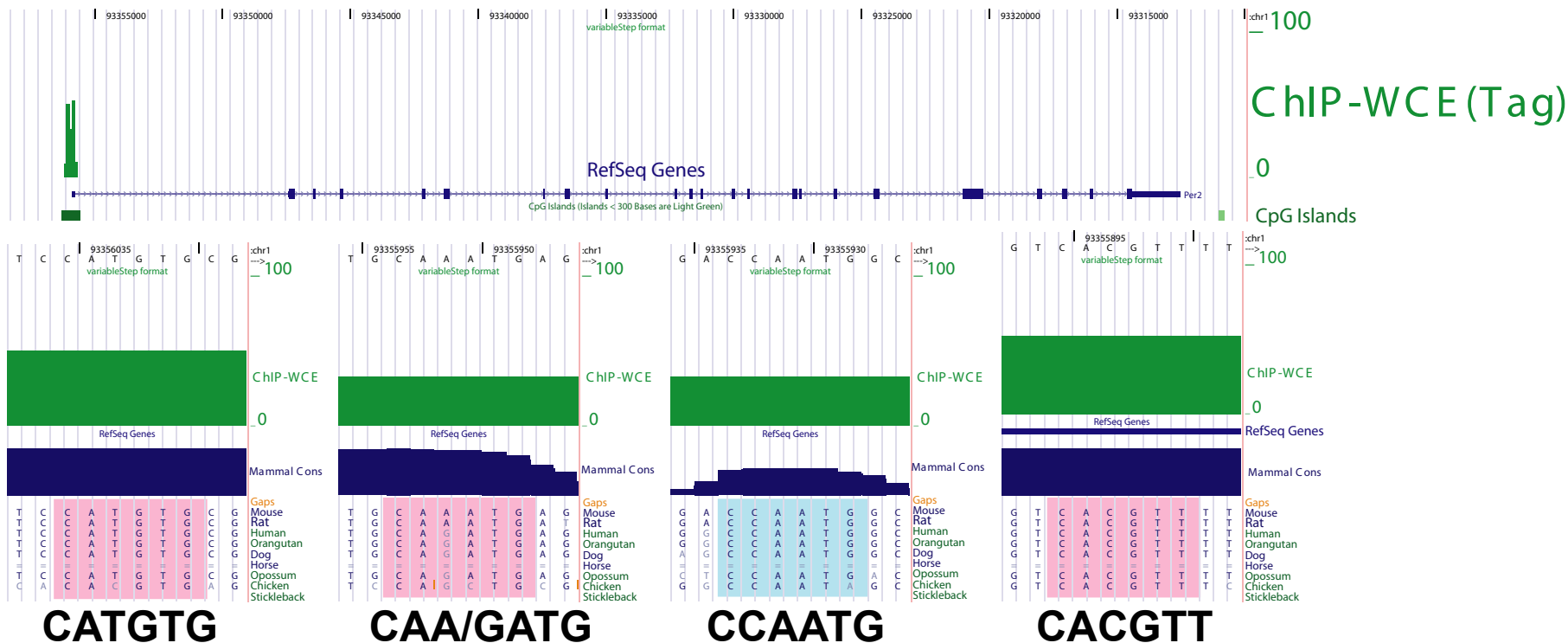
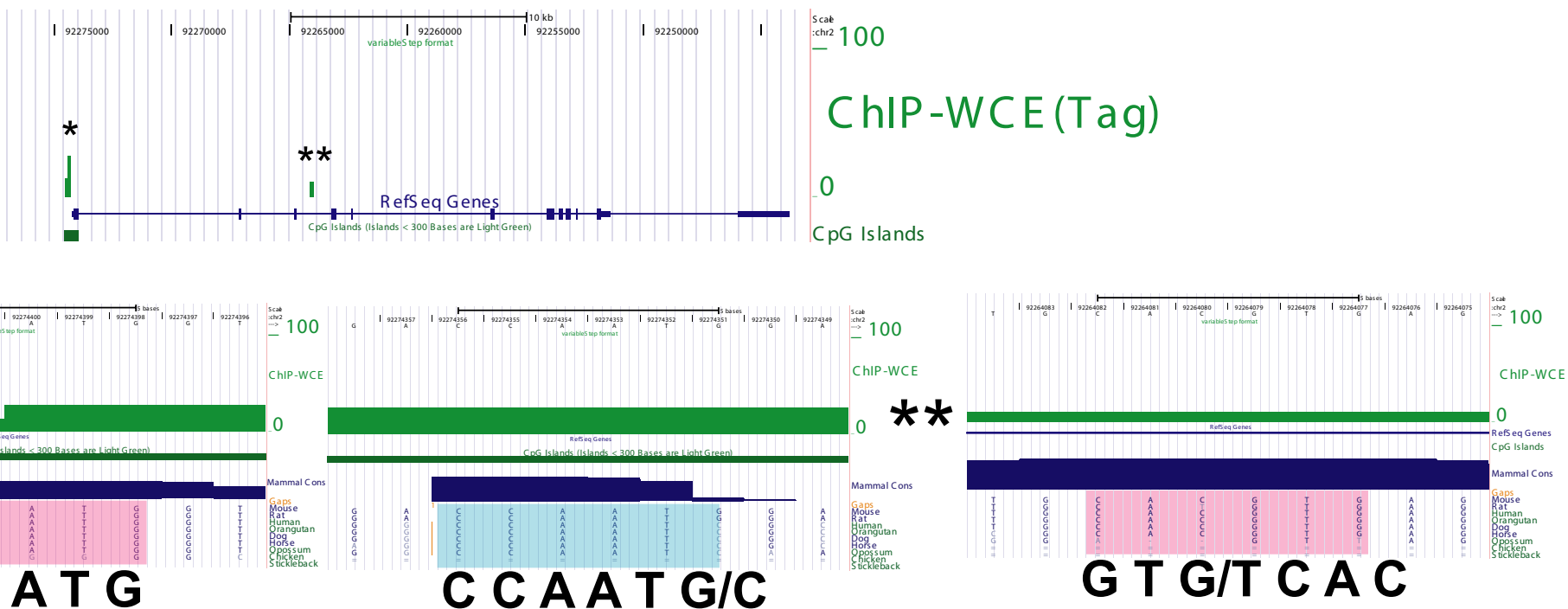


