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# The meiotic drive system on maize abnormal chromosome 10 contains few essential genes

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**Abstract**

In maize, a distal portion of abnormal chromosome 10 (Ab10) causes the meiotic drive of itself as well as many unlinked heterochromatic regions known as knobs. The Ab10 drive system, which encodes *trans*- as well as *cis*-acting components, occupies a large region of chromosome 10L equivalent to ~3% of the genome. Here we describe five new structural mutations of Ab10 (five deletions and a duplication) that arose from a screen for meiotic drive mutants. The high frequency of breakage events, detected both genetically and cytologically, suggest that the chromosome may be especially unstable. Very large deletions within the drive system are female-transmissible and plants homozygous for deficiencies lacking much of this interval can be grown to maturity. The data suggest that few genes required for normal growth and development lie within the portion of Ab10 responsible for meiotic drive. These and other published data suggest that meiotic drive systems tend to evolve in gene-sparse or otherwise information-poor regions of the genome where they are less likely to negatively affect individual fitness.

## Introduction

The phenomenon of meiotic drive in maize is associated with the presence of an unusual form of chromosome 10. When Abnormal chromosome 10 (Ab10) is present in the genome, it as well as other knobbed homologues are transmitted in female testcrosses at levels approaching 75% (as opposed to the expected 50%; Rhoades & Vilkomerson, 1942; Dawe & Cande, 1996). The Ab10 chromosome differs from the normal 10 (N10) chromosome cytologically as well as phenotypically. The distal long arm of Ab10 is extended by novel chromatin that is approximately equal to the length of the short arm of the 10th chromosome, or about 3% of the maize genome. There are four distinct regions within the novel chromatin: (1) a 'differential segment' with three conspicuous chromomeres just distal to the Ab10/N10 transition point; (2) a long region of 'central euchromatin'; (3) a large knob; and (4) a euchromatic distal tip (Figure 1). The central euchromatin contains a ~28 map unit region including three loci, *L13*, *O7*, *W2*, that is translocated and inverted from the distal end of N10. Also within the central euchromatin is a fourth locus, striated (*Sr2*) that is distal to the inverted region both on N10 and Ab10 (Rhoades & Dempsey, 1985). Recombination levels across the *L13-O7-W2-Sr2* interval appear to be typical of euchromatin in both N10 and Ab10 (although recombination between the chromosomes is rarely if ever observed due to the inversion; Rhoades & Dempsey, 1985).

The large knob and three chromomeres of Ab10 are composed primarily of tandem repeat arrays. The large knob is comprised primarily of long arrays of a 180 bp repeat sequence, while the three chromomeres predominantly contain a ~350 bp repeat sequence, designated TR-1 (Peacock et al., 1981; Ananiev, Phillips & Rines, 1998; Hiatt, Kentner & Dawe, 2002). Knobs composed of one or both repeats are found throughout the genome in at least 21 primarily interstitial positions on chromosome arms. In response to genetic information on Ab10, most if not all knobs demonstrate both meiotic drive and pronounced neocentromere activity (Rhoades & Vilkomerson,

