INTRAGENOMIC REDISTRIBUTION OF HOST TRANSCRIPTION FACTOR BINDING WITH TOXOPLASMA GONDII INFECTION.

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FIGURES

Figure S1. Overview of experiments

We illustrate below the experiments performed in this project. We infected human foreskin fibroblasts (HFF) with RH strain parasites in the tachyzoite stage of the *T. gondii* life cycle, harvesting cells with ~80% infection at 24 hours, harvesting concurrently grown uninfected cells for comparison. DNA and RNA were extracted for genome-wide assays to measure cytosine modifications and gene expression, and for mass spectroscopy to measure cytosine modifications. Chromatin was isolated to define loci of transposase-accessible, open chromatin, and cells were also profiled metabolically.

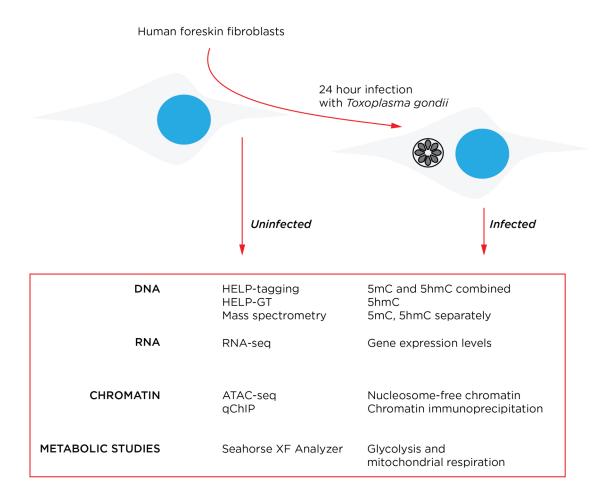


Figure S2. The distributions of ATAC-seq peaks relative to annotated gene promoters in infected cells.

We show the distributions of ATAC-seq peaks, indicating loci of transposase-accessible chromatin, relative to annotated RefSeq transcription start sites (TSS), in three situations. The top panel shows genes where no change was observed in transposase-accessible chromatin, the middle panel the genes where ATAC-seq peaks were lost overall, and the bottom panel the genes gaining ATAC-seq peaks. The vertical red lines show the 10% and 90% cutoffs of the distribution, showing that we can use a window of ± 150 kb from the TSS to capture 80% of loci of transposase-accessible chromatin.

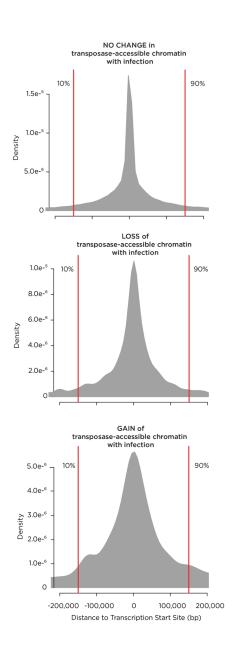


Figure S3. Lack of change of expression for the TFs identified from motif studies.

We show the results of RNA-seq for the TFs indicated by motif analysis to be enriched at loci changing transposase-accessibility, including the AP-1 components JUN and FOS. None of these genes had significant changes in expression.