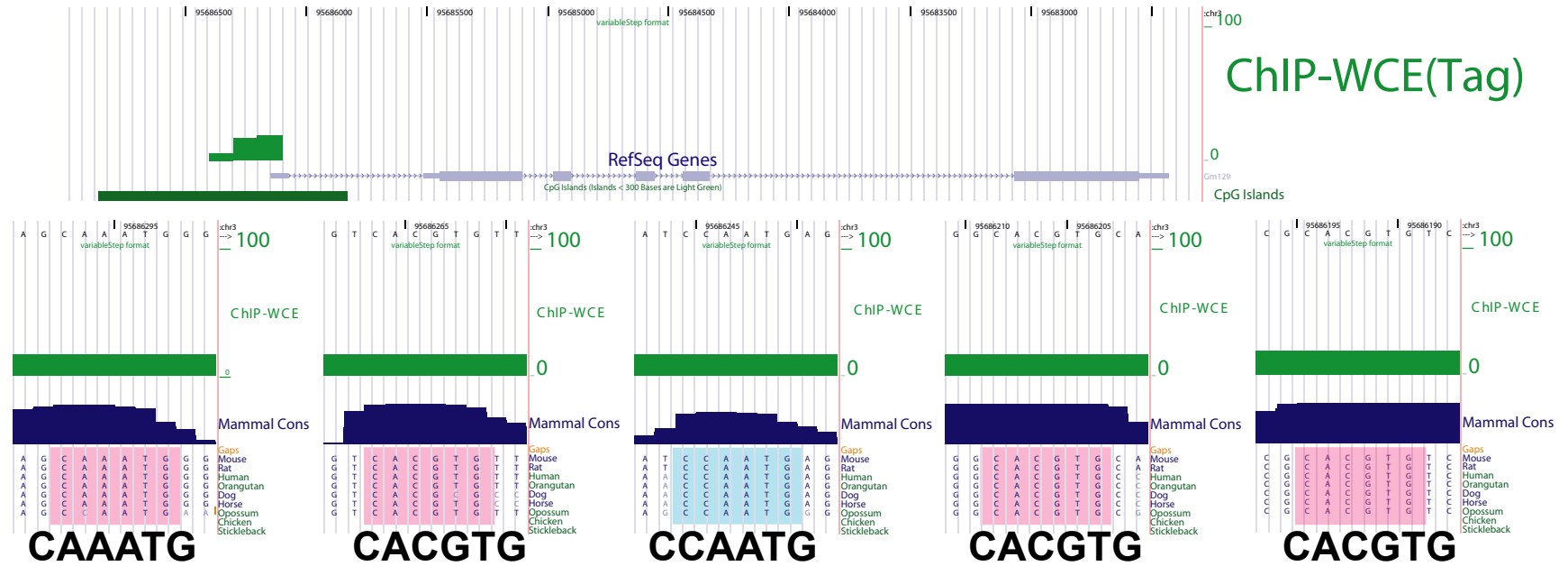
# Per2



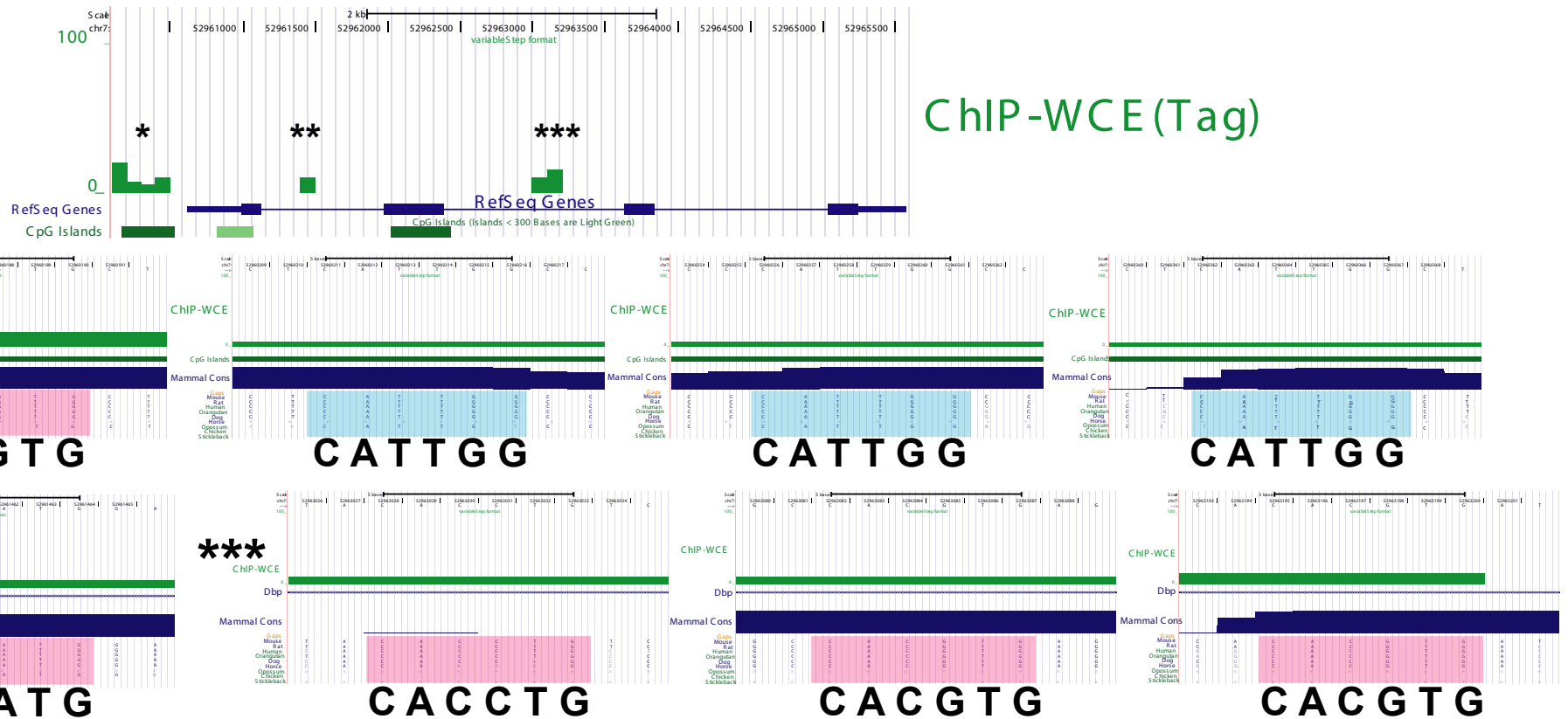
# Cry2



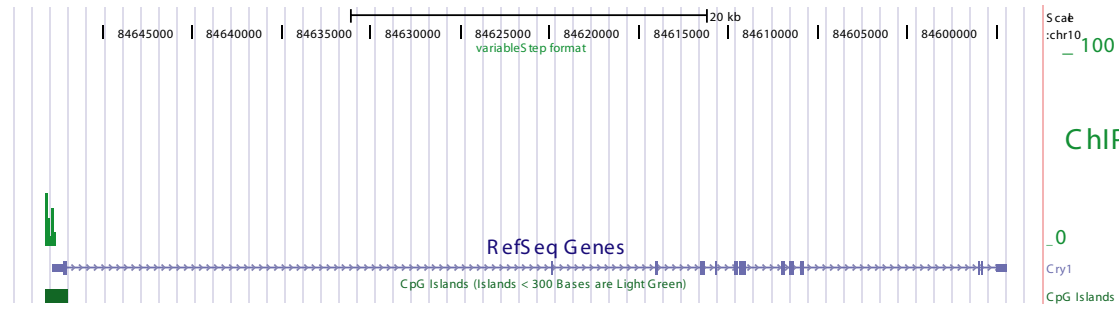
# Gm129



# Dbp



Cry1

