Identification of a BET family Bromodomain / Casein Kinase II / TAF-containing complex as a regulator of mitotic condensin function

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3 x Supplementary Tables (**Tables S1 - S3**)
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Supplementary References

SUPPLEMENTARY TABLES

Table S1 Complex identification by sequential affinity purification / LC-MS

(A) *S. pombe* (*Sp*) Mis16 is found in two distinct complexes

(B) The NCT complex: proteins associated with *Sp* Nrc1 (*SPAC631.02*)

Table S2 *ChIP-seq* peak classifications

Table S3 Materials used in this study

(A) Antibodies

(B) Fission yeast strains

(C) Oligonucleotides

Table S1: Complex identification by sequential affinity purification / LC-MS

(A) Mis16 is found in two distinct complexes (see **Figure 2E**)

Protein	Gene name	kD [a]	Notes	Mis18 (KLP738) [b]	Mis16 (KLP22) [b]	Hat1 (KLP19) [b]
Mis16	**SPCC1672.10**	48	5 x WD repeats	7 (14) [d]	20 (45)	16 (55)
Hat1	**SPAC139.06**	44	Acetyltransferase (Kat1, Hag603)	-	5 (10)	9 (52)
Mis18	**SPCC970.12**	22.5	Mini-chromosome InStability	8 (18)	6 (9)	-
-	**SPBC27B12.02**	13	Orphan (essential; mitochondrial [c])	7 (16)	3 (4)	-
-	**SPBC776.16**	28.7	Orphan (non-essential; SPB [c])	10 (17)	3 (4)	-

(B) The NCT complex - proteins associated with *Sp* Nrc1 (*SPAC631.02*) (see **Figure 4A**)

Protein	Gene Name	kD [a]	Peptides	Previous complexes	NOTES
Nrc1 (KLP219) [b]	**SPAC631.02**	84	38 (127) [d]	-	Tandem bromodomain. Non-essential
Cka1	**SPAC23C11.11**	40	15 (28)	CKII	Catalytic subunit (α). Essential
Ckb2	**SPBC2G5.02c**	29	4 (5)	CKII	Regulatory subunit (β, predicted). Non-essential
Ckb1	**SPAC1851.03**	27	3 (4)	CKII	Regulatory subunit (β). Non-essential
Taf1, Taf111	**SPAC2G11.14**	111	13 (15)	TFIID	ZnF (CCHC type). Essential. Antibody available.
Taf7, Ptr6	**SPAC13F5.02c**	45	4 (4)	TFIID	Essential. Antibody available.
Taf4	**SPAC23G3.09**	41	3 (4)	TFIID	Histone fold. Essential
Taf5, Taf51, Taf73	**SPBC15D4.14**	72	3 (4)	TFIID	6 x WD repeats; LisH dimerization motif. Essential
Taf6, Taf50	**SPCC16C4.18c**	50	2 (2)	TFIID, SAGA	Histone-fold (H4-like). Essential

KEY: Genes (Mis16, Hat1, Mis18 or Nrc1 (SPAC631.02)) were C-terminally tagged with TAP (CBP-2xProtA) and associated factors identified by sequential affinity purification / LC-MS (Kim et al., 2009; Silva et al., 2012) from the relevant fission yeast strains (see **Table S3**). Peptide identifications were accepted if they could be established at > 95.0% probability by the *Peptide Prophet* algorithm. Protein identifications were accepted if they could be established at > 95.0% probability and contained at least two identified peptides. Specifically enriched proteins and common contaminants were distinguished by comparison with our > 50 *Sp* TAP-purifications performed to date. Indicated proteins were identified in multiple preparations (representative run shown).

[a] Protein sizes (kD) are predicted by amino-acid composition (*PomBase*) and not determined experimentally

[a] BAIT is shaded (strain genotypes are provided in **Table S3**)

[c] $nmt1_{pr}$ *:: yfg*$^{+}$ *:: YFP* fusions (Matsuyama et al., 2006)

[d] Unique peptides (total peptides)

Table S2: *ChIP-seq* peak classifications (related to **Figures 4 - 6, S3 & S5 - S7**)

