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REVIEW

Maize centromeres: where sequence meets epigenetics

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Abstract **The centromere is a highly organized structure mainly composed of repeat sequences, which make this region extremely difficult for sequencing and other analyses. It plays a conserved role in equal division of chromosomes into daughter cells in both mitosis and meiosis. However, centromere sequences show notable plasticity. In a dicentric chromosome, one of the centromeres can become inactivated with the underlying DNA unchanged. Furthermore, formerly inactive centromeres can regain activity under certain conditions. In addition, neocentromeres without centromeric repeats have been found in a wide spectrum of species. This evidence indicates that epigenetic mechanisms together with centromeric sequences are associated with centromere specification.**

Keywords centromere, centromere inactivation, centromere reactivation, nondisjunction, maize

Introduction

The centromere, a unique chromosomal module, generally observed as the primary constriction at metaphase of mitotic cells, chiefly consists of thousands of repeat sequences, which are arranged in tandem (Henikoff et al., 2001; Malik and Henikoff, 2009). It is the region for kinetochore organization, spindle microtubule attachment, and mediating chromosome movement toward opposite poles. Therefore, the centromere plays an essential role in accurate cell division. Although functionally conserved among all eukaryotes, centromere sequences are rapidly evolving, showing significant variability in sequences and length between species (Henikoff et al., 2001), and can even be different among chromosomes from the same organism.

Maize centromere DNA consists of two basic types of repetitive elements referred to as centromere repeat C (CentC) and centromeric retrotransposon of maize (CRM). CentC, usually 156 bp in length and arranged in tandem repeat arrays, is detected in all ten homologous chromosomes centromere regions but shows significant polymorphism in copy number (Ananiev et al., 1998). CRM represents several different related retotransposons that are located in the maize centromere region (Ananiev et al., 1998; Presting et al., 1998; Mroczek and Dawe, 2003; Nagaki et al., 2003). It shows a more uniform amount between chromosomes compared to CentC (Birchler and Han, 2009). These two components are intermingled and account for almost all parts of core centromeres in maize (Nagaki et al., 2003; Jin et al., 2004).

Supernumerary chromosomes, also referred to as B chromosomes, are detected in a large number of species including in some maize lines. They have distinctive structures and behave differently from A chromosomes (Jones and Rees, 1982). The maize B chromosome possesses a near terminal centromere containing all the repeat sequences found in normal maize chromosomes (Lamb et al., 2005). In addition, a unique repeat has been identified in the B chromosome centromere region called *Zea mays* B-specific (ZmBs), which enables one to distinguish B chromosomes from all others (Alfenito and Birchler, 1993). The B chromosome undergoes nondisjunction primarily during the second pollen mitosis (Roman, 1947). Additionally, sperm with B chromosomes exhibits preferential fertilization of the egg rather than the polar nuclei in the process of double fertilization (Roman, 1948; Carlson, 1969). Thus, B chromosomes can accumulate to a high number after a few

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generations. It is an excellent model for chromosome analysis because B chromosomes in low copy number do not affect the phenotype of the corn plant (Carlson and Phillips, 1986). This is the reason why the maize B chromosome is used to study centromere function and chromosome behavior (Han et al., 2006; Birchler and Han, 2009; Han et al., 2009) as well as to establish plant artificial chromosomes (engineered minichromosomes) (Yu et al., 2006; Yu et al., 2007).

In contrast to the plasticity of centromere sequences, the kinetochore proteins are conserved among species (Henikoff et al., 2001). The centromere contains a unique histone H3 variant: centromere histone H3 (CENH3, also called CENP-A in mammals) (Zhong et al., 2002), which serves as the most widely used hallmark for functional centromeres. CENH3 and its homologous proteins have been identified in centromere regions in all eukaryotes to date (Jiang et al., 2003). The CENH3 nucleosomes are interspersed with regular H3 nucleosome regions throughout centromeres (Blower et al., 2002). Unlike typical histone components, it integrates into chromatin via a replication-independent pathway (Shelby et al., 2000). In maize centromeres, CENH3 interacts specifically with both CentC and CRM (Zhong et al., 2002). Further studies discovered that CENH3 is almost always involved with CentC and CRM, but not all CentC or CRM sequences are associated with CENH3 (Jin et al., 2004). Both depletion and overexpression of CENH3 result in disorderly kinetochore assembly and location, suggesting CENH3 plays a fundamental role in kinetochore formation (Allshire and Karpen, 2008). CENH3 colocalizes with another basic kinetochore protein CENP-C (centromere protein C) (Dawe et al., 1999), which is recruited by CENH3 (Van Hooser et al., 2001; Ando et al., 2002). Both are useful molecular markers for centromere activity.

