The Unique Centromeric Chromatin Structure of Schizosaccharomyces pombe Is Maintained during Meiosis*

Received for publication, January 24, 2002, and in revised form, March 12, 2002 Published, JBC Papers in Press, March 21, 2002, DOI 10.1074/jbc.M200765200

Julia B. Smirnova and Ramsay J. McFarlane‡

From the Molecular and Cell Biology Group, School of Biological Sciences, Memorial Building, University of Wales-Bangor, Deiniol Road, Bangor, Gwynedd LL57 2UW, United Kingdom

In meiosis I sister centromeres are unified in their polarity on the spindle, and this unique behavior is known to require the function of meiosis-specific factors that set some intrinsic property of the centromeres. The fission yeast, Schizosaccharomyces pombe, possesses complex centromeres consisting of repetitive DNA elements, making it an excellent model in which to study the behavior of complex centromeres. In mitosis, during which sister centromeres mediate chromosome segregation by establishing bipolar chromosome attachments to the spindle, the central core of the S. pombe centromere chromatin has a unique irregular nucleosome pattern. Deletion of repeats flanking this core structure have no effect on mitotic chromosome segregation, but have profound effects during meiosis. While this demonstrates that the outer repeats are critical for normal meiotic sister centromere behavior, exactly how they function and how monopolarity is established remains unclear. In this study we provide the first analysis of the chromatin structure of a complex centromere during meiosis. We show that the nature and extent of the unique central core chromatin structure is maintained with no measurable expansion. This demonstrates that monopolarity of sister centromeres, and subsequent reversion to bipolarity, does not involve a global change to the centromeric chromatin structure.

Functional centromeres are required for connection of the chromosomes to the spindle apparatus during mitotic and meiotic cell divisions (reviewed in Refs. 1–5). During meiosis centromeres undergo tandem functional reconfigurations (reviewed in Ref. 6). First, during or preceding meiosis I, centromeres are remodeled to a format that will ensure a reductional division by forming monopolar kinetochore attachments of sister chromatids. Subsequently, centromeres revert to a format that will enable bipolar attachment of sister centromeres during meiosis II, resulting in equational chromosome segregation. The factors responsible for establishing the polarity of centromeres during meiosis are intrinsic to the chromosomes and are not dependent upon the status of the meiotic spindle or the surrounding cellular environment (7).

A number of proteins have been identified that are required for the establishment of monopolar sister centromere spindle associations in meiosis I. In Saccharomyces cerevisiae, where “point-centromeres” are defined by a single nucleosome, the MAM1 gene product is essential for monopolarity, although no MAM1 homologues have been identified in any other organism (8). The fission yeast, Schizosaccharomyces pombe, possesses more complex centromere structures covering up to 110 kbp of repetitive DNA (9–13). Remodeling of S. pombe centromeres into a meiosis I configuration is dependent upon the Rec8 meiosis-specific cohesin (14) and the Bub1 kinetochore-associated spindle checkpoint protein (15). In wild type cells the Rec8 cohesin is located at the centromeres prior to both meiosis I and II, indicating that the presence of Rec8 at the centromeres alone is not sufficient to establish monopolar kinetochores (14).

The repetitive regions of S. pombe centromeres have been classified into three distinct sequence elements, the outer repeat elements (otr; also termed K repeats), the inner most repeats (imr), and the central regions (CC/cnt) (Fig. 1) (10–12). The otr regions consist of three different repeat units, termed the dg, dh, and cen253 repeats, that are present to varying degrees in the otr regions of each of the three S. pombe centromeres (Fig. 1) (13). The imr regions are imperfect inverted repeat elements that flank the cnt regions and the cnt regions share significant homology with each other (48% over 1.4 kbp), suggesting they are functionally similar (13).