	Nrc1 (As) TEL3- [a]	Nrc1 (As) TEL3- CEN- [b]	Cka1 (As) TEL3-	Cka1 (As) TEL3- CEN-	Taf7 (As) TEL3-	Taf7 (As) TEL3- CEN-	Cut3 (Me) TEL3-	Cut3 (Me) TEL3- CEN-
Total Peaks	2,107	2,029	2,159	2,127	3,223	3,184	1,829	1,759
Peak IS - Min	10	10	10	10	10	10	10	10
Peak IS - Max	26,562	6,758	121,770	29,970	33,928	33,938	37,342	5,298
Peak IS - Median	19.3	21.6	18.5	18.3	20.5	20.5	32.5	32.2
Peak IS - Average	193.3	134.9	220.8	152	381.4	376.7	108.7	55.5
IS 10 - 100	1,665	1,632	1,823	1,741	2,766	2,739	1,473 [c]	1,425 [c]
Major peaks [c] -								
IS 100 - 1,000	357	333	336	325	245	236	336 [c]	317 [c]
IS 1,000 - 10,000	81	64	64	59	188	187	16 [c]	16 [c]
IS > 10,000	4	0	3	2	24	22	4 [c]	1 [c]
MAJOR PEAKS	**442**	**397 (19.5%)**	**403**	**386 (18.1%)**	**457**	**445 (13.9%)**	**356**	**334 (19%)**

ChIP-seq data (from Asynchronous or Metaphase samples) was loaded into *Genplay* (Lajugie and Bouhassira, 2011) as individual tracks (50 bp windows; normalized and INPUT subtracted) relative to the annotated *Sp* genome (Sanger Center release *pombe090511*). The online version of this paper links to a *Genplay* project to provide a dynamic visualization of each track. Peaks for comparison and assignment to genomic features were automatically identified / quantified by optimized parameters: Regions of 10 successive Windows; Gap permitted, 1 window; **Threshold Island Score (IS), 10**; Minimum Island Length, 4 windows (see **Experimental Procedures**). After cleanup the range and distribution of island scores was used to identify Major peaks (> 50, Cut3; or > 100, all others) for certain analyses (e.g. **Figure 5A-F**). All sample data have been deposited in NCBI's Gene Expression Omnibus (Edgar et al., 2002) and are accessible through GEO series accession number GSE53955.

[a] Peaks at the Chromosome 3 telomeres (*TEL-3L*, 1 - 24,611; *TEL-3R*, 2,439,219 - 2,453,097: together includes 11 rRNAs and 3 misc RNAs) were excluded from all consideration because of repeat issues.

[b] Peaks at the centromeres (***CEN1***, *SPATRNAPHE.02 - SPNCRNA.95*; ***CEN2***, *SPBTRNAASN.01 - SPBTRNAARG.07*; ***CEN3***, *SPCTRNAALA.12 - SPCTRNAPHE.05*: together includes 54 tRNAs and 31 misc RNAs) were considered separately because of the special epigenetic signature and behavior at these locations.

[c] Major peaks for Cut3 are divided into IS [50 - 500], [500 – 5,000] and > 5,000 (shaded).

Table S3: Materials used in this study

(A) Antibodies

Anti-	Species	Info	Sera #	Source
Histone H3	Rabbit	Polyclonal	ab1791	Abcam
Histone H3-K9ac	Rabbit	Polyclonal	07-352	Millipore
Histone H3-K14ac	Rabbit	Polyclonal	07-353	Millipore
Histone H3-K18ac	Rabbit	Polyclonal	39129	Active Motif
Histone H4-K5ac	Rabbit	Polyclonal	39169	Active Motif
Histone H4-K12ac	Rabbit	Polyclonal	39165	Active Motif
Histone H4 tetra-ac	Rabbit	Polyclonal	39179	Active Motif
Histone H3-K4me3	Rabbit	Polyclonal	Ab8678	Marc Bedford
Histone H3-K36me3	Rabbit	Polyclonal	Ab9050	Abcam
Histone H3-K9me2	Rabbit	Polyclonal	39137	Active Motif
Histone H3-S10phos	Mouse	Monoclonal	06-816	Millipore
HA epitope tag	Mouse	Monoclonal	12CA5	-
FLAG M2	Rabbit	Polyclonal	5407	Alexa Fluor 488 Conjugate; Cell Signalling
FLAG M2	Mouse	Monoclonal	A2220	Affinity Gel; Sigma-Aldrich
Sp Cnp1 (SPBC1105.17)	Sheep	Polyclonal	-	Robin Allshire (Caterino and Hayes, 2007)
Sp Tbp1 (SPAC29E6.08)	Rabbit	Polyclonal	-	Rich Maraia (Huang et al., 2000)
Sp Taf7 (ptr6, SPAC13F5.02c)	Rabbit	Polyclonal	RTK7-8	Tetsuro Kokubo (Mitsuzawa et al., 2001)
T.brucei a-Tubulin	Mouse	Monoclonal	Tat1	Keith Gull (Woods et al., 1989)

