Supplemental Figure 1. ChIP-seq binding profiles of individual biological replicates. ChIPseq enrichment scores for each replicate are shown across a representative genomic region. The corresponding GEO entries are given in Supplemental File 1. The sample nomenclature starts with the target (e.g. H3K27ac), the strain the ChIP was performed in (e.g. N2), the developmental stage (e.g. Emb), and a unique data ID for each biological replicate (e.g. LW201)

Supplemental Figure 2. ChIP-seq ATAC-seq and DNase-seq profiles in embryo and L3 worms. ChIP-seq binding profiles for averaged data sets across a representative region of the X. Transcriptionally inactive regions are outlined in blue and active regions in green.

Supplemental Figure 3. Filtering DPY-27 ChIP-seq binding peaks. DPY-27 and Pol II ChIP-seq profiles are shown along with all peaks and top 50% of peaks based on average enrichment.

Supplemental Figure 4. Correlation of ChIP enrichment between data sets at TSS. (A)

Spearman rank correlations of average ChIP-seq enrichment within 1 kb regions centering at the GRO-seq defined TSS sites on the X chromosome. ChIP data from *dpy-21* (CB428) and wild type (N2) embryos are plotted. Histone modifications associated with active transcription show positive correlation with RNA Pol II binding. Moreover, high correlation between wild type and mutant ChIP scores support the idea that the DCC does not drastically change the distribution of the marks, but rather tunes their level. **(B)** Spearman rank correlations between standardized log2 ratios of ChIP-seq enrichment (*dpy-21*/wild type) at the 1 kb GRO-seq TSS regions across the X and autosomes. There was no strong correlation between change in RNA Pol II binding and change in histone modifications in the mutant compared to wild type. An X-specific negative correlation between DPY-27 binding and H3K27ac, supports DCC binding being linked to a reduction in H3K27ac on the X.

Supplemental Figure 5. ChIP-seq enrichment changes in the DCC mutant and knockdown.

(A) Data was analyzed as in Figure 3J and 4B, but all chromosomes are shown. DCC depletion or mutation did not significantly or specifically affect the level of H3K27me1, H3K4me1, and H3K27me2 on the X chromosomes. However, ChIP-seq data using antibodies against these modifications showed lower signal, precluding strong conclusion. (B) As in A, but change in histone modifications were calculated across 1 kb windows centering at Wormbase defined transcription start sites. Due to trans splicing of most genes in *C. elegans*, these TSS coordinates are less accurate, but majority fall within 500 bp of real transcription start sites (KRUESI *et al.* 2013). DCC depletion or mutation does not specifically affect H3K27me2 and H3K9me3 but leads to increased H3K27ac across these Wormbase defined transcription start sites. Boxplots are plotted as in Figure 3. * p-value ≤ 0.001 (two-tailed Student's t-Test).

Supplemental Figure 6. Analysis of histone modification changes in X;V fusion

chromosomes. (A) Standardized log2 ratios of H3K27ac enrichment within 200 bp centering at the wild type H3K27ac ChIP-seq peak summits in X;V versus wild type larvae. Individual data points within the middle, left and right most 1 Mb of each chromosome are plotted. The mean ratio for each region is shown as a line. p-values were generated from a two-tailed Student's t-Test are shown above the data. **(B)** Average DPY-27 ChIP-seq scores for 1 kb GRO-seq defined TSS regions are shown. For each 200 kb window moving along the chromosome with 20 kb steps, ChIP-seq and mRNA-seq ratios in X;V/wt were compared to the rest of the windows and a p-value

statistic was generated through t-test. Windows with a = p-value ≤ 0.01 are plotted. As opposed to the chromosome V, where the windows are clustered around the region of spreading (Figure 5E), on chromosome I and II, the windows containing significant changes in gene expression and histone modifications do not show noticeable clustering.

Supplemental Figure 7. Validation of MDT-15 antibody. modENCODE generated MDT-15 Q4097 antibody was validated by western blot analysis. In N2 wild type embryos, the antibody pulled down a protein (left panel) corresponding to a band whose intensity reduced upon *mdt-15* RNAi (compare MDT-15 signal in lane 1 to lane 4, which have similar loading based on tubulin blot). Image J was used to quantify band intensity and percentage reduction was calculated for each lane by taking a ratio of RNAi/vector MDT-15 signal versus tubulin control signal. The discrepancy between predicted MDT-15 protein size and the observed size is unclear.