In this review, we will focus on recent studies of centromeres in maize, showing that both epigenetic and sequence components are involved in centromere specification, which fact is emerging from evidence for centromere inactivation, reactivation and neocentromere formation.

Inactivation

In maize, a distinctive chromosome, B-9-Dp9, is a translocated chromosome between the B chromosome and the short arm of chromosome 9, which contains a reverse duplication of the 9S arm (Zheng et al., 1999). Using maize varieties containing this chromosome together with two 9-B chromosomes, a series of new chromosomes are released in the process of meiosis. The structure of these can be detected by FISH (Fig. 1). During meiosis I, the duplicated B-9-Dp9 sister chromatids fold back and pair, allowing recombination to produce fused sister chromatids and an acentric fragment at anaphase I. In the subsequent meiosis II, two sister centromeres of the dicentric chromosome move to opposite poles producing a bridge, which will lead to chromosomal breakage. The broken chromosome replicates and fuses with itself. It undergoes nondisjunction at the second pollen mitosis, which produces sperm with two broken chromosomes. Then if this sperm is involved in fertilization, two broken chromosomes will initiate the chromosome type of breakage-fusion-bridge (BFB) cycle, first described by Barbara McClintock (McClintock, 1939, 1941). The BFB cycle proceeds until one of the dicentric centromeres becomes inactive or the broken chromosomes are stabled by the addition of telomeres.

A collection of minichromosomes has been generated via the BFB procedure in maize (Zheng et al., 1999; Han et al., 2007a). Some of them are dicentric chromosomes as revealed by their being two centromere regions identified by FISH. For example, minichromosome #3 is speculated to arise from breaks at the same location on the two chromatids followed by the small fragments containing the B centromere being joined together to produce two identical B centromeres in a minichromosome. Although the DNA sequence of both of the dicentric centromeres remains intact, a single CENH3 signal is detected using CENH3 antibodies, indicating that only one of the two centromeres has function, whereas the other one is silenced.

Another case in point, minichromosome #5, is generated from a near B centromere break, which formed from a truncated centromere fragment lacking knob repeats. This fragment is then healed by fusing to a larger fragment with a break further from the centromere. Consequently, minichromosome #5 is a dicentric with one relatively large and another comparatively small centromere. Surprisingly, the small centromere is labeled for CENH3, and also that domain shows attachment to spindle microtubules. This evidence

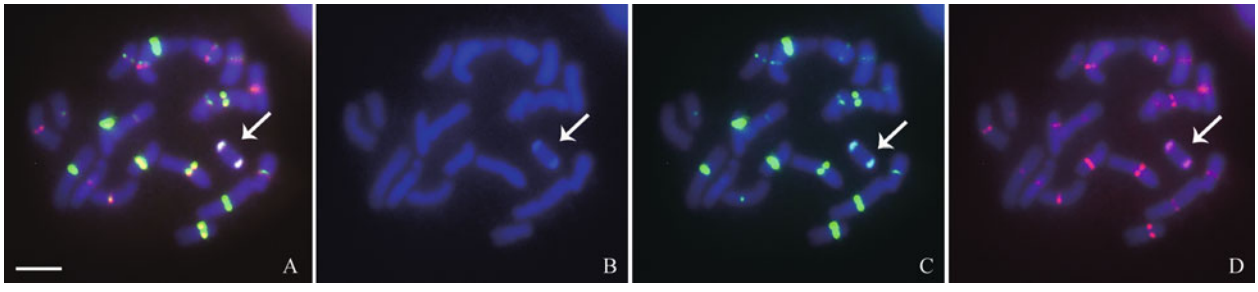

Figure 1 Newly formed dicentric chromosome from B-9-Dp9 with the two centromeres as large as normal B centromere. Arrow indicates the new dicentric chromosome. (A) Merged image. (B) DAPI. (C) CentC and (D) CRM. Scale bar = 10 μm

leads to an unexpected result that the small centromere is active whereas the large centromere is dormant.

