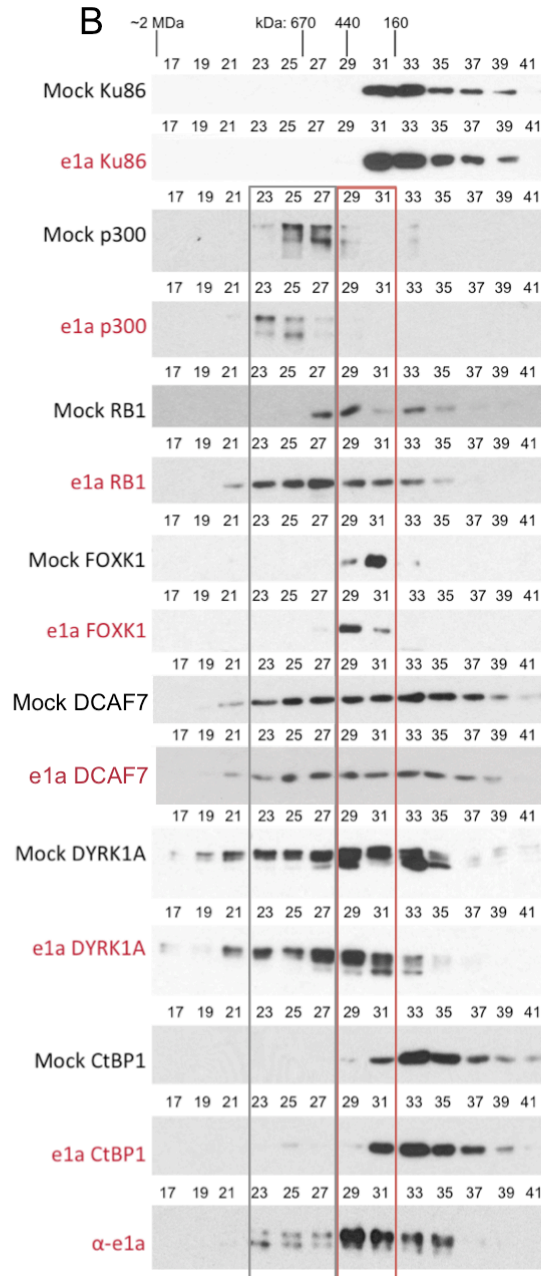
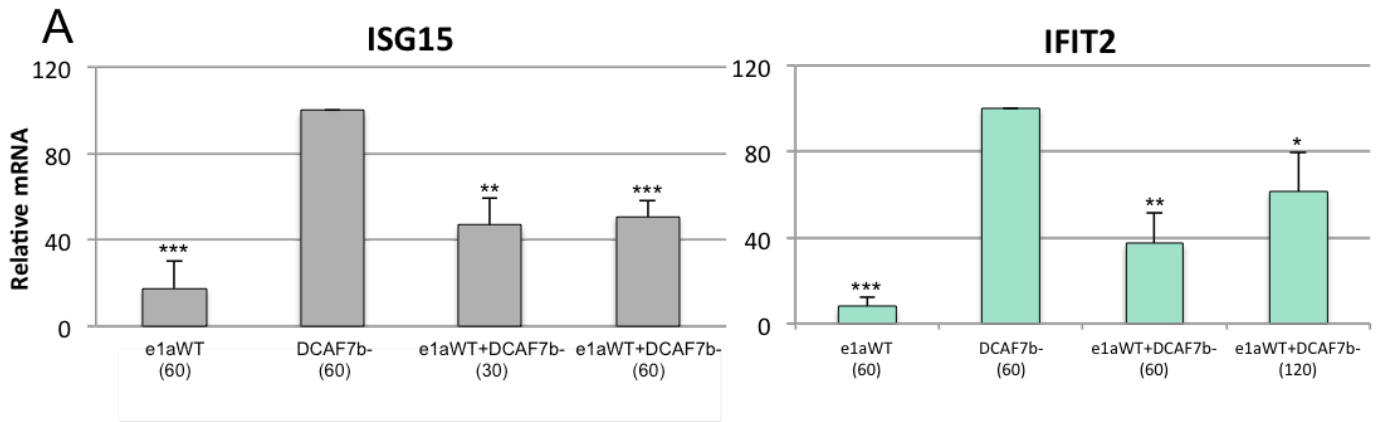