(B) Fission yeast strains

Name	Alt. name	Genotype	Used	Source
KLP377		*h⁻ ade6-M210 ura4-D18 leu1-32 smt-0 mat1_m-syh^S rpl42::cyh^R (sP56Q) cut3-477::NatMX6*	Figure 1	This work
KLP378		*h⁻ ade6-M210 ura4-D18 leu1-32 smt-0 mat1_m-syh^S rpl42::cyh^R (sP56Q) cut14-208::NatMX6*	Figure 1	This work
KFP26	KGY425, AP26	*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1*	Figures 1, S1	K.L Gould
KLP684		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 hat1Δ::KanMX6*	Figures 1, S1	This work
KLP686		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 nrc1Δ::KanMX6*	Figures 1, S1	This work
KLP402		*h⁺ ade6-M216 ura4-D18 leu1-32 his3-D1 cut3-477::NatMX6*	Figures 1, S1	This work
KLP712		*h? ade6-M2XX ura4-D18 leu1-32 his3-D1 cut3-477::NatMX6 hat1Δ::KanMX6*	Figures 1, S1	This work
KLP714		*h? ade6-M2XX ura4-D18 leu1-32 his3-D1 cut3-477::NatMX6 nrc1Δ::KanMX6*	Figures 1, S1	This work
KLP416		*h⁺ ade6-M216 ura4-D18 leu1-32 his3-D1 cut14-208::NatMX6*	Figure 1	This work
KLP713		*h? ade6-M2XX ura4-D18 leu1-32 his3-D1 cut14-208::NatMX6 hat1Δ::KanMX6*	Figure 1	This work
KLP715		*h? ade6-M2XX ura4-D18 leu1-32 his3-D1 cut14-208::NatMX6 nrc1Δ::KanMX6*	Figure 1	This work
KFP445		*h⁻ leu1-32 cnd2-1*	Figure 1	This work
KLP767		*h? leu1-32 cnd2-1 hat1Δ::KanMX6*	Figure 1	This work
KLP768		*h? leu1-32 cnd2-1 nrc1Δ::KanMX6*	Figure 1	This work
HSK107		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 nrc1Δ::KanMX6 hat1Δ::HphMX6*	Figure S1	This work
HSK109		*h? ade6-M2XX ura4-D18 leu1-32 his3-D1 cut3-477::NatMX6 nrc1Δ::KanMX6 hat1Δ::HphMX6*	Figure S1	This work
KFP273		*h⁻ ura4-D18 leu1-32 nda3-KM311*	Figure 2	P. Nurse
KLP303		*h⁺ ade6-M216 ura4-D18 leu1-32 his3-D1 Cut3.HA$_3$::KanMX6*	Figure 2	This work
2011Spd-5		*h⁺ ade6-M21X ura4-D18 leu1-32*	Figure 2	Bioneer
2011Spd-3		*h⁺ ade6-M21X ura4-D18 leu1-32 rtt109Δ::KanMX6*	Figure 2	Bioneer
2011Spd-4		*h⁺ ade6-M21X ura4-D18 leu1-32 hat1Δ::KanMX6*	Figure 2	Bioneer
2011Spd-1		*h⁺ ade6-M21X ura4-D18 leu1-32 gcn5Δ::KanMX6*	Figure 2	Bioneer
KLP346		*h⁻ cdc25-22 Cut3.HA$_3$::KanMX6*	Figures 2, S1, S4	This work
KLP694		*h? cdc25-22 Cut3.Flag$_5$::HphMX6 Nrc1.HA$_3$::KanMX6*	Figures 2, S1	This work
KLP698		*h? cdc25-22 Cut3.Flag$_5$::HphMX6 Nrc1.HA$_3$::KanMX6 hat1Δ::NatMX6*	Figures 2, S1	This work
KLP662		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 Mis16.HA$_3$::KanMX6*	Figures 2, S1	This work
KLP795		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 Mis16.HA$_3$::KanMX6 Hat1.Flag$_5$::HphMX6*	Figure 2	This work
KLP671		*h? cdc25-22 Cut3.Flag$_5$::HphMX6*	Figures 2, S1	This work
KLP672		*h⁻ cdc25-22 Cut3.Flag$_5$::HphMX6 Mis16.HA$_3$::KanMX6*	Figures 2, S1	This work
KFP26	KGY425, AP26	*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1*	Figure 1	K.L Gould
KLP402		*h⁺ ade6-M216 ura4-D18 leu1-32 his3-D1 cut3-477::NatMX6*	Figures 2, S1	This work
KLP716		*h? ade6-M2XX ura4-D18 leu1-32 his3-D1 cut3-477::NatMX6 Mis16.HA$_3$::KanMX6*	Figures 2, S1	This work
KLP416		*h⁺ ade6-M216 ura4-D18 leu1-32 his3-D1 cut14-208::NatMX6*	Figure 1	This work
KLP717		*h? ade6-M2XX ura4-D18 leu1-32 his3-D1 cut14-208::NatMX6 Mis16.HA$_3$::KanMX6*	Figure 2	This work
KFP26	KGY425, AP26	*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1*	Figure 3	K.L Gould
KLP689		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 Nrc1.HA$_3$::KanMX6*	Figure 3	This work