Besides minichromosomes, an A-B dicentric translocation chromosome was also isolated during the BFB cycle of B-9-Dp9 (Han et al., 2006). This chromosome contains exchanges between the short arm of chromosome 9 and the B chromosome centromere; therefore, it was named 9-B inactive centromere-1 (9-Bic-1). The fragment of chromosome 9 containing the white deficiency (*wd*) gene is replaced by the B chromosome centromere, so plants with homozygous 9-Bic-1 chromosomes are albino and cannot survive beyond the seedling stage. Several lines of evidence reveal that the centromere derived from the B chromosome is inactive: In mitosis, the translocated B chromosome centromere shows no sister chromatid cohesion and separates at metaphase; it is not capable of functional kinetochore assembly or microtubule attachment; only the centromere of chromosome 9 is labeled for CENH3 and phosphorylation of the serine-10 of H3, which is also a molecular marker for active centromeres.

These paradigms illustrate centromere function is lost whereas centromeric core sequences remain unchanged, showing that an obviously epigenetic component is involved with centromere activity. Are there any other factors which determine centromere destiny in dicentric chromosomes? It is generally assumed that in a dicentric chromosome, the smaller centromere is more prone to shut off whereas the larger one remains active and behaves as a functional centromere (Stimpson and Sullivan, 2010). Recent research on wheat has identified a tricentric chromosome in which two smaller centromeres are preferentially inactivated (Zhang et al., 2010). However, with respect to minichromosome #5, instead of the large centromere, the small one has function, suggesting that centromere inactivation is a complex process that does not depend on centromere size alone.

The dicentric plants discussed above can be stably passed on for generations because they are functionally monocentric in spite of being structurally dicentric. However, some other stable derivatives remain functionally dicentric after formation (Page and Shaffer, 1998; Higgins et al., 2005; Stimpson et al., 2010; Stimpson and Sullivan, 2010). This might be explained by the intercentromeric distance that influences inactivation in the dicentric chromosome. When dicentric centromeres locate closely in space, the possibility for incorrect spindle attachment and chromosome segregation is reduced; thus the two centromeres are both functional and may behave like a single one (Sullivan and Willard, 1998). In contrast, dicentric chromosomes with distant centromeres are more likely to undergo inactivation (Stimpson and Sullivan, 2010). However, *de novo* engineered dicentric human chromosomes are exceptions to this rule (Stimpson et al., 2010), which further complicates the relationship between intercentric distance and centromere inactivation.

Intriguingly, inactive centromeres are organized in a special chromatin structure different from functional centromeres, giving the absence of constriction in metaphase during mitosis. Due to the complex features of centromere inactivation and configuration, whether plant centromere sequences can behave as a functional centromere in a certain artificial chromosome is not clear. Therefore, uncovering the mystery of the mechanisms involved with centromere activity is of significant importance for the construction of artificial chromosomes.

Reactivation

Centromere inactivation is only one aspect of centromere epigenetics. On the other hand, inactive centromeres can regain activity under certain conditions. The analysis of a special recombination derivative of Dicentric-15 (Dic-15) gives insight into centromere reactivation (Han et al., 2009). Dic-15 contains a large active B centromere at one terminus and a small inactive one at the other end. When the inactive centromere is released following recombination during meiosis I, it exhibits CENP-C labeling, which means that the small centromere has recovered function (Fig. 2). Intrachromosomal recombination in Dic-15 may occur because of its foldback structure, which may separate the