Supplemental Figures

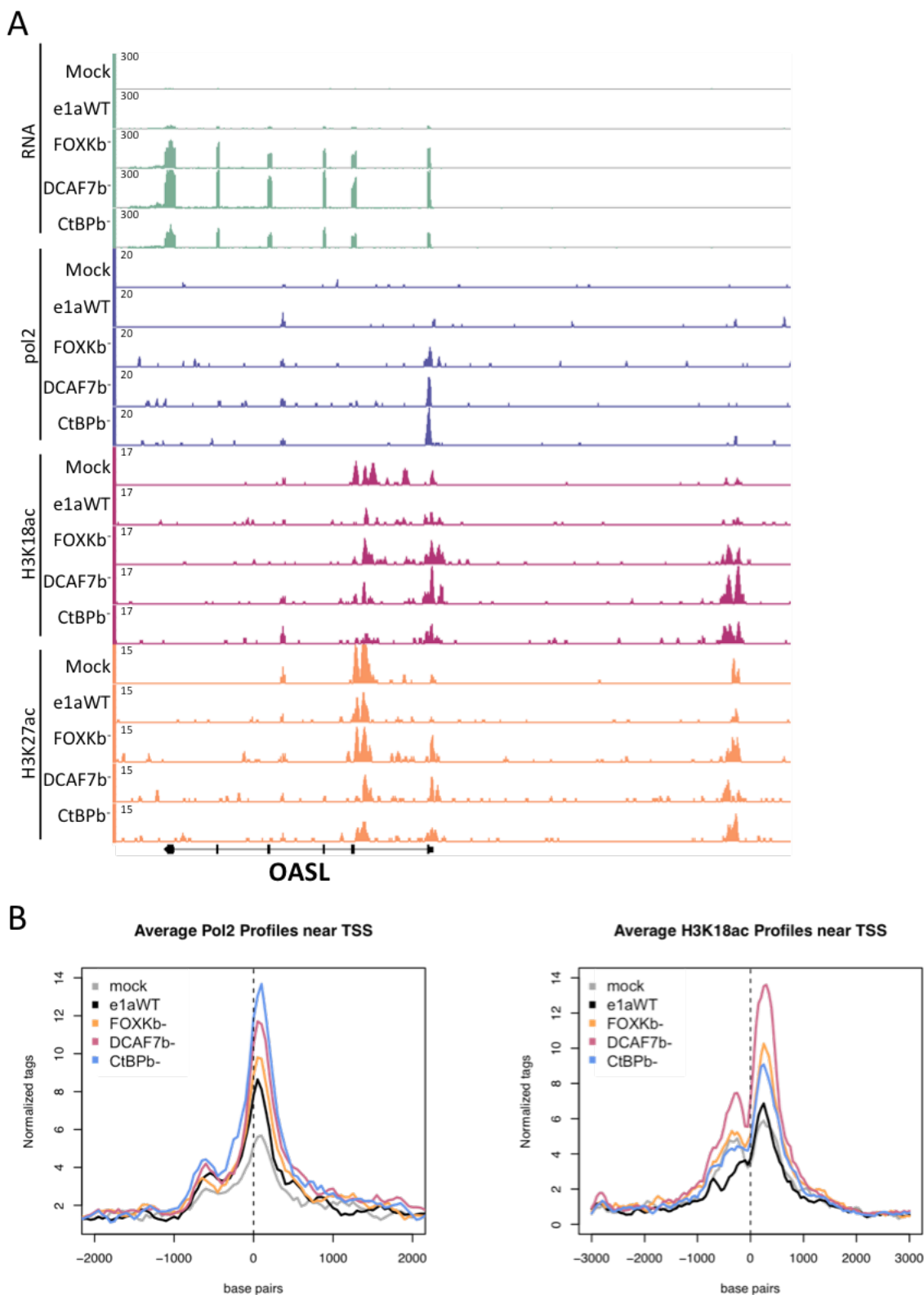


**Fig S1. The e1a C-terminal mutations interfere with phosphorylation at Ser89 but not Ser173. Related to Fig 1**  
Western blot of protein extract from HBTEC 24h p.i. with the indicated vectors using monoclonal antibodies against e1a (M58, top) and e1a pSer173 (bottom).