Despite the classification of the centromeric DNA sequence into three distinct domains, S. pombe cen1 appears to consist of only two functional domains during mitosis, a central region made up of the imr and the cnt and an outer region principally consisting of the otr region and cnt-distal sections of the imr. These two domains associate with a different group of proteins that localize to discrete positions within the nucleoplasm during interphase (16, 17). These disparate sets of proteins are required for centromere function, and it is proposed that there is a “flexible” boundary between the two regions, possibly defined by tRNA genes within the imr (16, 18). Studies of the chromatin structure of the centromeres of S. pombe have revealed that during the mitotic cell cycle the chromatin structure of the cnt and imr regions does not posses the regular nucleosome pattern observed throughout the rest of the genome (11, 12). In contrast to this, the otr regions do maintain the regular nucleosome pattern observed for bulk chromatin, throughout the mitotic cell cycle (11, 12). Recent work on mammalian chromosomes indicates that there is also a unique chromatin organization in centromere satellite regions (19), indicating that a specific higher order chromatin structure is most likely a unique feature of all centromeres (20). In S. pombe an enhancer element, located in the otr regions (K repeat region), is essential for maintaining the “irregular” chromatin structure in the centromere core region (21, 22). A model has been proposed suggesting the enhancer element is required to fold the centromeric DNA into a loop that is pivotal for the formation and/or maintenance of the central core chromatin structure (21). It is likely that the S. pombe CENP-A protein (Cnp1) is central to the formation of the unique chromatin in the centro-
meric region as it is associated with the imr and cnt, but not the otr, regions; consistent with this, cnp1- cells do not form the unusual central core chromatin structure (23). This suggests that the replacement of histone H3 in the centromeric regions by CENP-A may mediate the formation of a more fluid chromatin structure required for correct mitotic kinetochore formation. Two other S. pombe proteins, Mis6 and Mis12, are also required for the formation of the "unusual" centromere chromatin structure and both are needed for correct sister chromatid segregation (24, 25). Localization of Cnp1 to the central regions is dependent upon Mis6 function but not Mis12, indicating that Mis6-dependent establishment of Cnp1 alone is not enough to create a functional centromere unit exhibiting the irregular chromatin profile (23).

One intriguing feature of S. pombe centromeres is that certain regions of the repetitive elements flanking the cnt inner core can be deleted without any effect on the fidelity of mitotic chromosome segregation; however, the removal of these flanking repeats results in a reduction in the fidelity of segregation during meiosis I division with a high incidence of precocious sister chromatid separation (26, 27). This suggests that the chromosomal elements of S. pombe centromeres function differentially during the mitotic and meiotic cell cycles.

In this report we show that the functional reconfiguration of centromeres during the meiotic cell cycle does not involve a global change to the nature and extent of the specialized structure of the centromeric chromatin, and we discuss the implications of this in proposing the way in which centromere reconfiguration is mediated.

Experimental Procedures

Strains and Plasmids—Meiotic inductions were carried out using GP38 (BP184), pat1-114/pat1-114 ade6-M210/ade6-M216 urd-294/urd-294 leu1-32/leu1-32 arg1-2/arg1-2 end1-458/end1-458 h+ h− (28). Post-miosis I blocks were initiated using BP440; ade6-M216/ ade6-M210 leu1-32/leu1-32 mes1Δ/LEU2 /mes1Δ/LEU2 h+ h−. All strains were propagated and stored using standard conditions (29).

Plasmids were used to produce the Southern hybridization probes. otr dgI-specific probe was an 800-bp HindIII-EcoRI fragment from pKT311 (TM1-specific probe was a 700-bp EcoRI-BglII fragment from pBS-TM (30). The imr1B probe was a 1.6-kbp BamHI-EcoRI fragment from pJ3S. pJ3S was constructed by cloning a 1.6-kbp PCR product from the otr1B region using genomic DNA as a template; the following primer sequences were used: forward, 5′-TGTCGAATT- GAGATGTAAACG-3′; reverse, 5′-CTGTCGAGGCTAAGTACTG-3′. This fragment was cloned directly into pCR2.1-TOPO using the TOPO TA cloning kit (Invitrogen) to create pJ3S.

Meiotic Inductions—G1 arrest and synchronous meiotic induction of the pat1-114 homozygous strain was carried out as described by Cervantes and co-workers (28). Homozygous mes1Δ cells were blocked prior to meiosis II as described by Kishida and co-workers (31).