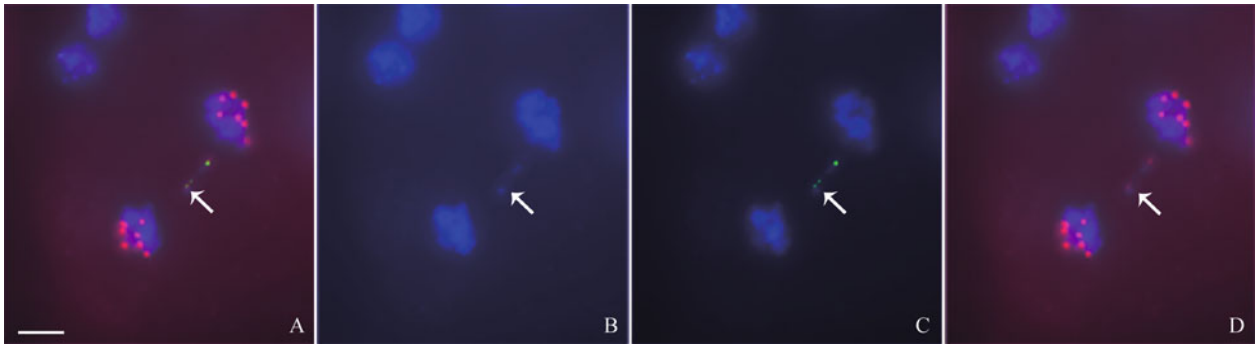

Figure 2 Reactivation of an inactive centromere. Immunolocalization analysis of CENP-C in meiosis in plants containing a dicentric chromosome with one centromere inactive. CENP-C signals are red; ZmBs is green; and DAPI-stained chromosomes are blue. Arrows indicate that the smaller centromere has gained CENP-C signals. (A) Merged image. (B) DAPI. (C) ZmBs. (D) CENP-C. Scale bar = 10 μm

small centromere from the large active one, and give rise to a chromosome merely containing two small centromeres. One of them can attract tubulin fibers and regain molecular properties of functional centromeres, for example, CENH3 and CENP-C are detected by their respective antibodies. The signal of H3 phosphorylated at Ser-10 is also detected, indicating that the former dormant small centromere becomes reactivated. So why does the reactived centromere occur at the formerly inactive site rather than at other sites along the arms? This finding raises the possibility that DNA sequences or topology is involved in centromere specification.

Nondisjunction

In maize lines with B chromosomes, the sister B chromatids proceed together to the same pole instead of disjoining at the second pollen mitosis (Roman, 1947). Two sections of the B chromosome are required for the function of nondisjunction. One is located in the proximal euchromatin (Lin, 1978) and another resides at the very distal tip of the long arm (Ward, 1973). It is necessary of these two sites and the centromere to co-exist in the same nucleus for nondisjunction, but it is not necessary for them to be present in the same chromosome (Roman, 1947; Lin, 1978).

Our previous work reveals that 9-Bic-1, together with 7-Bic-1, a translocation chromosome between an inactive B centromere and chromosome 7 (Fig. 3), retain the property of nondisjunction in the presence of normal B chromosomes (Han et al., 2007b), suggesting that the centromere adjacent knob and the B-repeat array are involved in nondisjunction (Han et al., 2007b). However, minichromosome #9 that is devoid of the knob also undergoes nondisjunction. Thus, the B-repeat array appears to play an important role in nondisjunction of the B chromosome.

Neocentromeres

Neocentromeres are ectopic centromeres arising at novel positions from canonical centromeres and having a unique, rather than repetitive DNA sequence, but somehow capable of organizing a functional kinetochore (Guerra et al., 2010). They have been identified in a wild spectrum of species including human. The first neocentromere reported in human was in chromosome 10 (Voullaire et al., 1993). An ectopic neocentromere was assembled over a unique sequence region in a truncated chromosome 10 lacking the normal centromeric satellite DNA. At present, 60 examples of human neocentromeres have been detected thoughout the genome (Amor and Choo, 2002). These findings suggest that human neocentromeres are able to be established regardless of the underlying DNA (Choo, 1997).

Classically defined plant neocentromeres show different characteristics from animal neocentromeres in morphology, inability to mediate sister chromatid cohesion, and rates of movement on the spindle (Dawe and Hiatt, 2004). Despite the existence of normal centromeres, neocentromeres can become established during meiosis elsewhere in the chromosome, but are unable to function fully as normal centromeres.