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	T25C12.0	s spp-21 T25C12.3	hpo-34 hpo-34 hpo-34	21ur-12253 C35	SC5.14 lev-8 H H05G16.2 C35C5.19 linc-33 trop-9 H H	F52D10.7 F52D10.9	Y71H9A.10 F52H2.10 Y71H9A.6 cutl-11 M
ATAC_EE_N2	60	the design of the	a da ana	L		والأقر والانداد والمراجع	
DNasel_EE_N2	200_	h					iiti
DPY27_N2_Emb_SE30_CJ39_CJ132_	SE172 ¹⁰	Mart 1 - 6-44					
DPY30_N2_Emb_BC003_BC086_SE2	1_SE252_LW49	h		1			
SDC3_N2_Emb_SE13_SE213_SE214	_SE215_LW48			1			
RNAPoIII_N2_Emb_8WG16_CJ22_LW	124 5	-11-0				A	
AMA1_N2_Emb_CJ90_CJ98_LW125	5_ 1	and a second			14		
H3K4me3_N2_Emb_SE180_SE255	-25						
H3K27ac_N2_Emb_LW201_LW204_L\	N215			1. I			
H3acetyl_N2_Emb_SE211_SE271	-2 5_						tanan ya kuningi kana kuningi ka
H4K16ac_N2_Emb_SE143_SE146_SE	256						
H4panAc_N2_Emb_SE205_SE206_LA	S63						a na an
H3K4me1_N2_Emb_LW166_LAS53	-25_						
H2AZ_N2_Emb_SE51d_SE93dd	-2 10			1	L		
H3K27me1_N2_Emb_LW157_LW160	-25			·	·····	AA	
H3K27me2_N2_Emb_LW212_LW227	-2 - 5 - 5 -	A		*, ******** ,****			1
H4K20me1_N2_Emb_CJ95_SE265_LV	V116 ³				Al		
H3_N2_Emb_SE212_SE223	-23	ukteren under andere					
lgG_N2_Emb_CJ101_SE142_SE145	3_ 3_						
CBP1_N2_Emb_LW197_SE339_BC05	3 ⁻² -			 1		 .	
MDT15_N2_Emb_LW189_SE340	-25_			1 1		6 . I .	
PQN85_N2_Emb_BC072_BC047_SE1	66					L.	
PHA4_OP37_Emb_ControlRNAi_LAS2	2_LAS23_LW106						
H3K9me3_N2_Emb_ControlRNAi_LAS	89_LAS90		· · · · · · · · ·		· · · · · · · · · · · · · · · · · · ·		

ATAC_L3_N2	40 _	all a de bre		
DPY27_N2_L3_SE262_SEA153	10_	سالاستربه فالطريع أليانهم		
PollI_N2_L3_modEn	200 :: -2		La contraction de la contracti	
H3K27ac_L3_FE_modEN	5_ 		A the second	
H3K27me3_L3_FE_modEN	ة: 			
H3K9me3_L3_FE_modEN	5_			
H3K4me3_N2_L3_modEn	5_			
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ce10 chi	rX: 4,330	0,000 4,34	40,000	4,350,000	4,360,000	4,370,000	4,380,000	4,390,000 BefSeg Gene	4,400,000	4,410,000	4,420,000	4,430,000	4,440,000	4,450,000	4,460,0	000
dop- C24A8.1:	5 → → → + + + + + + + + + + + + + + + +	cst-2 F14H12.161 F14H12.151 F14H12.151 F14H12.141 F14H12.141 F14H12.141 cs 6 6 F14H12.131 F14H12.131 F14H12.131 F14H12.132 F14H12.141	F14H12.91 F14H12.3	F14H12.2	F14H12.7 F14H12.17 H12.6 F14H12.8 R1	R160.4 R160.5 H H R160.5 80.3 M 1160.10 R160.6 ★↔	R160.2 ist-2 	R160.8 dpy-23 dpy-23 R160.11	D1079.1 D1079.2	H R160.111 F1 H R160.111 F1 F15A8.41 F15A8.41 F15A8.171 M01E10.51 F15A8.151 M01E10.51 dop-1 do	eeteeteeteeteeteeteeteeteeteeteeteeteet	F15A8.181 F F15A8.6 F F15A8.6 F F15A8.6 F F15A8.161 F F15A8.161 F	1548.111 W0343. ↓ ← ← F1548.1 ↓ 1548.121 B0244.1 1548.121 B0244.1 F10C1.1 F10C1.1 F25F6 R04D3	41 F02E8. 131 131 161 121 121 131 131 131 131	aps-2 H atg-16.1	#
		F14H12.12 F14H12.11 F14H12.1	1 51				dpy-23 pro	moter re	ex-1	le	ower level	binding				
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Pol II	- 															





and Pol II binding at 1kb TSS regions on autosomes









MDT-15 (Q4097)



MDT-15 IP w/ 1 ug SDI-Q4097 Blotted w/ same 1:2000 dilution Expected band size: 84.5 kDa (isoform A) 84.2 kDa (isoform B)



% reduction of signal upon RNAi:

82 71