Chromatin Isolation—To standardize the amount of chromatin DNA isolated, a fixed number of fresh cells were used for each preparation (1 × 10⁴, 0.5 g of wet weight). Cells were harvested and resuspended in 2 ml of preculture solution (20 mM Tris-HCl (pH 8.0), 0.07% 2-mercaptoethanol, 3 mM EDTA (pH 8.0)) and incubated at 30 °C for 15 min with gentle shaking. Cells were reharvested and resuspended in 2 ml of freshly prepared lyticase buffer (37.5 mM Tris-HCl (pH 7.0), 0.75 mM sorbitol, 1.25% glucose, 6 mM EDTA (pH 8.0)) and lyticase (2 mg/ml). The time of incubation of cells to produce spermatogonia varied from sample to sample and was strain and meiotic time point-dependent. All subsequent steps were carried out on ice. Spermatogonia were washed once in 1 M sorbitol and resuspended in 2 ml of lysis buffer (18% Ficoll-400, 10 mM KH₂PO₄, 10 mM KH₂PO₄ at pH 6.8, 1 mM MgCl₂, 0.25 mM EDTA, 0.25 mM EDTA (pH 8.0), 1 mM phenylmethanesulfonyl fluoride). Chromatin was harvested at 15,000 × g for 40 min at 4 °C.

Micrococcal Nuclease (MNase) Digestion—For MNase digestion, pelleted chromatin preparation was resuspended in 1 ml of PC buffer (20 mm PIPES (pH 6.4), 0.1 mm CaCl₂, 0.5 mm MgCl₂, 1 mm phenylmethanesulfonyl fluoride), and MNase (Roche Molecular Biochemicals) was added to 500 units/ml (this corresponds to 500 units/0.5 g wet weight cells, see above). Preparations were incubated at 32 °C and stopped at varying time points following addition of MNase. MNase reactions were terminated by adding 1% SDS and 10 mM EDTA (pH 8.0). DNA was purified as described by Mizuno and co-workers (32). DNA was analyzed on a 1.2% agarose gel and transferred to a nylon membrane (Hybond N, Amersham Biosciences) and subjected to Southern hybridization with specific probes using Amersham Biosciences hybridization buffers as described by the manufacturer. Radiographs were obtained using a Bio-Rad Personal Imager, and nucleosome banding was distinguished using Bio-Rad Quantity One software.

Results

cnt Distal Sequences of imr1 Are Not Unique to the Inner Core of cen1—The previously proposed structures of the S. pombe centromeres suggests that the DNA sequence of the imr regions is unique to each centromere (see Fig. 1 for updated centromere maps; Ref. 12). To confirm and update this, prior to designing probes for chromatin analysis (see below), the imr1 sequences were subclassified into two sections, one distal to the central region (imr1A) and one proximal to the central region (imr1B) (Fig. 1). Sequences from both regions were used in blast searches against the S. pombe genome sequence, which contains sequence information for over 81% of the centromeric DNA for all three chromosomes (www.sanger.ac.uk/Projects/S_pombe blast_server.shtml; 13). The imr1A sequence used for the search was the full 1550-bp sequence between the reported otr dgI boundary and the alanine tRNA gene located within the center of the imr (as reported by Takahashi et al. (12)). The data base search using imr1A revealed a number of regions of identical sequence within cen2 and cen3, clearly demonstrating the imr1A region contains sequences that are not unique to cen1. The extent of the identity ranged from regions covering 208 bp to 1083 bp (identity ranged from 95 to 99%). These regions represent significant sequence duplications of imr1 DNA within the centromeres of the other two chromosomes. Importantly, the imr1A sequences in cen2 and cen3 are located on the cnt distal side of the otr-imr boundaries of imr2 and imr3. The majority of the duplications are relatively small (up to 400 bp) and are repeats of the 400-bp region immediately adjacent to the otr1L-dgI region. These short duplications appear to be within stretches of dh DNA on the other centromeres as illustrated in Fig. 1B. We therefore suggest that the cnt-distal portion of imr1 is made up of a region of dh DNA. Interestingly, this would mean that imr regions on all three centromeres are directly flanked by dh sequences.

As one larger stretch of imr1A sequence is located at the most cnt-distal point of otr2L. This sequence structure suggests that this region (see Fig. 1B) could be an inverted duplication of the dgI-imr1A boundary of cen1. A second imr1A duplication of 962 bp is located in a region distal to the dgIII-dhIII repeats on the right-hand arm of cen3; the significance of these duplications is unclear, although there may be advantages to having imr1A elements at some of the distal flanks of centromeres.