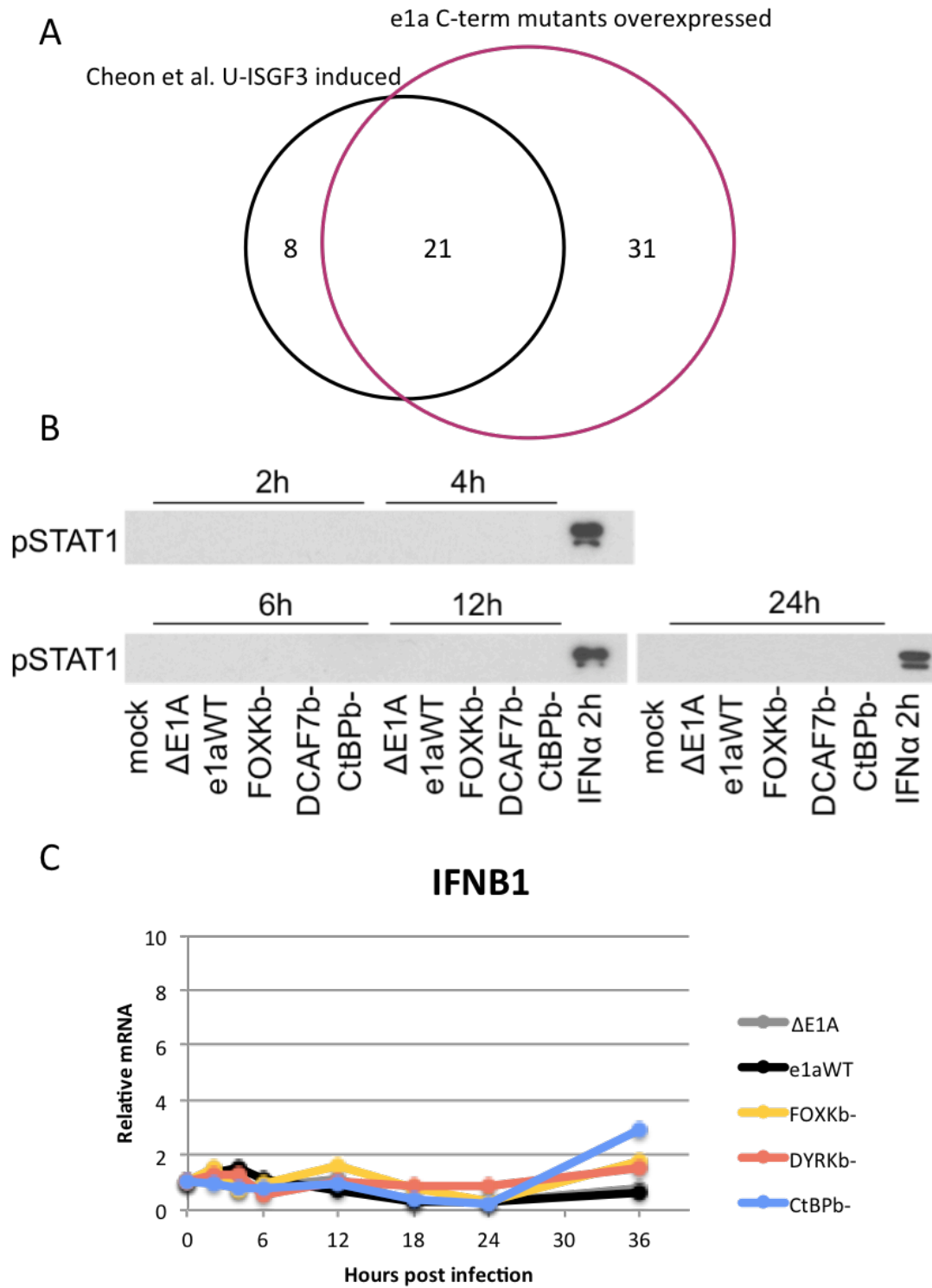


**Figure S2. e1aWT and DCAF7b<sup>-</sup> coinfection and e1a nuclear protein complexes. Related to Fig 4**

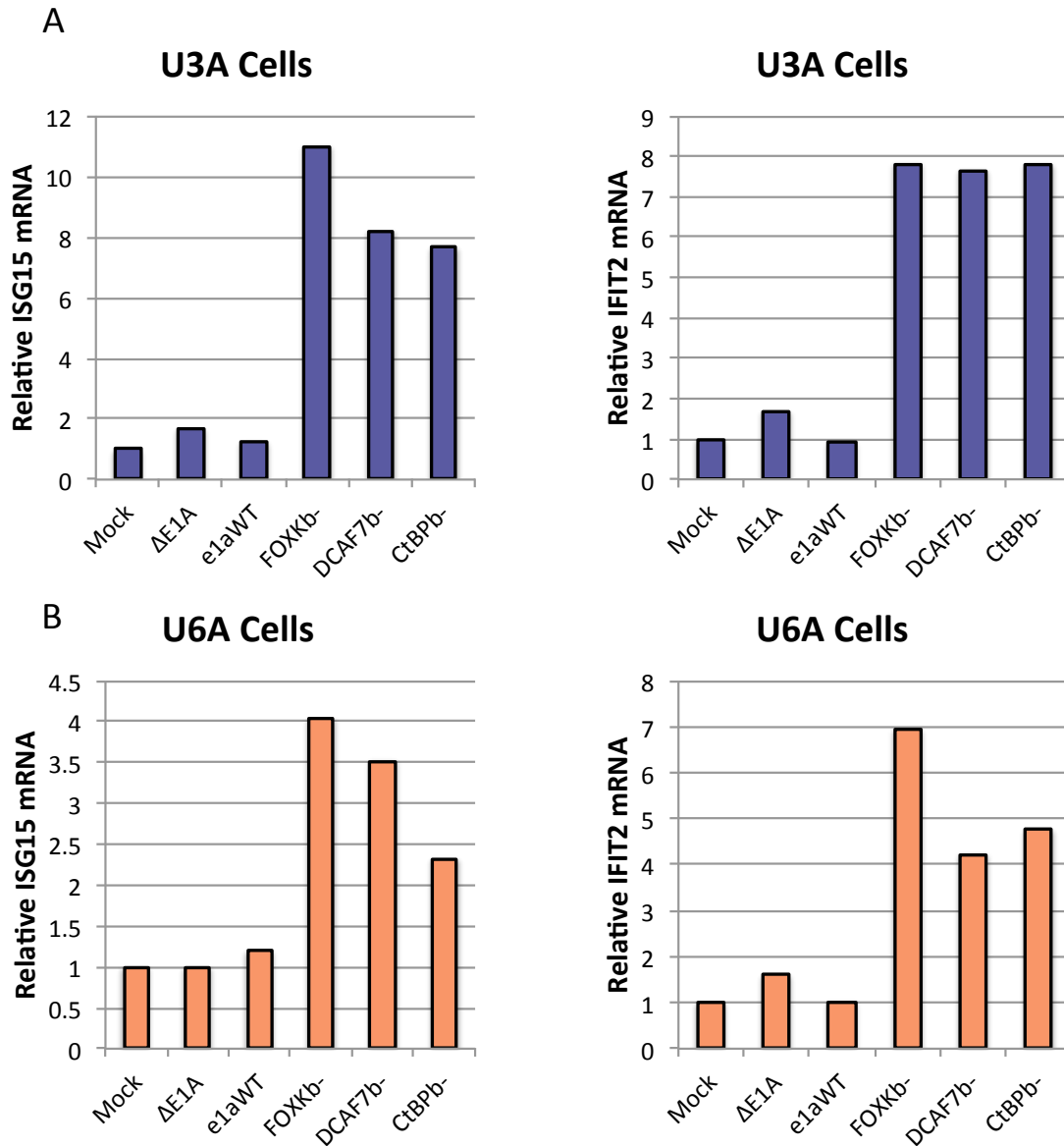
(A) Relative levels of ISG15 and IFIT2 mRNA as assayed by qRT-PCR from HBTECs infected or coinfecting with indicated vectors. moi for individual vectors indicated in parentheses. Data are represented as averages of percent of DCAF7b<sup>-</sup> (moi 60) activation + S.D. (B) Western blots of Superose 6 column fractions from mock or e1aWT-vector infected HeLa nuclear extract (24 h p.i.). Non-e1a interacting nuclear factor Ku86 is shown as a control.