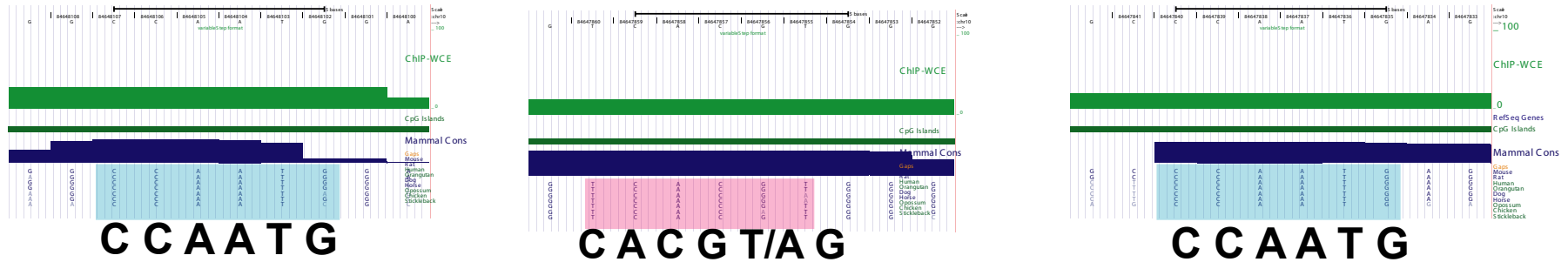


ChIP-WCE

0

Cry1

CpG Islands



Per1

ChIP-WCE

RefSeq Genes  
CpG Islands

RefSeq Genes

CpG Islands (Islands < 300 Bases are Light Green)