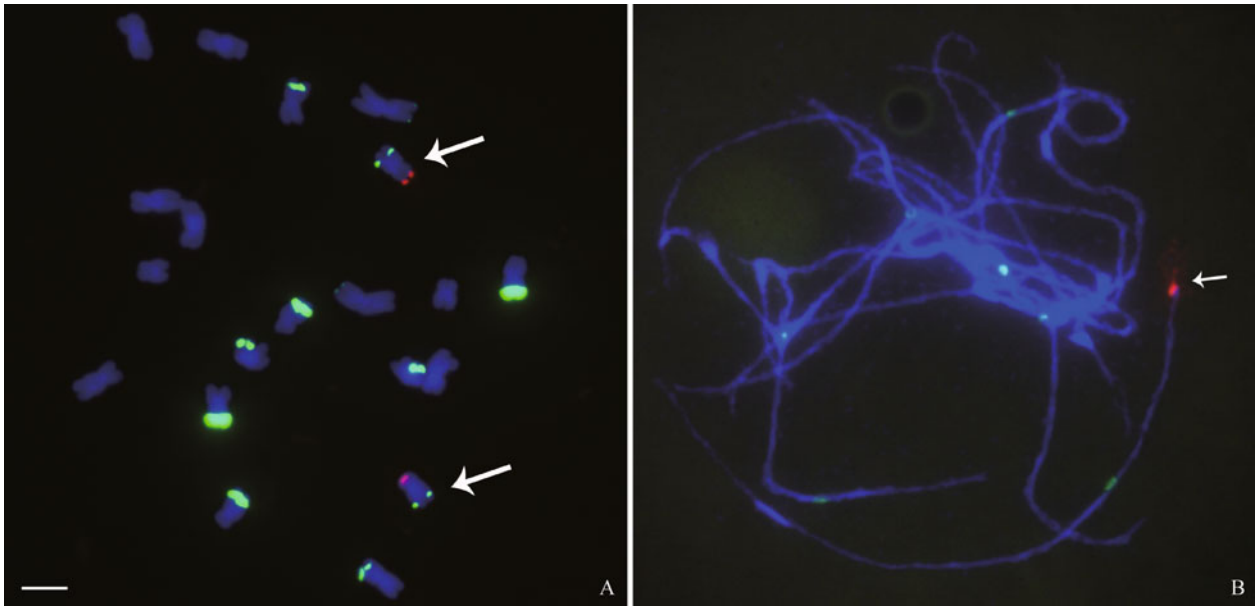

Figure 3 7-Bic-1 at mitotic metaphase and meiosis pachytene. The inactive B centromere from 9Bic-1 was translocated to chromosome arm 7S and named 7-Bic-1. (A) Mitotic metaphase. Knob is green and red is B repeats; arrows indicate the new translocation chromosomes containing an inactive B centromere. (B) Meiotic pachytene, CentC is green and red is B repeats, arrow indicates the inactive B centromere. Scale bar = 10 μm

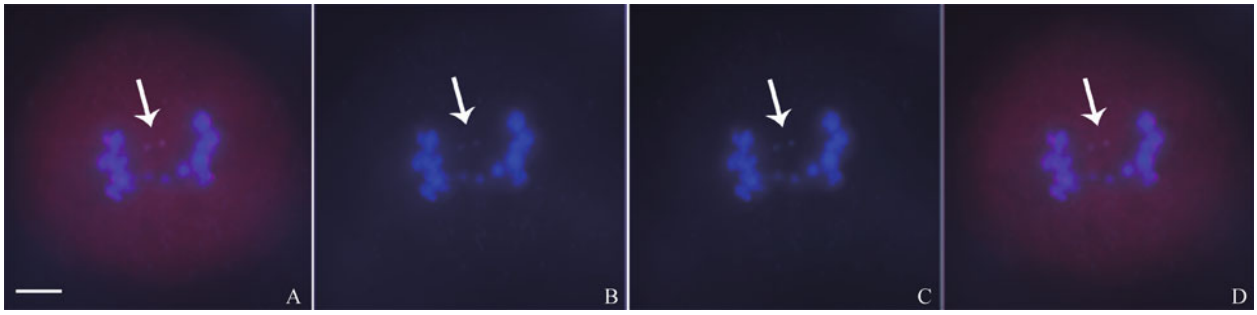

Figure 4 Immunolocalization analysis of SGO1 in a plant containing one minichromosome (#3) derived from the B chromosome (Arrows). The mini-B exhibits SGO1 signal at anaphase I. The DAPI-stained chromosomes are blue; maize SGO antibody (SGO1) is magenta; ZmBs is green. (A) Merged image. (B) DAPI. (C) ZmBs. (D) SGO1. Arrow indicates the sister chromatids separated at meiosis I. Scale bar = 10 μm