**Figure S3. Chromatin marks related to transcriptional activation at e1a C-terminal induced ISGs. Related to Fig 5**  
**(A)** Genome browser track of RNA-seq and ChIP-seq enrichment upstream of and across the OASL gene from HBTEC infected for 24h. **(B)** Metagene plots showing average tag density of Pol2 or H3K18ac ChIP-seq enrichment around TSS of 52 genes expressed 2-fold higher by all three e1a C-terminal mutants using chromatin from cells mock-infected or infected with indicated e1a expressing Ad5 vector for 24 h. Data was normalized so there were equal numbers of mapped reads across samples.

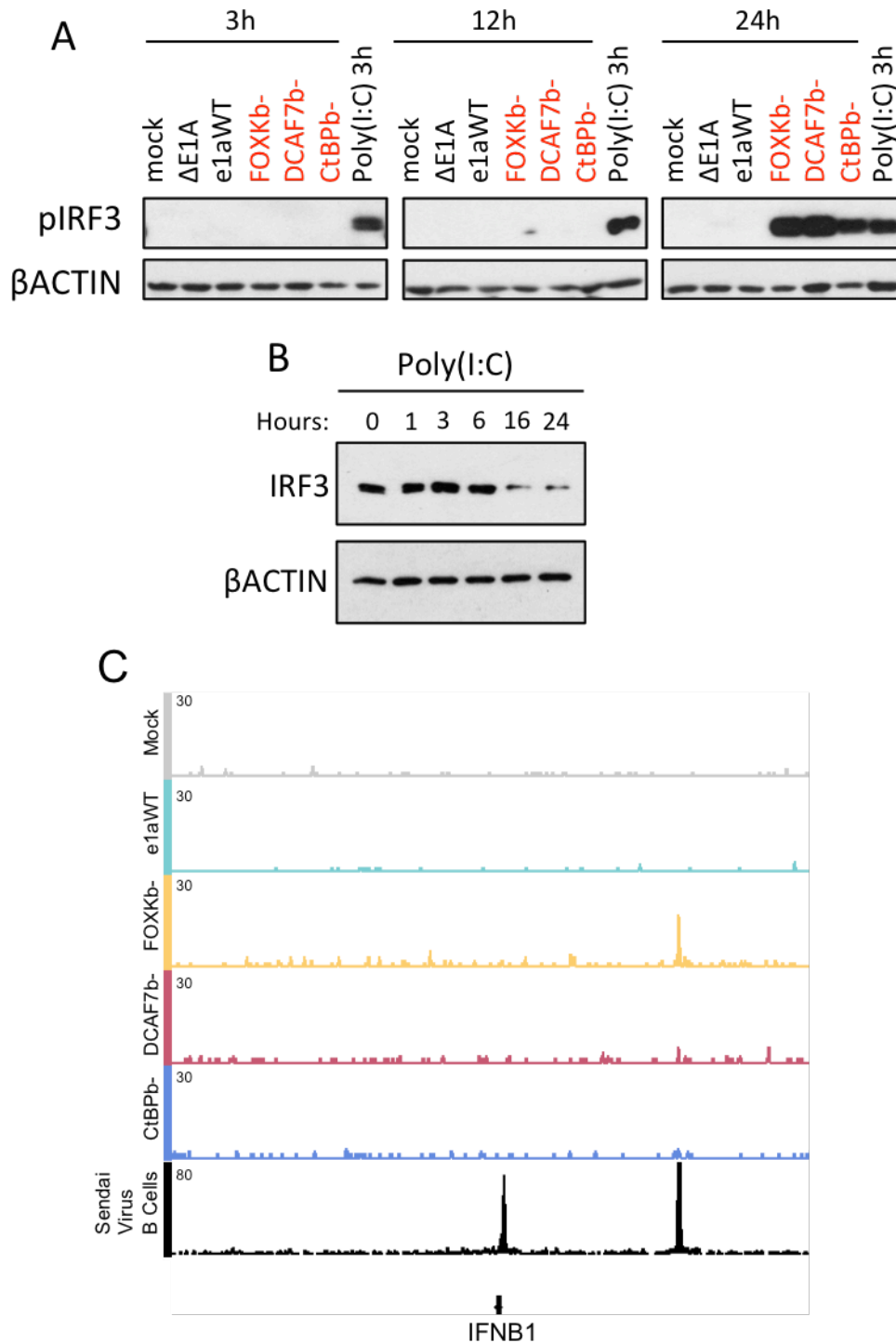


**Figure S4. STAT1 is not phosphorylated at its activating site following infection. Related to Fig 6**  
**(A)** Venn diagram showing overlap of U-ISGF3 induced genes (Cheon et al., 2013) and e1a C-terminal mutant overexpressed genes. **(B)** Western blots for phosphorylated Y701 STAT1 from lysates of infected HBTEC at various times p.i. An extract from HTBEC treated with 10ng/mL IFN $\alpha$  for 2h was used as a positive control for STAT1 phosphorylated at Y701. **(C)** IFNB1 mRNA assayed by qRT-PCR during a time course of infection of HBTEC.