\*

ChIP-WCE

RefSeq Genes

CpG Islands

Mammal Cons

CACGTTG

CACGTTG

CCAATG

CCAATG

\*\*

ChIP-WCE

RefSeq Genes

CpG Islands

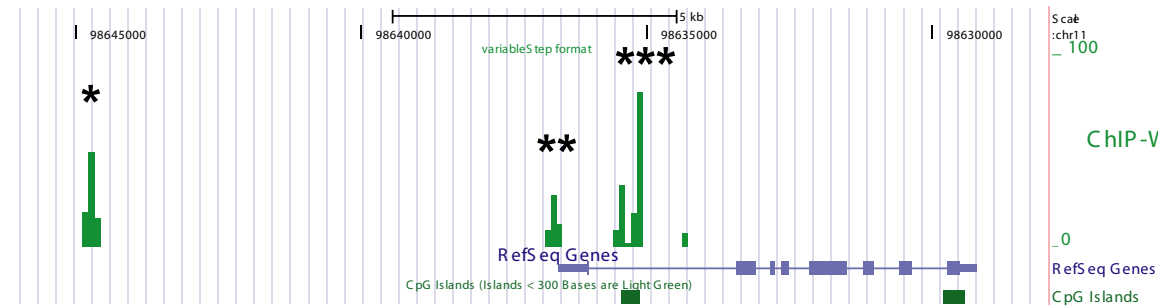
Mammal Cons

CATTGG

CACGTTG

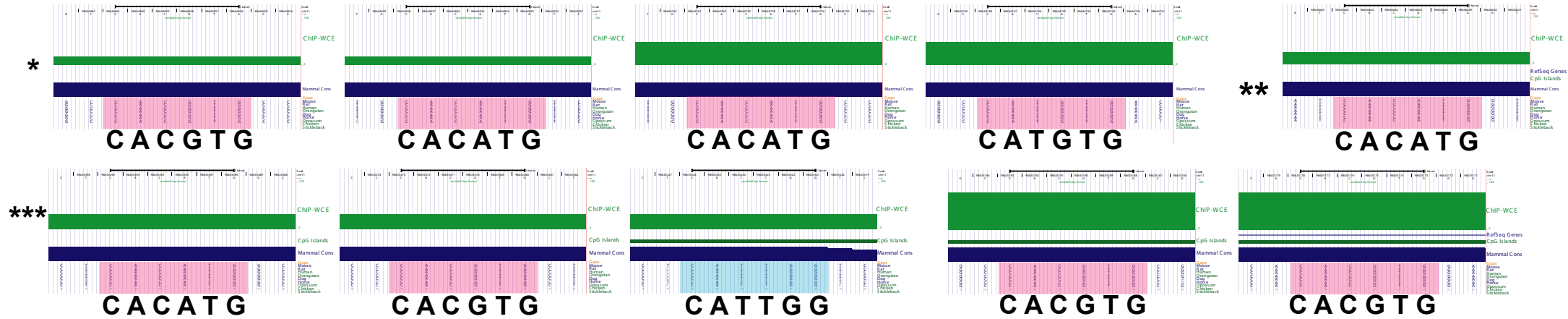
CCAATG

# Rev-erb $\alpha$

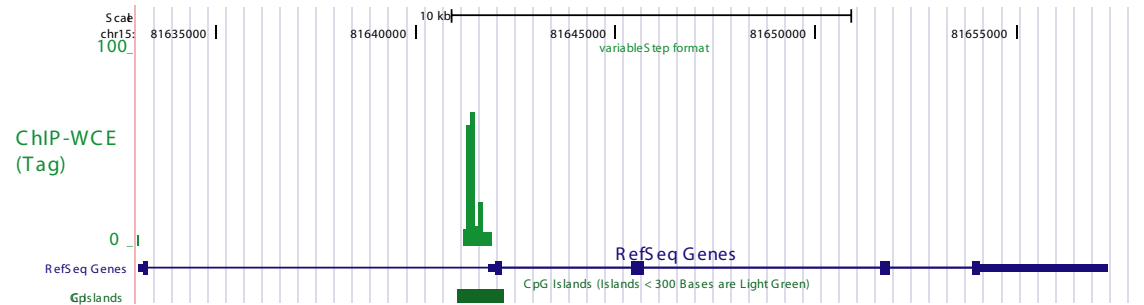


ChIP-WCE (Tag)