The first neocentric activity in maize was described for an abnormal type of chromosome 10 (Ab10). The subterminal region of this chromosome consists of repeat elements known as knobs, which can drive poleward movement of Ab10 in meiosis (Rhoades and Vilkomerson, 1942). A recent study described the formation of a novel centromere on a maize chromosome 3 fragment present in oat (Topp et al., 2009). The normal centromere region was lost due to chromosome breakage. Although unstable in size, it could transmit across generations in the oat genome background.

Another report in barley reveals that canonical centromere repeats are not required for kinetochore assembly (Nasuda et al., 2005). This evidence also supports the idea that epigenetic components rather than the underlying DNA sequence condition neocentromere formation.

Sister chromatid cohesion

Some minichromosomes show different patterns in chromosome cohesion from normal chromosomes during meiosis. Referring to normal chromosomes, sister chromatids hold together in anaphase I and then separate in anaphase II. In contrast with normal chromosomes, in some minichromosomes, especially those with small centromeres, the sister chromatids separate precociously in anaphase I. And in the subsequent anaphase II, minichromosomes progress to one of the two poles (Han et al., 2007a).

The protein Shugoshin (SGO) plays a key role in sister chromotid cohesion. This protein preserves centromeric cohesion from cleavage by separase to ensure monopolar orientation of sister chromatids in meiosis I (Watanabe, 2005). It has been noted that no SGO1 signal is detected if centromere cohesion is lost in anaphase I in a meiotic mutant defective in this process (Hamant et al., 2005). However, by immunocytochemistry and FISH, we find that SGO1 remains despite sister chromatids separation in anaphase I for minichromosome #3 (Fig. 4). Thus, for such small chromosomes, SGO appears not to be sufficient for sister cohesion during the first meiotic division. Other factors such as pericentromeric heterochromatin might also be involved in this process.

Conclusions

In most eukaryotes, centromere DNA is similarly composed of repetitive satellites, which might suggest that centromere location and function are determined by centromeric repeat sequences. However, the results described above indicate a much more complex mechanism. On the one hand, intact centromeres becoming inactive suggests the centromere sequence itself does not guarantee centromere speciation and neocentromere formation in unique sequence regions demonstrates centromeres can be formed on non-repeat sequences. Both lines of evidence indicate an epigenetic component, rather than DNA sequence, in centromere determination. On the other hand, the case of centromere reactivation occurs at the formerly inactive centromere site, revealing that sequence or structure of the underlying DNA can also contribute to centromere specification. How the centromere activity is established and maintained; why sequences with centromeric repeats can become dormant; what factors are associated with centromere activation and reactivation are still open questions.

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References

Alfenito M R, Birchler J A (1993). Molecular characterization of a maize B chromosome centric sequence. Genetics, 135(2): 589–597

Allshire R C, Karpen G H (2008). Epigenetic regulation of centromeric chromatin: old dogs, new tricks? Nat Rev Genet, 9(12): 923–937

Amor D J, Choo K H A (2002). Neocentromeres: role in human disease, evolution, and centromere study. Am J Hum Genet, 71(4): 695–714

Ananiev E V, Phillips R L, Rines H W (1998). Chromosome-specific molecular organization of maize (*Zea mays* L.) centromeric regions. Proc Natl Acad Sci U S A, 95(22): 13073–13078

Ando S, Yang H, Nozaki N, Okazaki T, Yoda K (2002). CENP-A, -B, and -C chromatin complex that contains the I-type alpha-satellite array constitutes the prekinetochore in HeLa cells. Mol Cell Biol, 22 (7): 2229–2241