Name	Alt. name	Genotype	Used	Source
KLP741		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 nrc1-BD1*.HA$_3$::KanMX6*	Figure 3	This work
KLP742		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 nrc1-BD2*.HA$_3$::KanMX6*	Figure 3	This work
KLP740		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 nrc1-BD1*/BD2*.HA$_3$::KanMX6*	Figure 3	This work
KLP694		*h? cdc25-22 cut3.Flag$_5$::HphMX6 Nrc1.HA$_3$::KanMX6*	Figure 3	This work
KLP759		*h? cdc25-22 cut3.Flag$_5$::HphMX6 nrc1-BD1*/BD2*.HA$_3$::KanMX6*	Figure 3	This work
KLP686		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 nrc1Δ::KanMX6*	Figure 3	This work
KLP402		*h⁺ ade6-M216 ura4-D18 leu1-32 his3-D1 cut3-477::NatMX6*	Figure 3	This work
KLP714		*h? ade6-M2XX ura4-D18 leu1-32 his3-D1 cut3-477::NatMX6 nrc1Δ::KanMX6*	Figure 3	This work
KLP769		*h? ade6-M2XX ura4-D18 leu1-32 his3-D1 cut3-477::NatMX6 Nrc1.HA$_3$::KanMX6*	Figure 3	This work
KLP773		*h? ade6-M2XX ura4-D18 leu1-32 his3-D1 cut3-477::NatMX6 nrc1-BD1*.HA$_3$::KanMX6*	Figure 3	This work
KLP775		*h? ade6-M2XX ura4-D18 leu1-32 his3-D1 cut3-477::NatMX6 nrc1-BD2*.HA$_3$::KanMX6*	Figure 3	This work
KLP771		*h? ade6-M2XX ura4-D18 leu1-32 his3-D1 cut3-477::NatMX6 nrc1-BD1*/BD2*.HA$_3$::KanMX6*	Figure 3	This work
KLP689		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 Nrc1.HA$_3$::KanMX6*	Figures 4-6, S3, S5-6	This work
KLP797		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 Cka1.Flag$_5$::HphMX6*	Figure 4	This work
KLP800		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 Nrc1.HA$_3$::KanMX6 Cka1.Flag$_5$::HphMX6*	Figure 4	This work
KLP802		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 nrc1-BD1*.HA$_3$::KanMX6 Cka1.Flag$_5$::HphMX6*	Figure 4	This work
KLP803		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 nrc1-BD2*.HA$_3$::KanMX6 Cka1.Flag$_5$::HphMX6*	Figure 4	This work
KFP26	KGY425, AP26	*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1*	Figures 4, S3, S5	K.L Gould
KLP707		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 ckb1Δ::KanMX6*	Figures 4, S1	This work
KLP709		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 ckb2Δ::KanMX6*	Figures 4, S1	This work
KLP402		*h⁺ ade6-M216 ura4-D18 leu1-32 his3-D1 cut3-477::NatMX6*	Figures 4, S1	This work
KLP743		*h? ade6-M2XX ura4-D18 leu1-32 his3-D1 cut3-477::NatMX6 ckb1Δ::KanMX6*	Figures 4, S1	This work
KLP745		*h? ade6-M2XX ura4-D18 leu1-32 his3-D1 cut3-477::NatMX6 ckb2Δ::KanMX6*	Figures 4, S1	This work
KLP722		*h? cdc25-22 Cut3.Flag$_5$::HphMX6 Cka1.HA$_3$::KanMX6*	Figures 4, 6, S4	This work
KLP725		*h? cdc25-22 Cut3.Flag$_5$::HphMX6 Cka1.HA$_3$::KanMX6 nrc1Δ::NatMX6*	Figure 4	This work
KLP706		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 Cka1.HA$_3$::KanMX6*	Figures 4, 5, S3, S5-6	This work
KLP694		*h? cdc25-22 cut3.Flag$_5$::HphMX6 Nrc1.HA$_3$::KanMX6*	Figures 4, 6, S3-4, S6-7	This work
KLP689		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 Nrc1.HA$_3$::KanMX6*	Figure S2	This work
KLP813		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 NatMX6-Fba1$_p$-Nrc1.HA$_3$::KanMX6*	Figure S2	This work
KLP814		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 NatMX6-Adh1$_p$-Nrc1.HA$_3$::KanMX6*	Figure S2	This work
KLP816		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 NatMX6-Lys4$_p$-Nrc1.HA$_3$::KanMX6*	Figure S2	This work
KLP817		*h⁻ ade6-M210 ura4-D18 leu1-32 his3-D1 NatMX6-Ade4$_p$-Nrc1.HA$_3$::KanMX6*	Figure S2	This work
KLP828		*h? ade6-M2XX ura4-D18 leu1-32 his3-D1 cut3-477::NatMX6 NatMX6-Lys4$_p$-Nrc1.HA$_3$::KanMX6*	Figure S2	This work
KLP830		*h? ade6-M2XX ura4-D18 leu1-32 his3-D1 cut3-477::NatMX6 NatMX6-Ade4$_p$-Nrc1.HA$_3$::KanMX6*	Figure S2	This work
KLP829		*h? ade6-M2XX ura4-D18 leu1-32 his3-D1 cut14-208::NatMX6 NatMX6-Lys4$_p$-Nrc1.HA$_3$::KanMX6*	Figure S2	This work
KLP831		*h? ade6-M2XX ura4-D18 leu1-32 his3-D1 cut14-208::NatMX6*	Figure S2	This work