0  
RefSeq Genes  
CpG Islands

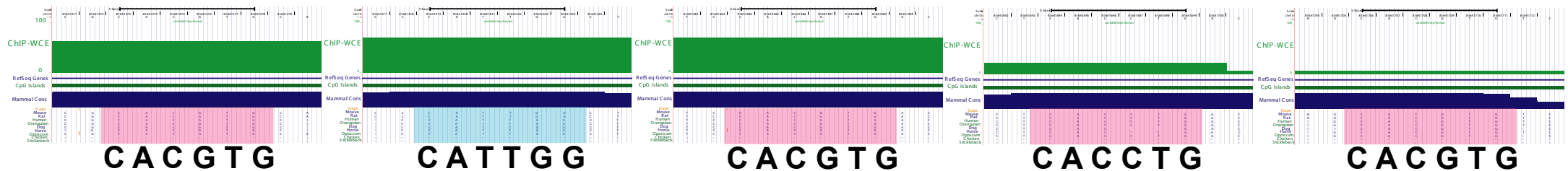


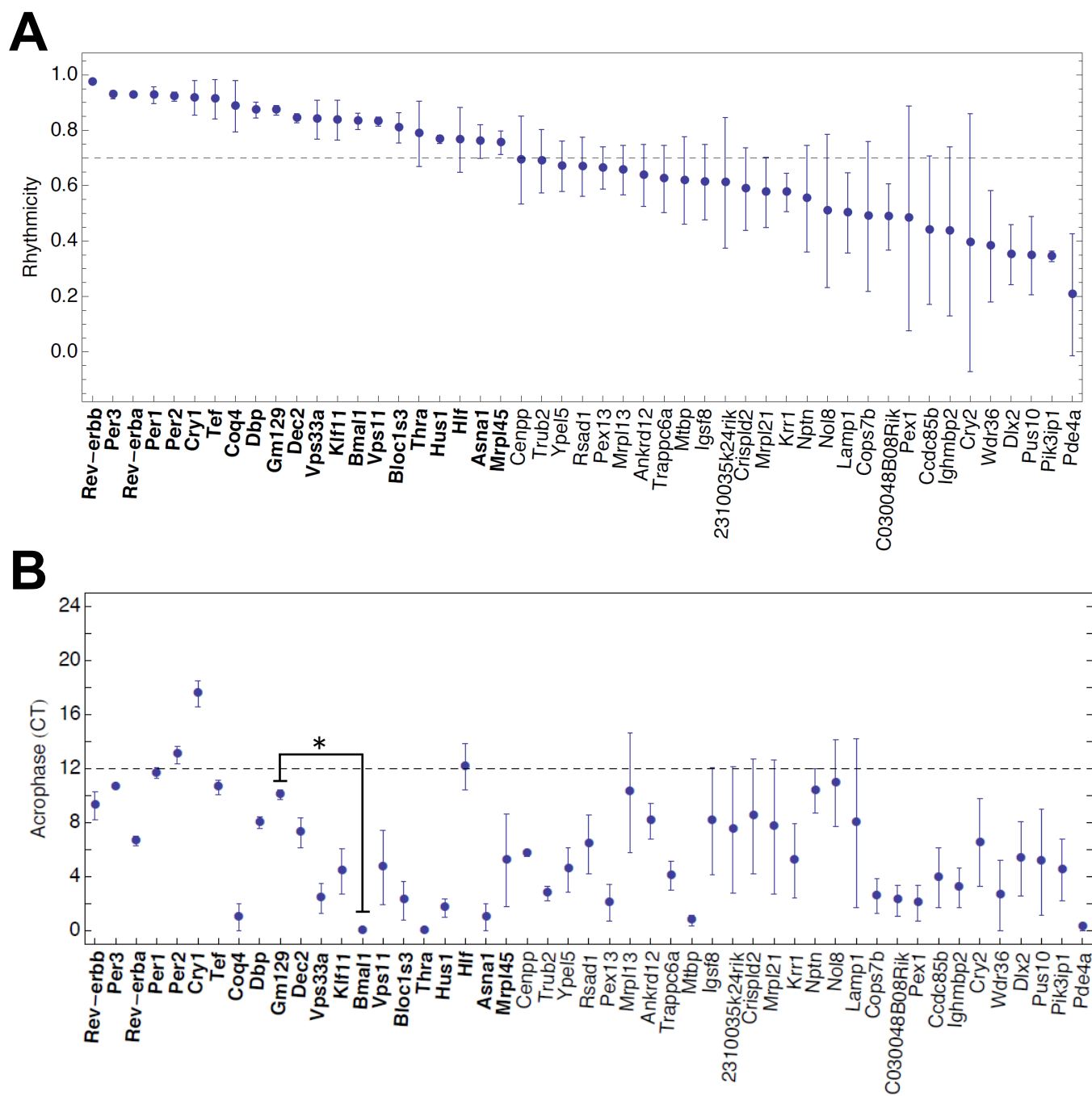
# Tef



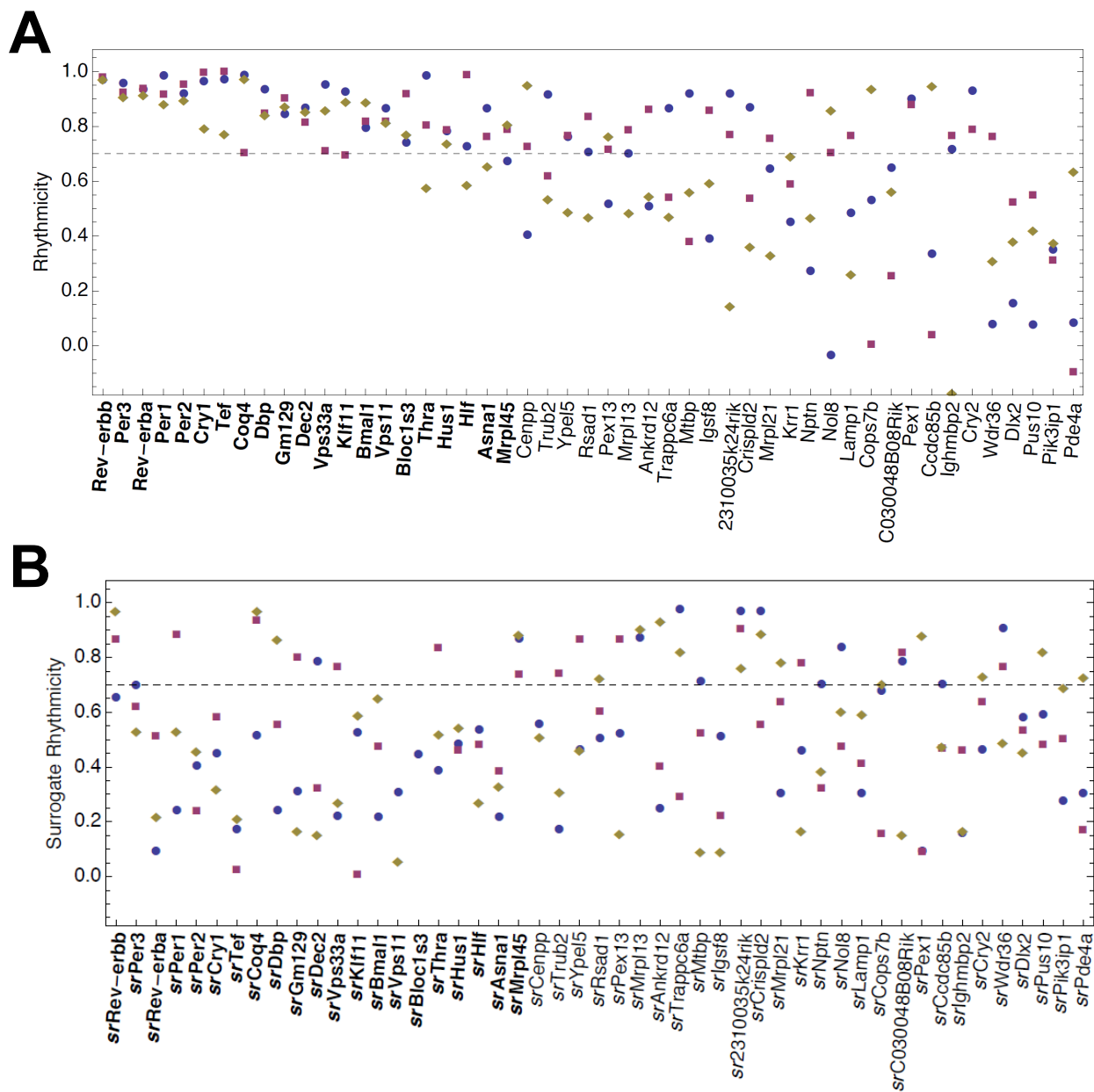
ChIP-WCE (Tag)

