Supplementary Table III – Overall numbers of genes upregulated or downregulated more than 2 fold in *teb1-1* compared to wt.

	25 °C	36 °C	Both temperatures
Genes upregulated more than 2 fold	244	224	152
Genes downregulated more than 2 fold	84	41	29

Supplementary Table IV – Iron metabolism genes downregulated in teb1-1 strain at 25°C and 36°C

	Gene expression	Gene expression
Iron metabolism gene	compared to wt	compared to wt
	(microarray cells 25°C)	(microarray cells 36°C)
str1	0.293	0.177
str3	0.214	0.1395
fip1	0.4485	0.4705
frp1	0.212	0.2215

Supplementary Table V – Histone average expression levels in teb1-1 strain at 25°C and 36°C

	Gene expression	Gene expression
	compared to wt	compared to wt
Histone genes	(microarray – cells at	(microarray – cells at
	25°C)	36°C)
hta1	0.77	1.022
htb1	0.6025	0.697
hta2	0.286	0.3395
h3.1: <i>hht1</i>	0.3915	0.4505
h3.2: <i>hht2</i>	0.4215	0.5745
h3.3: <i>hht3</i>	0.3135	0.37
h4.1: <i>hhf1</i>	0.321	0.3465
h4.2: <i>hhf2</i>	0.409	0.5155
h4.3: <i>hhf3</i>	0.4115	0.429



Β









1140000

SPCC482 SPCTRNALYS SPCTRNALYS





Figure S2



Figure S3



Figure S4

-N

+N

wt

Urea

α H3

Gu-HCI

-N

teb1-1

Supplementary Figure Legends

Figure S1. Teb1 binds specific genomic loci. Representation of ChIPchip data demonstrating Teb1 binding to specific regions across the genome, and the absence of binding at the non-repetitive regions of the centromere. Repetitive regions were not included in the analysis as these sequences are absent from the microarray data. (A) Teb1 binding at Chr 1 and its centromere (B) Teb1 binding on Chr 2, which contains also the mating type locus. (C) Teb1 binding on Chr 3.

Figure S2. Teb1 does not bind promoters of the histone variants (A) *pht1* (H2A variant); (B) *cnp1* and (C) SPBC800.13 (H4 variant).

Figure S3. Teb1 binds the same regions near the ends of Chr I that are bound by CENP-A upon neocentromere formation. (A) Representation of ChIP-chip data highlighting Teb1 binding near the left and right termini of Chr 1. (B) Sites of Cnp1 or Swi6 binding after centromere disruption and neo-centromere formation on Chr 1 (from *Ishii et al*, 2008). Boxes highlighting regions of the left and right arms depict the same positions shown in the boxes in (A), allowing direct comparison.

Figure S4. Histone H3 is degraded in a Teb1 and Isp6-dependent manner during extraction of G1-arrested cells, but not extracted with Gu-HCI. (A) Top: FACS analysis of wt and *teb1-1* cells arrested in G1 by nitrogen starvation for 16h at 25°C, shifted to 36°C for 1.5h, re-fed to allow resumption of growth and sampled every 30 min for 5 hours. Bottom: Western blot of TCA extracts from the following: (a) asynchronously growing cells; (0) cells starved for nitrogen for 16h; (OB) the same cells 1.5h at 36 °C, before re-feeding; (1 through 5) following re-feeding for 1 to 5 hours as indicated. Both wt and *teb1-1* cells recover efficiently from G1 arrest. Lane (e) was used for the protein marker. Both Cdc2 proteolysis and histone

H3 clipping occur in extracts of wt G1 arrested cells (left panel). However, neither histone H3 clipping nor Cdc2 proteolysis occur in extracts of *teb1-1* cells (right panel). (B) Isp6 is required for histone clipping in extracts of G1-arrested cells. Asynchronous or nitrogen starved (16 hours) cultures of cells of the indicated genotypes were analysed by FACS. (C) Western blot analysis reveals that impairment of Isp6 function abolishes histone clipping in these TCA extracts. (D) Histone H3 clipping is detected in extracts of nitrogen starved cells prepared in the presence of 8M Urea but not in the presence of Gu-HCl.