Name	Alt. name	Genotype	Used	Source
		NatMX6-Ade4$_p$-Nrc1.HA$_3$::KanMX6		
KLP22		*h$^+$ ade6-M216 ura4-D18 leu1-32 his3-D1 Mis16.CTAP(2X)::KanMX6*	Table 1	This work
KLP19		*h$^+$ ade6-M216 ura4-D18 leu1-32 his3-D1 Hat1.CTAP(2X)::KanMX6*	Table 1	This work
KLP738		*h$^+$ ade6-M216 ura4-D18 leu1-32 his3-D1 Mis18.CTAP(2X)::KanMX6*	Table 1	This work
KLP219		*h$^+$ ade6-M216 ura4-D18 leu1-32 his3-D1 Nrc1.CTAP(2X)::KanMX6*	Table 1	This work

(C) Oligonucleotides

Name	Sequence	Used
C1 (cnt1)-For	5'-tcgatgaaagaaaagcatttga-3'	Figure S1
C1 (cnt1)-Rev	5'-gcaaatggccagagtatttca-3'	
C3.5 (cnt1)-For	5'-cggcggcgttataaactaag-3'	Figures 2, 3, 4, 6 & S1
C3.5 (cnt1)-Rev	5'-tactccatggcgtttccttc-3'	
C3.5' (cnt1)-For	5'-ccgttgcaacttacatcagc-3'	Figures 4 & S4
C3.5' (cnt1)-Rev	5'-cgaaagatggtcaattgctt-3'	
C9 (cnt1)-For	5'-gaacaaaatagtaagcgttagtgt-3'	Figure S1
C9 (cnt1)-Rev	5'-ctgcttgctcttttcgttcc-3'	
C4 (cnt1)-For	5'-ccagaattttccagacaatcg-3'	Figure S1
C4 (cnt1)-Rev	5'-tcggttaatgggaaaatcaaa-3'	
C5 (cnt1)-For	5'-caccttctggagttctttgga-3'	Figure S1
C5 (cnt1)-Rev	5'-atgcagggttatcggacaat-3'	
I1 (imr1)-For	5'-ggcaatgtcacaaagtttcaa-3'	Figure S1
I1 (imr1)-Rev	5'-tgaataacaatgctggtagcc-3'	
I2 (imr1)-For	5'-tcagtaatcacatccgatgaaca-3'	Figure S1
I2 (imr1)-Rev	5'-ttttcgatggacaccactctt-3'	
I3 (imr3)-For	5'-cgaatatcgtgaaatttgcttg-3'	Figure S1
I3 (imr3)-Rev	5'-tgaaggctgttgatttgtgg-3'	
I4 (imr1)-For	5'-tttttcgctcatattcgttgaa-3'	Figure S1
I4 (imr1)-Rev	5'-tcccagtaaatcaggtttgga-3'	
O2 (otr1)-For	5'-tcatgaatctttcccaagca-3'	Figures 1 & S1
O2 (otr1)-Rev	5'-gcaccgtttttccaaatgtc-3'	
GFR (chr2)-For	5'-gcatcgtttttcgcacaata-3'	Figures 2, 3, 4, 6, 7, S1 & S4
GFR (chr2)-Rev	5'-catggcatggcattttgtta-3'	
A1 (chr1)-For	5'-tctgatgccattggttttga-3'	Figure S4
A1 (chr1)-Rev	5'-caaaacagcctccttaacttca-3'	
B2 (chr1)-For	5'-accgcaccaatgtttctttatt-3'	Figure S4
B2 (chr1)-Rev	5'-tgtcgtgcgagtatccctacta-3'	
C1 (chr1)-For	5'-ggcccaaggtgttgacatta-3'	Figure S4
C1 (chr1)-Rev	5'-ggcccatggcaaagtaagta-3'	

