



Supplemental Fig. S10. Genome-Wide Analysis of Marks of Strong and Weak Enhancers Around TOP2A CCR Centers Compared to CCR Flanking Regions in K562 Cells.

(A) Increased signals of strong enhancers tagged by dual H3K4me1/H3K27ac marks at CCR centers compared to 1 kb flanking sequences on either side in 100 nt sliding windows. The plot shows higher H3K4me1/H3K27ac signals directly at CCR centers. p-value < 0.001 for DMSO, VP16, pBQ; p-value < 0.01 for mitoxantrone, genistein; Kruskal-Wallis test.

(B) Increased signals of weak enhancers tagged by H3K4me1 but not H3K27ac at CCR centers compared to 1 kb flanking sequences on either side in 100 nt sliding windows. The plot shows higher H3K4me1 signals directly at CCR centers. p-value < 0.001 for DMSO, VP16, pBQ; p-value < 0.01 for mitoxantrone, genistein; Kruskal-Wallis test.

(A, B) K562 cell line data from The ENCODE Project Consortium 2012 for H3K4me1 and H3K27ac (www.encodeproject.org) (The ENCODE Project Consortium 2012) (See Supplemental Table S4 for GEO Accession numbers and Production Laboratories) converted from GRCh37/hg19 to GRCh38/hg38 using liftOver (<http://genome.sph.umich.edu/wiki/LiftOver>) (Hinrichs et al. 2006) were plotted to determine relationships to TOP2A CCRs. Amplified samples; TOP2 poisons indicated; same treatments merged where applicable (Supplemental Table S1). See also Fig. 7B, C.