0  
RefSeq Genes  
CpG Islands

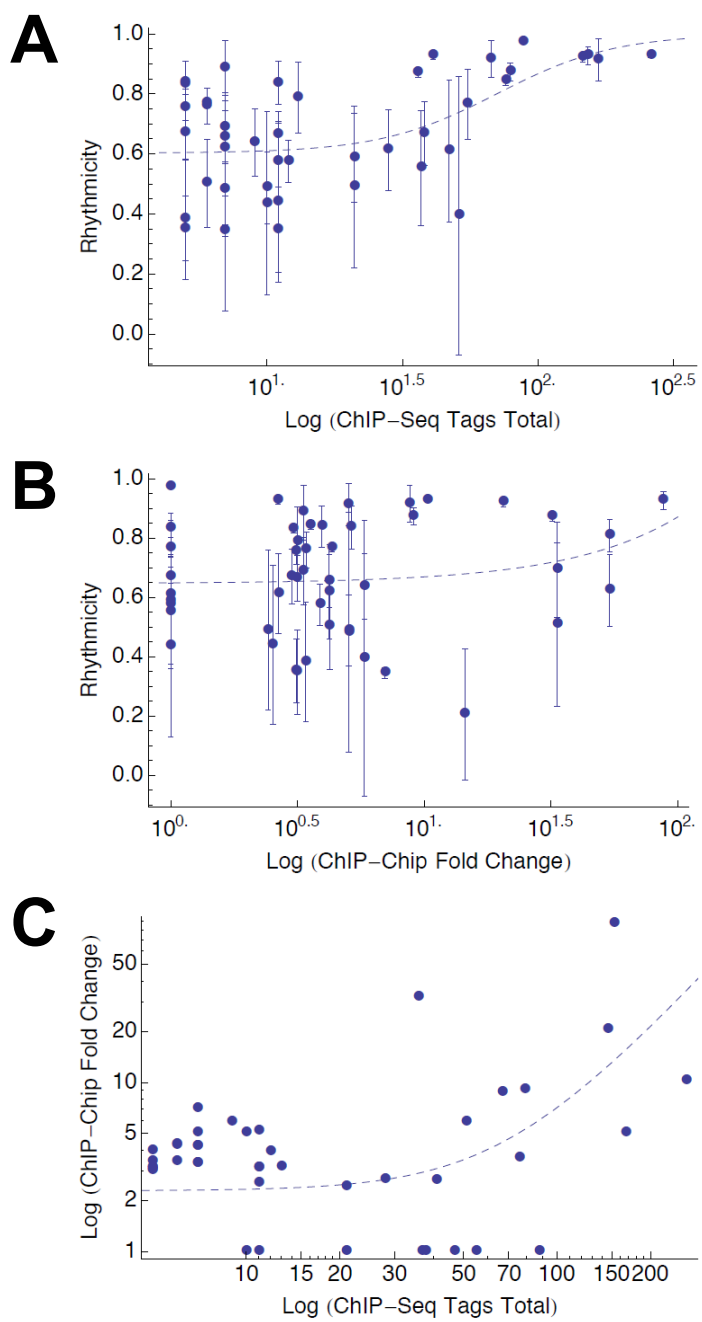




**Fig.S5**



**Fig.S6**