D1 (chr1)-For D1 (chr1)-Rev	5'-accaattgttcctactgagcaaa-3' 5'-ttgtgtgcgttccaactaactaa-3'	Figure S4
D2 (chr1)-For D2 (chr1)-Rev	5'-tttgcaacaagtgcgtcaat-3' 5'-gatgctagtgcatggagcaa-3'	Figure S4
E1 (chr2)-For E1 (chr2)-Rev	5'-tagcgagaatagctttagcatgg-3' 5'-cgatagggaatacattacccaca-3'	Figure S4
G1 (chr2)-For G1 (chr2)-Rev	5'-tttggtcaaacaaatcgatca-3' 5'-ttaattgtgtggcgcttgac-3'	Figures 4, 7 & S4
H1 (chr3)-For H1 (chr3)-Rec	5'-ttgtttttgccaaatggttttat-3' 5'-aaacaagtttcaatgagcgaaaa-3'	Figures 4, 7 & S4

SUPPLEMENTARY FIGURES

Figure S1 - Hat1-Mis16-dependent centromeric H3ac and H4ac levels at the core-centromere anti-correlate with condensin through mitosis (related to **Figures 1** & **2**). **(A)** *hat1*Δ / *nrc1*Δ show a negligible additive effect in their individual rescue of *cut3-477*. Strains were isolated by crossing and tetrad dissection, spotted as 10-fold serial dilutions onto non-selective YES plates, and incubated as indicated. WT, wild type. **(B)** Cell-cycle synchronization by *cdc25-22*. Populations were arrested at the G_2/M boundary by incubation at a non-permissive temperature for *cdc25-22* before synchronized release. Aliquots were then collected at 25-minute intervals for ChIP or cell-septation analyses (Kim et al., 2009). **(C)** Schematic depicts the fission yeast chromosome I centromere, including the core (*cnt*, ~ 4kb), inner (*imr*, ~ 8kb) and outer (*otr*, ~ 18kb) repeats, and the relative location of tRNAs (black bars) and various ChIP primer pairs (*O2* - *C1*; see **Table S3**). The core and innermost repeats are ~ 90% cenH3 (*Sp* Cnp1), with the outermost repeats heterochromatic (H3-K9me2/3) (Kim et al., 2009; Pidoux et al., 2009; Williams et al., 2009). **(D)** Condensin enrichment across the metaphase centromere (see also **Figures 6B** & **S3**). Cells were synchronized by *cdc25-22* and ChIP used to monitor condensin (Cut3.HA_3) at G_2/M (T_0) and metaphase (T_{75}). In each duplex PCR the upper band is the test region (see panel **C**), lower band (GFR; Gene Free Region) is a condensin-free location as a background control (Kim et al., 2009). **(E-I)** Centromeric H3ac and H4ac are mitotically regulated in a Hat1-dependent manner to temporally anti-correlate with condensin. Cells were *cdc25-22* synchronized and ChIP used to monitor condensin and the indicated histones / modifications thereof at the chromosome I centromere core or outermost repeats (*C3.5* or *O2*: see panel **C**) at times indicated. The septation index (SI; peak shaded) and ChIP profiles of Cut3, H3-S10phos, H3-K4me3, H3-K9me2, H3 and histone variant CenH3 (Cnp1) confirmed synchronous progression and equivalent antibody access at each time point (Chen et al., 2008;

Kim et al., 2009). Panels **E / F** (related to **Figure 2C**), **G**, **H** and **I** (related to **Figure 2D**) are derived from different synchronized populations. **(J)** Mis16.HA$_3$ binding at the centromere parallels mitotically regulated H3-K9ac (related to **Figure 2F**). Note delayed mitotic progression in Mis16.HA$_3$ relative to WT cells (similar to *hat1*Δ in panel **H**). **(K)** Mis16.HA$_3$ (see **Figure 2G**) and *ckb1*Δ (see **Figure 4**) repair the anaphase chromosome segregation defects of *cut3-477*. Replicate cultures were shifted from 25ºC to 34ºC (non-permissive for *cut3-477*) for three hours, fixed, and the percentage (mean ± s.d.) of late anaphase cells with chromosome segregation defects determined.

Figure S2 - Analyses of Nrc1 (related to **Figures 2** & **3**). **(A-C)** Cells show a dose-dependent response to Nrc1 levels (related to **Figure 2**). **(A)** Nrc1 protein levels were modulated by 'knocking-in' alternate promoters of predicted expression outputs to Nrc1.HA$_3$: *fba1*$_{pr}$ (~ 256 mRNAs/hr) > *lys4*$_{pr}$ (~ 64 mRNAs/hr) > *ade4*$_{pr}$ (~ 32 mRNAs/hr) (Hiraoka et al., 2009) and the resulting Nrc1.HA$_3$ expression determined by immunoblotting. Nrc1.HA$_3$ runs at ~ 90 kD (*); the cross-reacting lower kD bands are likely break-down products since not seen in the untagged strain. Tbp1 is a loading control. **(B)** Nrc1 overexpression (*fba1*$_{pr}$ or *adh1*$_{pr}$ driven) led to slow-growth and compromised cell health (note red staining of colonies due to accumulation of the vital stain phloxin; *Sigma*). These cells also exhibited defects in mating / sporulation / germination (not shown). Strains were spotted as 10-fold serial dilutions onto YES/phloxin plates and incubated as indicated. **(C)** Nrc1 showed dose-dependent interactions with mutant *condensin*: overexpression (*lys4*$_{pr}$) is synthetic and reduced expression (*ade4*$_{pr}$) suppressive. Strains were spotted as 10-fold serial dilutions onto YES plates and incubated as indicated. **(D)** *nrc1*Δ showed negative genetic interactions with mutants related to chromosome segregation (*DASH* complex), centromere function, histone acetylation or histone variant H2A.Z (Pht1)