Birchler J A, Han F P (2009). Maize centromeres: structure, function, epigenetics. Annu Rev Genet, 43(1): 287–303

Blower M D, Sullivan B A, Karpen G H (2002). Conserved organization of centromeric chromatin in flies and humans. Dev Cell, 2(3): 319–330

Carlson W R (1969). Factors affecting preferential fertilization in maize. Genetics, 62(3): 543–554

Carlson W R, Phillips R (1986). The B-chromosome of maize. Crit Rev Plant Sci, 3(3): 201–226

Choo K H A (1997). Centromere DNA dynamics: latent centromeres and neocentromere formation. Am J Hum Genet, 61(6): 1225–1233

Dawe R K, Hiatt E N (2004). Plant neocentromeres: fast, focused, and driven. Chromosome Res, 12(6): 655–669

Dawe R K, Reed L M, Yu H G, Muszynski M G, Hiatt E N (1999). A maize homolog of mammalian CENPC is a constitutive component of the inner kinetochore. Plant Cell, 11(7): 1227–1238

Guerra M, Cabral G, Cuacos M, González-García M, González-Sánchez M, Vega J, Puertas M J (2010). Neocentrics and holokinetics (holocentrics): chromosomes out of the centromeric rules. Cytogenet Genome Res, 129(1-3): 82–96

Hamant O, Golubovskaya I, Meeley R, Fiume E, Timofejeva L, Schleiffer A, Nasmyth K, Cande W Z (2005). A REC8-dependent plant Shugoshin is required for maintenance of centromeric cohesion during meiosis and has no mitotic functions. Curr Biol, 15(10): 948–954

Han F P, Gao Z, Birchler J A (2009). Reactivation of an inactive centromere reveals epigenetic and structural components for centromere specification in maize. Plant Cell, 21(7): 1929–1939

Han F P, Gao Z, Yu W C, Birchler J A (2007a). Minichromosome analysis of chromosome pairing, disjunction, and sister chromatid cohesion in maize. Plant Cell, 19(12): 3853–3863

Han F P, Lamb J C, Birchler J A (2006). High frequency of centromere inactivation resulting in stable dicentric chromosomes of maize. Proc Natl Acad Sci U S A, 103(9): 3238–3243

Han F P, Lamb J C, Yu W C, Gao Z, Birchler J A (2007b). Centromere function and nondisjunction are independent components of the maize B chromosome accumulation mechanism. Plant Cell, 19(2): 524–533

Henikoff S, Ahmad K, Malik H S (2001). The centromere paradox: stable inheritance with rapidly evolving DNA. Science, 293(5532): 1098–1102

Higgins A W, Gustashaw K M, Willard H F (2005). Engineered human dicentric chromosomes show centromere plasticity. Chromosome Res, 13(8): 745–762

Jiang J M, Birchler J A, Parrott W A, Dawe R K (2003). A molecular view of plant centromeres. Trends Plant Sci, 8(12): 570–575

Jin W W, Melo J R, Nagaki K, Talbert P B, Henikoff S, Dawe R K, Jiang J M (2004). Maize centromeres: organization and functional adaptation in the genetic background of oat. Plant Cell, 16(3): 571–581

Jones R N, Rees H (1982). B chromosomes. London, Academic Press

Lamb J C, Kato A, Birchler J A (2005). Sequences associated with A chromosome centromeres are present throughout the maize B chromosome. Chromosoma, 113(7): 337–349

Lin B Y (1978). Regional control of nondisjunction of the B chromosome in maize. Genetics, 90(3): 613–627

Malik H S, Henikoff S (2009). Major evolutionary transitions in centromere complexity. Cell, 138(6): 1067–1082

McClintock B (1939). The behavior in successive nuclear divisions of a chromosome broken at meiosis. Proc Natl Acad Sci U S A, 25(8): 405–416

McClintock B (1941). The stability of broken ends of chromosomes in *Zea mays*. Genetics, 26(2): 234–282

Mroczek R J, Dawe R K (2003). Distribution of retroelements in centromeres and neocentromeres of maize. Genetics, 165(2): 809–819