**Figure S5. STAT1 and STAT2 are not necessary for e1a C-terminal activation of ISGs. Related to Fig 6**

(A) STAT1 mutant U3A cells were infected for 24h with the indicated vectors prior to RNA isolation and qRT-PCR to determine relative ISG15 and IFIT2 mRNA levels. (B) Same as with A but in STAT2 mutant U6A cells.



**Figure S6. Phosphorylation of IRF3 protein by infections and Poly(I:C). Related to Fig 7**

(A) HBTEC were infected for the indicated times or transfected with poly(I:C) 20ug/mL for 3h as a positive control for pIRF3. Level of pSer396 IRF3 was assayed by western blot. (B) HBTEC were transfected with Poly(I:C) 20ug/mL for the indicated times and total IRF3 protein was assayed by western blot. (C) Genome Browser image demonstrating the absence of IRF3 binding at its promoter following Ad5 vector infection/e1a expression, while Sendai virus infected B lymphocytes induces binding. GEO: GSE44939 (Freaney et al., 2013).

**Table S1** Number of host cell genes expressed greater than two times higher and less than two times lower than in HBTECs infected with the e1aWT vector. Related to Fig 2

<b>e1a mutant</b>	<b>Genes 2X &gt; e1aWT</b>	<b>Genes 2X &lt; e1aWT</b>
P300b-	728	241
RBb-	454	790
FOXKb-	105	68
DCAF7b-	146	105
CtBPb-	119	138