The duplication of imr1A sequences in otr regions of the other centromeres prevented these sequences being used for hybridization-based analysis of imr1A chromatin structure (see below). Moreover, the identification of these duplications in otr regions in cen2 and cen3 supports the model that proposes the imr1A region of cen1 is functionally distinct from the imr1B, cen1B, and cen1L regions (18).

From this analysis of the centromere sequence, probes were designed that would be specific to the otr (highly conserved dg region), imr1B, and cnt regions (Fig. 1). These probes were used in all the chromatin analyses described below.

Unusual Centromeric Structure Is Maintained in Diploid Cells—Previous analyses of centromeric chromatin structure in
**S. pombe** have employed haploid cells. To elucidate the nature of the centromeric chromatin structure during meiosis, diploid cells were induced to traverse meiosis in a highly synchronous fashion (see below). Chromatin analysis of asynchronous (data not shown; Fig. 6) and G1 (Fig. 2) arrested diploid cells show that the *imr1B* and *cnt1* chromatin exhibits a smearing pattern in response to MNase treatment. In contrast, the *otr* regions have the more uniform nucleosome pattern of bulk chromatin. These data are similar to those obtained for haploid cells, indicating that the structure of centromeric chromatin is similar in both haploids and diploids.

**Unique Chromatin Is Maintained within the Centromere Central Core during Meiosis and Does Not Exhibit Expansion into the Outer Most Repeats**—Loss of *otr* sequences (K' and L sequences, using the nomenclature of Clarke and co-workers (26, 27)) from *cen1* results in normal centromere function during mitosis, but aberrant meiotic function (26, 27). This leaves open the question of how centromeres are reconfigured and whether or not this involves a sustained global change to the structural organization of the centromeric chromatin. To address this, a diploid strain homozygous for the *pat1*–114 mutation and homozygous at the *mat* locus (*h*+/−*h*−) was employed to induce a synchronous meiosis from which chromatin extracts were made at specific time points. Pat1 is a kinase that regulates entry into meiosis by inactivating factors required for meiotic entry (reviewed in Ref. 33). *pat1*–114 cells can be in-

---

**Fig. 1. Regional map of the three S. pombe centromere regions (adapted from Wood et al. (13)).** A, the map indicates the positions of the three major sequence domains of the centromeres, *otr, imr,* and *cnt* regions and the approximate positions of centromeric tRNA genes. *imr1* is subdivided into *imr1A* and *imr1B* subdomains. Regions exhibiting large duplication with *imr1A* (refer to "Results") are indicated by dark pink boxes. Small regions of homology with the *cnt1*-distal region of *imr1A* are indicated with an asterisk. Two regions are indicated in *cen2* and *cen3*, where no sequence data are currently available; the question mark adjacent to the asterisk indicates that short *cnt1*-distal *imr1A* homologies may or may not be in these repeat elements. The large open box encompassing the left arm of *otr2L* is the region highlighted in B. B, this region is well annotated in the *S. pombe* database, giving a clear indication that the small *imr1A* duplication is within an *otr dh* region. The numbers correspond to the exact nucleotide position on *S. pombe* cosmid c28F2 (www.sanger.ac.uk/Projects/S_pombe/).
duced to enter a synchronous meiosis by shifting cells grown at the permissive temperature of 25 °C to the restrictive temperature of 34 °C, thereby inactivating the mutant Pat1 kinase. Homozygous pat1Δ–114 diploid cells arrested in G2 prior to thermal induction traverse a highly synchronous meiosis indistinguishable from a normal asygotic meiosis (34).