function. The latter group includes mutants that compromise H2A.Z deposition (SWR-Complex) or acetylation (*pht1-NΔ*, *4KR*, or *4KQ*): of note many of these alleles also suppress mutant *condensin* (Kim et al., 2009) (**Figure 1B**). The calculation of genetic scores is as previously (Roguev et al., 2008; Ryan et al., 2012). **(E)** Specific binding of Nrc1-BD1 to the acetylated histone H4 N-terminus. Purified recombinants (Nrc1 residues S199-G526: WT, BD1*, BD2* or BD1*/BD2*) were used to probe histone-peptide arrays in duplicate, and consistent areas of relative enrichment identified from heat maps of the normalized mean intensity (set to a maximal value of one in each array: see **Experimental Procedures** (Fuchs et al., 2010; Rothbart et al., 2012)). Panel is an extension of **Figure 3E**. Of note, all hyperacetylated H4 peptides (≥ 4 of K5ac, K8ac, K12ac, K16ac and K20ac) on the array were enriched for WT Nrc1-binding. Specific peptide #'s are from (Rothbart et al., 2012). **(F)** Reproducibility of the approach. Scatter-plot compares the binding of *nrc1-BD2** in two independent analyses (12 peptide-spot repeats per array; pairwise correlation co-efficient 0.64). **(G)** Nrc1 shows limited selectivity for the specific acetylated residues on di-acetylated histone H4 (average and SEM from 24 individual spots per peptide over two independent analyses).

Figure S3 - Comparison of NCT and condensin localization (related to **Figures 4** & **6**). ChIP-seq data was loaded into *Genplay* (Lajugie and Bouhassira, 2011) as individual tracks (50 bp windows; normalized and INPUT subtracted) relative to the annotated *Sp* genome (Sanger Center release *pombe090511*): the online version of this paper links to a *Genplay* project to dynamically visualize each track. As noted, specific ChIP-seq libraries were prepared from asynchronous (i.e. primarily G_2) or cell-cycle synchronized cells (see **Figure 6A**). All sample data have been deposited in NCBI's Gene Expression Omnibus (Edgar et al., 2002) and are accessible through GEO series accession number GSE53955.

(A-D) Centromeric distribution. **(A)** Upper schematics depict the structure of all three fission yeast centromeres (window size (in bp) around centerpoint as indicated). Lower annotation tracks depict the genomic elements (e.g. genes, tRNAs and miscRNAs) at each location. **(B)** TBP peaks identify the tRNAs that demark the inner / outer centromere and peri-centromeric boundary; H3-K9me2 enrichment identifies heterochromatin (i.e. the outer repeats of each centromere) (Partridge, 2008; Wood et al., 2002). **(C)** Nrc1.HA_3 is mitotically regulated to nadir at metaphase (T_{75}). Of note the Nrc1 peaks in synchronized are less defined than in asynchronous populations, suggesting that delocalization of the factor may begin at G_2/M. **(D)** Condensin (Cut3.$FLAG_5$) is most highly enriched over the core centromere at metaphase (T_{75}) (Nakazawa et al., 2008): compare the 'Peak count' scale for each track to that along the chromosome arms in **Figure S5**.

(E-I) Distribution at representative regions along the chromosome arms. The red-boxed regions were extracted for **Figures 4 - 6** but are aligned here to facilitate comparison. Note the reduced 'Peak count' scale for each track relative to at the centromere (**B-D**). **(E)** Each 10 kb window is named for the centered genomic feature (annotation track; red transcripts go to right): this does not mean that the factor under test is definitively regulated by the expression (or otherwise) of that feature. *qPCR primers* indicates the location of directly tested regions (e.g. **Figure S5**). **(F)** The strong similarity between regions enriched for Nrc1, Cka1 and Taf7 (elements of the NCT complex); **(G)** Nrc1 delocalization through mitosis, with the nadir at metaphase (~ T_{75}); **(H)** Condensin occupies many of the same locations as the NCT, but temporally anti-correlates with Nrc1. **(I)** TBP, H3-K4me3 and H3-K36me3 identify the promoters, 5' ends and ORFs of transcriptionally active genes.