**Fig.S7**

Fig.S8

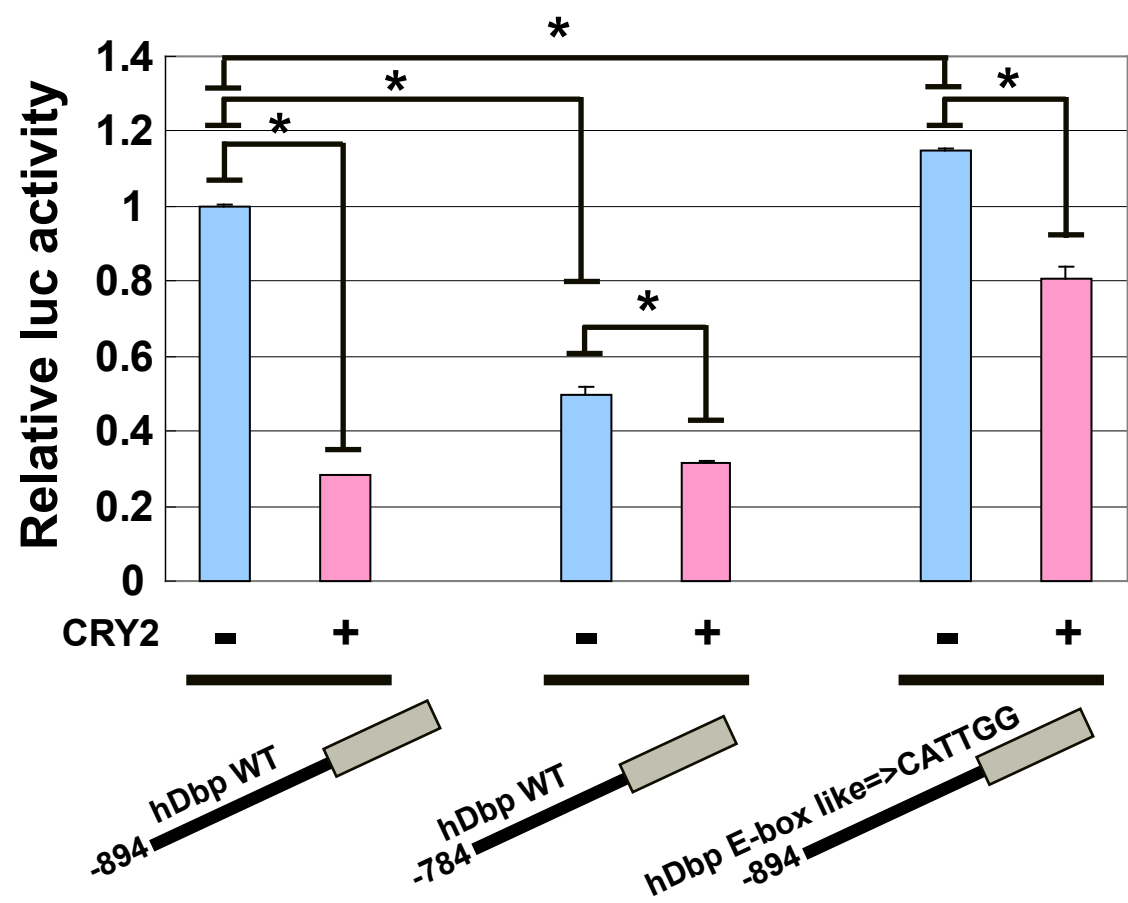
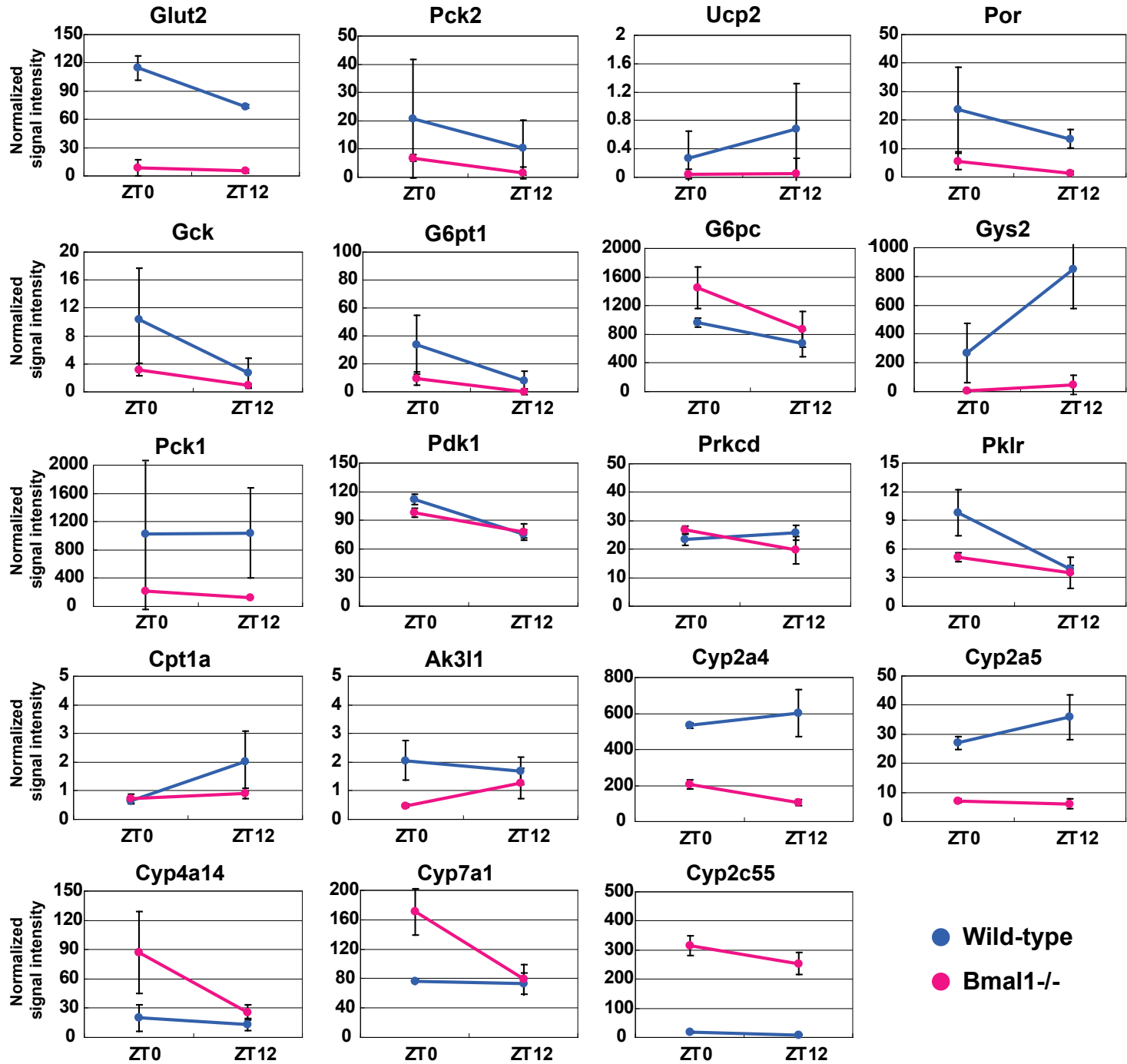