A number of synchronous asygotic meiose were induced and temporally monitored by measuring key meiotic events, including meiotic commitment (by return to mitotic permissive temperature), premeiotic S phase (FACS), and meiosis I and meiosis II divisions (microscopy). Meiotic inductions exhibited a high degree of reproducibility and temporal profiles were uniform in nature. A maximum of two chromatin preparations were made from each meiotic induction. Chromatin preparations were made prior to premeiotic S phase, following DNA replication but prior to meiosis I division, post-meiosis I (pre-spore formation). Fig. 3 shows the points at which chromatin preparations were made relative to major temporal landmarks. Each chromatin preparation was subjected to MNase digestion and Southern blot analysis using either otr, imr1B, or cnt1 hybridization probes (Fig. 1). In all cases the imr1B and cnt regions exhibited a nonspecific sensitivity to MNase, whereas the otr regions have a regular nucleosome pattern analogous to bulk chromatin; examples of this for three different chromatin preparations are shown in Fig. 4. The presence of the MNase labile chromatin in the central core region is similar to that found during the mitotic cell cycle. Moreover, the extent of the unique chromatin is restricted to the central core and does not extend globally to the otr regions.

Although there is limited homology between the cnt regions (13), the cnt2 sequence has been reported as unique (12). To ensure that the three chromosomes behave in a similar fashion a cnt2-specific hybridization probe was used on a range of chromatin preparations taken from the meiotic time points indicated in Fig. 3. In all cases the cnt2-specific probe showed a smeared chromatin pattern indicating that the central core of cen2 behaves in a similar fashion to cnt1 throughout meiosis (Fig. 5).

To gain further temporal resolution between meiosis I and meiosis II divisions and to dismiss any effects of the pat1Δ–114 mutation, a homozygous mes1Δ diploid was constructed. mes1Δ cells fail to complete meiosis and arrest as binucleate cells following meiosis I when Rec8, a modulator of monopolarity, remains localized at the centromere (14). The mes1Δ/mes1Δ diploid was heterozygous at the mating type locus and homozygous pat1Δ. Diploid cells heterozygous at the mat locus will enter meiosis when nitrogen becomes limiting in the media. Despite the lack of synchrony of meiotic initiation in pat1Δ diploids, all cells arrest at the same point between meiosis I and meiosis II. Fig. 6 shows the cnt1 and imr1B regions are extremely sensitive to MNase, whereas the MNase sensitivity of the otr regions mirrors that of the bulk chromatin; these data are consistent with those from the thermally induced meiosis in the homozygous pat1Δ–114 diploids. Both the otr and bulk chromatin exhibit a low background degradation in mes1Δ blocked cells. Moreover, the otr region in the mitotic mes1Δ diploid appears to be slightly more refractory to MNase digestion than bulk chromatin (compare otr and EtBr images in upper panel of Fig. 6). This was not apparent in mes1Δ diploids (Fig. 1) and may reflect some subtle change in centromere chromatin in proliferating mitotic diploid cells. Attempts to employ mutants (for example, met4Δ) to block the mitotic cell cycle prior to meiosis I division resulted in high background degradation of chromosomes, presumably due to the presence of structures that render the chromosomes more labile during arrest such as recombination intermediates.

**FIG. 2. Chromatin structure for the otr, imr, and cnt regions of a G2-arrested diploid cell prior to meiotic induction.** EtBr image shows the nucleosome pattern for bulk chromatin. The three right-hand images are radiographs of Southern filters probed with region specific probes. Mono- to tetranucleosome bands are indicated on the left. The patterns for the otr regions mimic the pattern for bulk chromatin with distinct nucleosome bands. The imr and cnt regions show a more smeared pattern with no clearly quantitatively distinguishable bands (using Quantity One analysis; see “Experimental Procedures”).

**DISCUSSION**

Successful traversal of meiosis involves a number of major changes in the behavior of chromosomes. Pivotal to these events are the changes in the relationship between sister centromeres, switching in early meiosis from mitotic bipolar spindle associations to monopolar associations which maintain cohesion at the centromeres; this is followed by a final reversion back to bipolar spindle associations and loss of centromeric cohesion during meiosis II. Paliulis and Nicklas (7) have recently shown that grasshopper chromosomes from meiosis I cells maintain monopolar association when they are transferred to spindles in meiosis II cells; the reciprocal is also true, with meiosis II chromosomes having bipolar spindle associations when placed onto the meiosis I spindle. This clearly demonstrates that it is factors directly associated with the chromosomes that determine sister centromere polarity and that once polarity is established it cannot be changed by altering the cellular or spindle environment.