Figure S4 - qPCR analysis of *ChIP-seq* peaks (related to **Figures 4F** & **6F**). qPCR-ChIP (rather than duplex PCR-ChIP as in **Figures 2C**, **2D**, **2F**, **3C** & **4C**) was used to test representative *ChIP-*

seq predictions. All samples were prepared from synchronized populations (**Figure 6A**). **(A-B)** qPCR confirmed nine sites of Nrc1 / condensin enrichment: eight on the chromosome arms (*A-H*: **Figure S3E**) and at the core centromere (*c3.5'*: **Figure S3A**). Data (mean + SD) is normalized to *GFR* (set to 1). **(C-D)** The association of Cka1 with the chromosome arms (*G1*, *H1*, *A1* and *B2*; see also **Figure 6F**) and core centromere (*c3.5'*; see also **Figure 4C**) is mitotically regulated. Data (mean + SD) is normalized to *GFR*-T_0 (set to 1).

Figure S5 - *ChIP-seq* peak behavior supports a TFIID-independent role for Taf7 (related to **Figures 4E** & **5G** and **Table S2**). **(A)** ChIP-seq data was loaded into *Genplay* (Lajugie and Bouhassira, 2011) as individual tracks (50bp windows; normalized and INPUT subtracted) relative to the annotated *S. pombe* genome (Sanger Center release *pombe090511*). Peaks in each track were then automatically identified / quantified by optimized parameters: Regions of 10 successive Windows; Gap permitted, 1 window; Threshold Island Score (IS), 10; Minimum Island Length, 4 windows (' 6 windows for H3-K36me3). Data was then exported to *Excel*, TEL3L/R and CEN1-3 removed (see **Experimental Procedures** and **Table S2**), and the size-distributions of the remaining peak widths graphed in *Prism*. ChIP-seq samples were from asynchronous populations (Asyn) with the exception of Cut3 (**H** / **H'**; T_{75} from **Figure 6F**). **(B)** H3-K4me3 is highly enriched over a ~ 780 nt region promoter-proximal to active RNApII genes (see **Figure S3I**). * Many H3-K4me3 'peaks' > 1 kb are actually a merge (during automated calling) of distinct peaks from two adjacent divergently transcribed genes. *Excl* indicates the number of excluded peaks > 2000 nt width. **(C)** H3-K36me3 is enriched through the body of active RNApII genes (see **Figure S3I**); hence the wide range of peak widths. **(D / D')** TBP peaks: All (IS >10) / Major (IS >100). **(E / E')** Taf7 peaks. All (IS >10) / Major (IS >100). **(F / F')** Nrc1 peaks. All (IS >10) / Major (IS >100). **(G / G')** Cka1 peaks. All (IS >10) / Major (IS >100). **(H / H')** Cut3 peaks. All (IS >10) / Major (# IS >50).

Figure S6 - NCT and condensin localization at the histone loci (related to **Figure 5G**). *ChIP-seq* data (from asynchronous or cell-cycle synchronized cells as noted) was loaded into *Genplay* (Lajugie and Bouhassira, 2011) as individual tracks (50bp windows; normalized and INPUT subtracted) relative to the annotated *S. pombe* genome (Sanger Center release *pombe090511*). **(A)** Each 10 kb test window is named for the centered histone locus (upper schematic), all of which are poorly transcribed outside of S-phase (Marguerat et al., 2006; Oliva et al., 2005; Peng et al., 2006). **(B-E)** The relative enrichment of various factors or histone modifications by chromosomal position. **(B)** TBP and Taf7 are most enriched at the intergenic promoter regions, while Nrc1 and Cka1 bind across each histone gene (note unidirectional spread at the unpaired *hta2*$^{+}$ locus). **(C)** Note the nadir of Nrc1 binding at metaphase (T_{75}). **(D)** Condensin is highly enriched at the 3′ end of each histone gene. Of note maximal Cut3 recruitment at each locus is delayed relative to most other locations (i.e. $T_{100} > T_{75}$: compare to **Figure S3**). **(E)** TBP, H3-K4me3 and H3-K36me3 identify the promoters, 5′ ends and ORFs of transcriptionally active genes.

Figure S7 - Condensin shows a strong preference for the structural RNA genes (related to **Figure 6**). **(A-C)** Data is presented as the percentage of Cut3 peaks (major or all: see **Table S2**) per feature, and percentage coverage of each feature. For panel **C** the *Fisher's exact test* (by *R*) was used to estimate the probability of the observed coverage of structural RNAs: *p-value* for both datasets $< 2.2 \times 10^{-16}$ (the smallest value *R* will provide).

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