Nagaki K, Song J Q, Stupar R M, Parokonny A S, Yuan Q P, Ouyang S, Liu J, Hsiao J, Jones K M, Dawe R K, Buell C R, Jiang J M (2003). Molecular and cytological analyses of large tracks of centromeric DNA reveal the structure and evolutionary dynamics of maize centromeres. Genetics, 163(2): 759–770

Nasuda S, Hudakova S, Schubert I, Houben A, Endo T R (2005). Stable barley chromosomes without centromeric repeats. Proc Natl Acad Sci U S A, 102(28): 9842–9847

Page S L, Shaffer L G (1998). Chromosome stability is maintained by short intercentromeric distance in functionally dicentric human Robertsonian translocations. Chromosome Res, 6(2): 115–122

Presting G G, Malysheva L, Fuchs J, Schubert I Z (1998). A *Ty3/gypsy* retrotransposon-like sequence localizes to the centromeric regions of cereal chromosomes. Plant J, 16(6): 721–728

Rhoades M M, Vilkomerson H (1942). On the anaphase movement of chromosomes. Proc Natl Acad Sci U S A, 28(10): 433–436

Roman H (1947). Mitotic nondisjunction in the case of interchanges involving the B-type chromosome in maize. Genetics, 32: 391–409

Roman H (1948). Directed fertilization in maize. Proc Natl Acad Sci USA, 34(2): 36–42

Shelby R D, Monier K, Sullivan K F (2000). Chromatin assembly at kinetochores is uncoupled from DNA replication. J Cell Biol, 151(5): 1113–1118

Stimpson K M, Song I Y, Jauch A, Holtgreve-Grez H, Hayden K E, Bridger J M, Sullivan B A, Copenhaver G P (2010). Telomere disruption results in non-random formation of de novo dicentric chromosomes involving acrocentric human chromosomes. PLoS Genet, 6(8): e1001061

Stimpson K M, Sullivan B A (2010). Epigenomics of centromere assembly and function. Curr Opin Cell Biol, 22(6): 1–9

Sullivan B A, Willard H F (1998). Stable dicentric X chromosomes with two functional centromeres. Nat Genet, 20(3): 227–228

Topp C N, Okagaki R J, Melo J R, Kynast R G, Phillips R L, Dawe R K (2009). Identification of a maize neocentromere in an oat-maize addition line. Cytogenet Genome Res, 124(3-4): 228–238

Van Hooser A A, Ouspenski I I, Gregson H C, Starr D A, Yen T J, Goldberg M L, Yokomori K, Earnshaw W C, Sullivan K F, Brinkley B R (2001). Specification of kinetochore-forming chromatin by the histone H3 variant CENP-A. J Cell Sci, 114(Pt 19): 3529–3542

Voullaire L E, Slater H R, Petrovic V, Choo K H A (1993). A functional marker centromere with no detectable alpha-satellite, satellite III, or CENP-B protein: activation of a latent centromere? Am J Hum Genet, 52(6): 1153–1163

Ward E J (1973). Nondisjunction: localization of the controlling site in the maize B chromosome. Genetics, 73(3): 387–391

Watanabe Y (2005). Shugoshin: guardian spirit at the centromere. Curr Opin Cell Biol, 17(6): 590–595

Yu W C, Han F P, Gao Z, Vega J M, Birchler J A (2007). Construction and behavior of engineered minichromosomes in maize. Proc Natl Acad Sci U S A, 104(21): 8924–8929

Yu W C, Lamb J C, Han F P, Birchler J A (2006). Telomere-mediated chromosomal truncation in maize. Proc Natl Acad Sci U S A, 103 (46): 17331–17336

Zhang W L, Friebe B, Gill B S, Jiang J M (2010). Centromere inactivation and epigenetic modifications of a plant chromosome with three functional centromeres. Chromosoma, 119(5): 553–563

Zheng Y Z, Roseman R R, Carlson W R (1999). Time course study of the chromosome-type breakage-fusion-bridge cycle in maize. Genetics, 153(3): 1435–1444

Zhong C X, Marshall J B, Topp C, Mroczek R, Kato A, Nagaki K, Birchler J A, Jiang J M, Dawe R K (2002). Centromeric retroelements and satellites interact with maize kinetochore protein CENH3. Plant Cell, 14(11): 2825–2836