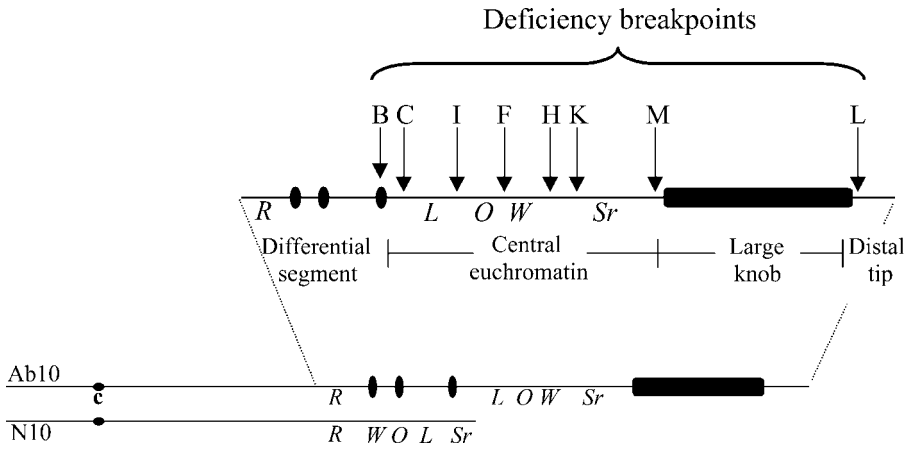



*Figure 1.* Schematic diagram illustrating the cytological difference between the Ab10 and N10 chromosomes. The centromere is indicated with a 'c'. Four cytological regions of the novel chromatin containing the drive system are indicated as the differential segment, central euchromatin, large knob, and distal tip. The positions of three chromomeres are indicated within the differential segment. Letters below the lines (*R*, *W*, *O*, *L* and *Sr*) indicate the relative location of mapped genes on N10 and Ab10. On the Ab10 enlargement the locations of the deficiency breakpoints are indicated (the breakpoints for Df(L), Df(L-2) and Df(L-3) are all represented by Df(L)).

1942; Longley, 1945; Rhoades & Dempsey, 1966). Neocentromeres are cytologically visible structures formed when knobs interact tangentially with microtubules and are transported toward spindle poles in advance of the true centromeres (Yu et al., 1997). Ab10 also encodes a factor(s) that increases recombination between knobs and centromeres (Rhoades & Dempsey, 1966; Robertson, 1968; Nel, 1973). Given these observations, Rhoades proposed a mechanistic model to explain meiotic drive (Rhoades, 1952). Following recombination between knobs and centromeres, neocentromere activity pulls knobs towards the upper and lower (basal) megaspores of the female tetrad. Only the basal megaspore develops to form an egg, so knobs and their linked genes are preferentially recovered through the female. Ab10 does not demonstrate meiotic drive through the male, presumably because all four products of male meiosis become gametes. Knobs at locations on other chromosomes most likely arose subsequent to the drive system to take advantage of the trans effects of the Ab10 meiotic drive system (Buckler et al., 1999).

The cytological locations of the various functions encoded on Ab10 have been roughly identified using a series of terminal deficiencies. Emmerling and Miles generated Ab10 deficiencies using X-rays, whereas Rhoades and Dempsey used a maize strain with unusually high levels of chromosome breakage (Emmerling, 1959; Miles, 1970; Rhoades & Dempsey, 1985). All five of the deficiency strains identified by Rhoades and Dempsey are maintained in this laboratory. Each deficiency has a different breakpoint proximal to the *Sr2* locus within the central euchromatin (Figure 1). One deficiency, Df(H), retains neocentromere activity but lacks the recombination effect, demonstrating that the two functions are genetically separable (Rhoades & Dempsey, 1986, 1989). More recently we demonstrated that the neocentromere activity retained in the Rhoades and Dempsey deficiencies is limited to knobs containing the TR-1 repeat, and that the genes responsible for TR-1-mediated neocentromere activity lie proximal to the Df(I) breakpoint (Hiatt, Kentner & Dawe, 2002). Neocentromere activity of the knobs containing the 180 bp repeat is at least partially conferred by genetic information distal to the Df(K) breakpoint (Hiatt, Kentner & Dawe, 2002).

Most meiotic drive systems carry inversions that help to maintain the critical *trans*-acting functions in tight linkage with the *cis*-acting DNA. On Ab10 there is an inversion and a translocation that links the neocentromere-promoting loci with the knobs/neocentromeres themselves (Rhoades & Dempsey, 1985; Hiatt, Kentner & Dawe, 2002). While this has clear benefits for the drive system, regions of the genome containing 'normal' genes, that is, those not directly involved in mediating meiotic drive, can be trapped in the polymorphism and accumulate deleterious alleles (Lyttle, 1991). In well-studied systems such as *Segregation Distorter* in *Drosophila* and the *t*-haplotype in mice, some (but not all) accessions of the drive chromosomes are homozygous inviable as a result of deleterious or lethal alleles (Silver, 1985;

Lyttle, 1991). Here we describe the genetics of six new structural mutations of Ab10. We argue that meiotic drive systems tend to evolve in information-poor regions of the genome that will have minimal impact on the fitness of the organism.

## Materials and methods

### *Screen for suppressor of meiotic drive mutants*

An open pollinated field was used to screen for mutants that suppress meiotic drive. The cross used is diagrammed below:

| Female parent | | Male parent |
|---|---|---|
| *r*-Ab10/*R*-N10 with *Mu* activity | × | *Rst*-N10/*Rst*-N10 |

The *R* locus is linked to Ab10 by approximately 2 cM and has several distinguishable alleles (*R* – colored kernel; *r* – colorless kernel; *Rst* – colorless kernel with colored spots). The female parent contained active *Robertson Mutator (Mu)* elements (Walbot, 1992) and was heterozygous for Ab10. The male parent was homozygous for *Rst*-N10. All females were detasseled by hand. A total of 8230 ears were screened for mutants in meiotic drive (i.e., close to 50% segregation) in the years 1995, 1996, and 1999. Segregation ratios for each mutant were determined by testcrosses to homozygous N10 male parents over multiple generations. The location of the Df(M) breakpoint was refined by crossing homozygous Df(M) plants to homozygous *sr2* plants and observing the resulting progeny (*sr2* was obtained from the Maize Genetics Cooperation Stock Center).