A number of proteins are known to be required for the development of sister centromere monopolarity during meiosis I, including the fission yeast spindle checkpoint protein Bub1 and the meiosis-specific cohesin, Rec8 (14, 15). It remains unknown how these factors mediate their effect on the centromere. A number of studies have shown that during mitosis the chromatin within the central core of centromeres is unlike the bulk of chromatin found throughout the genome (11, 12, 19). Moreover, it is known that there are a whole range of proteins that associate with centromeric DNA to form the chromatin structure required for mitotic bipolar spindle associations. How the proteins interact with the DNA to produce the specialized chromatin structure that confers a functional centromere remains conjecture, as does the way in which the centromere structure changes to provide a unique cell division cycle during meiosis.

We took advantage of the fact that the centromeres of the fission yeast are relatively large and complex in nature (13), offering a unique model in which to study the behavior of complex centromeres during the transition from mitosis to meiosis and through the proceeding meiotic cell cycle. Work by Clarke and co-workers (26, 27) identified significant differences in the structural requirements of fission yeast centromeres during mitosis and meiosis. Their work showed that deletion of cnt1 distal sections of one arm of the flanking otr region of cen1 (K' and L as reported by Clarke and co-workers (26, 27)) re-
sulted in no increased missegregation during mitosis, but did result in a significant increase in precocious sister centromere disjunction in meiosis I. This observation suggests that the large more complex centromeres of \textit{S. pombe} are essential to ensure a proper sister polarity reconfiguration and cohesion maintenance during meiosis I. In this report we establish that the boundaries of the centromere central core regions that possess the unique chromatin structure during mitosis are not globally extended into the otr regions during the meiotic cell cycle. Although the chromatin assay employed in this study may have missed very transient changes to the extent of this unusual structure, this seems unlikely as the unusual chromatin is likely to be dependent upon the establishment of the histone H3 homologue, Cnp1 (CENP-A homologue), within the central core in meiosis as it is in mitosis, although this has not...
been directly tested (23). Once Cnp1 establishes the unique chromatin structure that confers functionality to the centromere, it is unlikely that this becomes de-established and re-established in a very short temporal frame.

This study is the first investigation into the global behavior of a complex eukaryotic centromere during meiosis. We show that there is no major change in the nature and extent of the unique chromatin at the central core of S. pombe centromeres during meiosis. During mitosis the unique structure is dependent upon the establishment of the H3 analogue Cnp1 (CENP-A). This work suggests that the mitotic boundaries of Cnp1 establishment are maintained during meiosis, although this has not been directly tested. It has been proposed that the chromatin boundaries in cen1 are set by the tRNA genes located centrally within imr1 (Fig. 1; Ref. 16), a proposal supported by the observation that tRNA genes can function as distinct chromatin boundary elements (18). Our data demonstrate that the limits of the unusual chromatin are maintained throughout meiosis, implying that the imr1 tRNA genes maintain function as chromatin boundary elements during meiosis.

The factors that mediate the maintenance of sister centromere cohesion during meiosis I reductional division do not exert their effect through a global change to the limits of the unique chromatin structure. Thus, the specific requirement for the cnt-distal otr elements for maintaining sister cohesion during meiosis I is not for mediating the expansion of the unique central core. It seems likely that these regions provide a platform for other factors to function. Rec8 provides an attractive candidate, and although Rec8 is enriched at the centromeres, it associates with all centromeric regions during meiosis and not specifically the otr regions (34). Exactly how meiotic reconfiguration of centromeres is mediated remains an open question. However, it is clear from this study that there is not a global change to the chromatin structure at the nucleosome level within the centromeres.

Acknowledgments—We are grateful to R. Allshire, C. Shimoda, G. Smith, and M. Yanagida for providing strains and plasmids. We thank R. Gwilliam, Y. Shaw, and V. Wood for providing unpublished information on the centromere sequences. We also thank A. Battersby, M. Davies, D. Pryce, M. Sanford, S. Whitehall, and J. Wakeman for critical review of this manuscript.