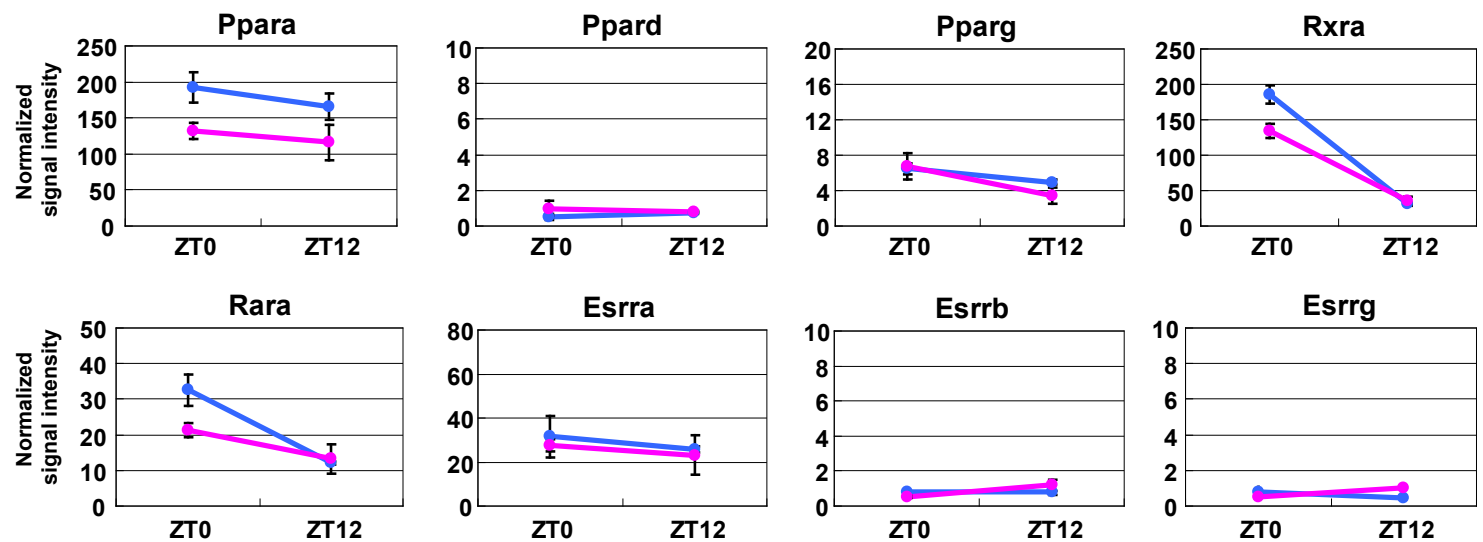


Fig.S9

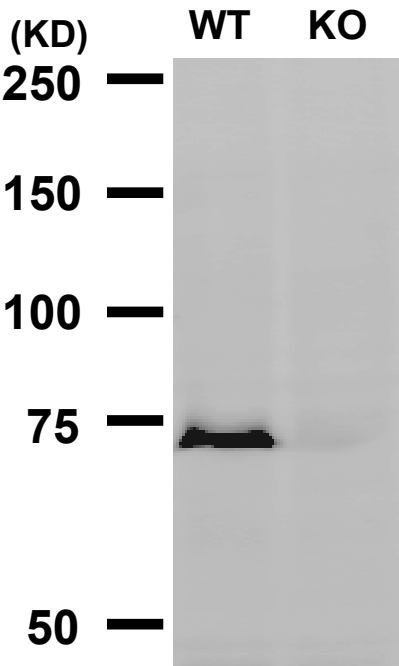
A



B



**Fig.S10**



## Figure legends

### Figure S1-4. Genomic maps of BMAL1 binding sites from ChIP-seq.

BMAL1 binding promoters of *Per2*, *Cry2*, *Gm129*, *Dbp*, *Cry1*, *Per1*, *Rev-erba* and *Tef* from ChIP-seq in UCSC genome browser view. The y-axis shows difference of Tag number between anti-BMAL1 and WCE (control). The ChIP-seq tag profile shows green color above the RefSeq genes. Each enlarged view is shown on the downside. These BMAL1 binding sites include E-box and E-box like element (Red shadow) and CCAATG element (Blue shadow) as in enlarged view. Mammal Cons (deep blue color) show conservation histogram in mammals in UCSC genome browser view. CpG islands are regions where CpGs are present at significantly higher levels than is typical for the genome as a whole. These data were derived from NIH3T3 mouse fibroblasts.

### Figure S5. Correlation between data and cosine fit.

(A) Rhythmicity indicates correlation between data and cosine fit. The dashed line indicates the chance level. (B) *Gm129* and *Bmal1* are almost antiphasic with acrophases separated by 10 hours. (\* $p < 0.001$ )

### Figure S6. Determination of rhythmicity level above chance.

(A) Rhythmicity of each sample. (B) Rhythmicity of each sample is compared with that evaluated from random surrogate;  $p < 0.2$  (one-sided  $t$ -test) correlates with rhythmicity  $> 0.7$ . (C) Correlation between rhythmicity and  $p$ -value.

**Figure S7. Relationship between ChIP-signals and rhythmicity**

(A) Correlation between rhythmicity and total ChIP-seq tags. (B) Correlation between rhythmicity and ChIP-chip fold-change. (C) Correlation between ChIP-seq tags and ChIP-chip fold-change.

**Figure S8. Differences in expression pattern of metabolic genes in the liver of *Bmal1*<sup>-/-</sup> mice.**

Temporal expression profiles of selected hepatic metabolic genes. Abscissa presents time at ZT (Zeitgeber time) 0 and 12, ordinate presents signal intensity from microarray. Shown are the mean  $\pm$  S.E. of 3 mice of each genotype. (A) Genes relevant to glucose and drug metabolism. (B) Nuclear receptors.