### *Cytological confirmation*

Ab10 chromosomes from all confirmed heritable mutations were examined cytologically for observable chromosome aberrations. Anthers were collected and fixed, and meiocytes were prepared as described previously (Dawe & Cande, 1996; Hiatt, Kentner & Dawe, 2002). To determine whether there were interstitial deletions or extra chromatin, measurements were taken using DeltaVision software (Applied Precision, Inc.) on computationally straightened chromosomes. Distances were measured for each of four distinct regions of Ab10 (Table 1). The region between the first two chromomeres is too small to obtain accurate measurements. The non-parametric Mann–Whitney U-test was used to compare the section lengths of each mutant chromosome with the progenitor Ab10 chromosome.

*In situ* hybridization (Hiatt, Kentner & Dawe, 2002) was used to identify knob repeats on the Ab10 chromosome. The TR-1 repeat is a major component of the three chromomeres on Ab10, and the 180 bp repeat is the predominant repeat in the large Ab10 knob (Hiatt, Kentner & Dawe, 2002). For two putative mutants, Df(L-2) and Df(L-3), *in situ* hybridization with a telomere specific probe was also performed (Bass et al., 1997). The protocol used was the same as that used for the knob repeats.

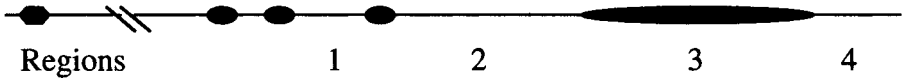
## Results

The experiments described here were designed to identify mutations in genes required for meiotic drive. Towards this end a strain containing active transposable elements in the *Robertson's mutator* (*Mu*) family was crossed to a strain carrying Ab10, and the resulting Ab10/N10 heterozygotes were test crossed to screen for heritable defects in meiotic drive. Alleles of the *R* gene, which are linked to Ab10 by 2 cM (Rhoades & Vilkomerson, 1942), were used to score for meiotic drive. In previous work, a sample of 3110 ears from this cross yielded a cytologically undetectable mutation (*Ab10-smd1*) that reduces both neocentromere activity as well as meiotic drive of the Ab10 chromosome (Dawe & Cande, 1996). A small deficiency of the distal tip of Ab10, called Df(L), was also recovered in this original screen (though it was not described in Dawe & Cande, 1996). We have now screened an additional 8230 ears for defects in meiotic drive and have confirmed one more cytologically undetectable mutation and an additional five structural mutations of Ab10 that abolish meiotic drive.

### *Cytological analysis: five Ab10 deficiencies and a duplication*

Three-dimensional light microscopy and fluorescent *in situ* hybridization were used to identify any cytologically visible defects in the various mutant strains. By analyzing chromosomes in the pachytene stage of meiotic prophase we determined that five of the loss-of-drive mutants, Df(B), Df(L) Df(L-2), Df(L-3) and Df(M), were terminal deficiencies of Ab10. The new deficiency chromosomes were given designations in keeping with the nomenclature used for previously described Ab10 deficiency chromosomes (Rhoades &

*Table 1.* Length measurements of chromosomal regions taken from progenitor Ab10 and various derivative forms of Ab10




| Genotype | Sample number | Regions of Ab10 chromosome | | | |
|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 4 |
| Progenitor Ab10 | 9 | 1.9[a] (0.3) | 4.4 (0.9) | 6.0 (1.4) | 2.3 (0.5) |
| Dp(A) | 9 | 2.0 (0.3) | 4.9 (1.0) | 7.4[b] (1.2) | 8.0[b] (2.3) |
| Df(L-2) | 7 | 1.8 (0.5) | 3.6 (1.0) | 4.9 (0.7) | – |
| Df(L-3) | 7 | 2.1 (0.2) | 4.7 (0.6) | 5.7 (0.4) | – |
| Df(M) | 6 | 1.9 (0.2) | 4.2 (1.4) | – | – |
| Df(B) | 6 | 1.7 (0.2) | – | – | – |

[a] Mean length of the region, all measurements in micrometers. Standard deviation indicated in parentheses.
[b] Indicates length values which are significantly different from the progenitor Ab10 length measurement.