REFERENCES
1. Henikoff, S., Ahmad, K., and Malik, H. S. (2001) Science 293, 1098–1102
2. Pidoux, A. L., and Allshire, R. C. (2000) Curr. Opin. Cell Biol. 12, 308–319
3. Clarke, L. (1999) Curr. Opin. Cell Biol. 11, 212–218
4. Choo, K. H. A. (2001) EMBO J. 20, 520–531
5. Gilbert, N., and Allan, J. (2001) Proc. Natl. Acad. Sci. U. S. A. 98, 11949–11954
6. Choo, K. H. A. (2001) Dev. Cell 1, 165–177
7. Marshall, L. G. (1999) Curr. Opin. Genet. Dev. 9, 445–454
8. Ngan, V. K., and Clarke, L. (1997) Mol. Cell. Biol. 17, 3305–3314
9. Takahashi, K., Chen, E. S., and Yanagida, M. (2000) Science 288, 2215–2219
10. Saitoh, S., Takahashi, K., and Yanagida, M. (1997) Cell 90, 131–143
11. Kiniola, B., Toole, E., McIntosh, J. R., Mellone, B., Allshire, R., Mengarelli, S., Hultenby, K., and Ekwall, K. (2001) Mol. Biol. Cell 12, 2767–2775
12. Donze, D., and Kamakaka, R. T. (2001) EMBO J. 20, 520–531
13. Wood, V., Gwilliam, R., Rajandream, M.-A., Lyne, M., Lyne, R., Stewart, A., Sgouros, J., Peat, N., Hayles, J., Baker, S., et al. (2002) Nature 415, 861–880
14. Watanabe, Y., and Nurse, P. (1999) Nature 400, 461–464
15. Bernard, P., Maure, J.-F., and Javerzat, J.-P. (2001) Nat. Cell Biol. 3, 522–526
16. Partridge, J. F., Bortjorgst, B., and Allshire, R. C. (2000) Genes Dev. 14, 783–791
17. Kiniola, B., O’Toole, E., McIntosh, J. R., Mellone, B., Allshire, R., Mengarelli, S., Hultenby, K., and Ekwall, K. (2001) Mol. Biol. Cell 12, 2767–2775
18. Donze, D., and Kamakaka, R. T. (2001) EMBO J. 20, 520–531
19. Gilbert, N., and Allan, J. (2001) Proc. Natl. Acad. Sci. U. S. A. 98, 11949–11954
20. Choo, K. H. A. (2001) Dev. Cell 1, 165–177
21. Marshall, L. G. (2000) Curr. Opin. Genet. Dev. 9, 445–454
22. Ngan, V. K., and Clarke, L. (1997) Mol. Cell. Biol. 17, 3305–3314
23. Takahashi, K., Chen, E. S., and Yanagida, M. (2000) Science 288, 2215–2219
24. Saitoh, S., Takahashi, K., and Yanagida, M. (1997) Cell 90, 131–143
25. Kiniola, B., Toole, E., McIntosh, J. R., Mellone, B., Allshire, R., Mengarelli, S., Hultenby, K., and Ekwall, K. (2001) Mol. Biol. Cell 12, 2767–2775
26. Clarke, L., and Baum, M. P. (1990) Mol. Cell. Biol. 10, 1863–1872
27. Hahnenberger, K. M., Carbon, J., and Clarke, L. (1991) Mol. Cell. Biol. 11, 2206–2215
28. Cervantes, M. D., Farah, J. A., and Smith, G. R. (2000) Mol. Cell 5, 883–888
29. Moreno, S., Klar, A., and Nurse, P. (1991) Methods Enzymol. 194, 795–823
30. Allshire, R. C., Javerzat, J.-P., Redhead, N. J., and Cranson, G. (1994) Cell 76, 157–169
31. Kishida, M., Nagai, T., Nakaseko, Y., and Shimoda, C. (1994) Curr. Genet. 25, 497–503
The Unique Centromeric Chromatin Structure of *Schizosaccharomyces pombe* Is Maintained during Meiosis

Julia B. Smirnova and Ramsay J. McFarlane

*J. Biol. Chem.* 2002, 277:19817-19822. doi: 10.1074/jbc.M200765200 originally published online March 21, 2002

Access the most updated version of this article at doi: 10.1074/jbc.M200765200

Alerts:
- When this article is cited
- When a correction for this article is posted

Click here to choose from all of JBC's e-mail alerts

This article cites 34 references, 17 of which can be accessed free at http://www.jbc.org/content/277/22/19817.full.html#ref-list-1