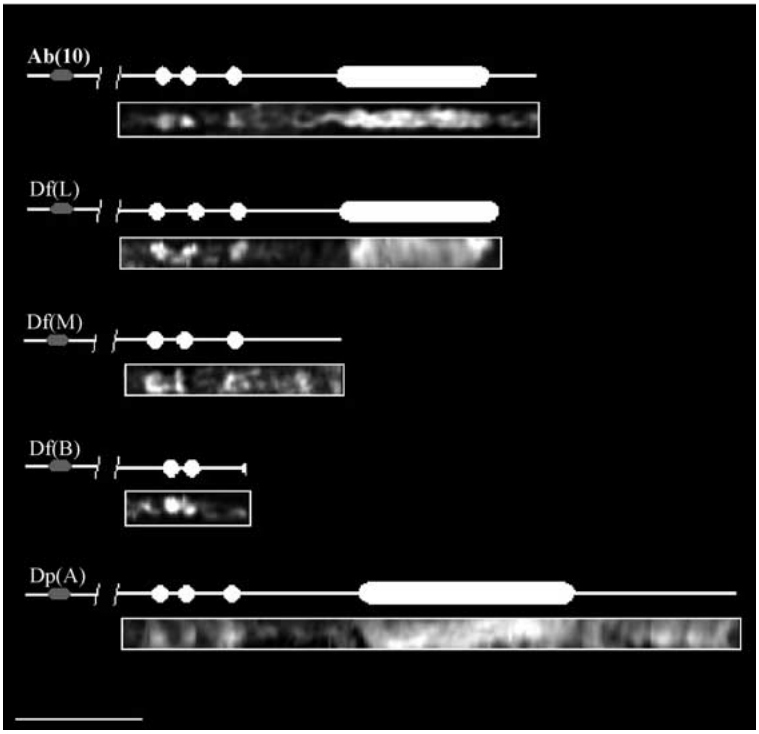



*Figure 2.* Computationally straightened distal ends of Ab10 and its derivatives. Each chromosome is represented by an actual image and a schematic diagram above. The diagrams show the centromere as a small gray oval, the chromomeres as small white circles and the large knob as a large white oval. Df(L), Df(L-2) and Df(L-3), which have a similar breakpoints, are all represented by a Df(L-3) chromosome. The bar represents 5 μm.

Dempsey, 1985). Df(L), Df(L-2) and Df(L-3) appear to have similar breakpoints just distal to the large knob (Figures 1 and 2). Since the tip can sometimes be difficult to identify, we used *in situ* hybridization with a telomere probe to confirm the deficiency. Telomeres are quickly added to the ends of broken chromosomes in other plant species (Werner et al., 1992; Tsujimoto, Yamada & Sasakuma, 1997). Both Df(L-2) and Df(L-3) showed telomere sequence hybridization at the distal end of the large knob, confirming the loss of the euchromatic distal tip (data not shown). There do not appear to be any other significant deletions or rearrangements on these chromosomes (Table 1).

Unlike the Df(L) chromosomes, Df(M) and Df(B) are missing the large knob (Figures 1 and 2). The

*Table 2.* Segregation ratios of Ab10 and its derivatives

| | Progenitor Ab10 | Df(L) | Df(L-2) | Df(L-3) | Df(M) | Df(B) | Dp(A) |
|---|---|---|---|---|---|---|---|
| Female segregation ratios[a] | 77.0%[b] (7.4%)[c], 8407(19)[d] | 47.1%[b] (4.5%), 3418(13) | 43.7%[b] (2.3%), 4497(10) | 42.2%[b] (4.3%), 6535(15) | 47.4%[b] (2.5%), 5626(14) | 42.0%[b] (1.7%), 853(4) | 49.6% (4.6%), 947(4) |
| Male segregation ratios[a] | 49.5% (4.7%), 2077(7) | 52.4% (4.9%), 991(5) | NA[e] | NA | Normal[f] | 0% | Normal[g] |

[a] Plants heterozygous for N10 and Ab10 (or its derivatives) were testcrossed to N10 homozygotes, unless otherwise noted.
[b] Female segregation ratios that were significantly less than the Mendelian expectation of 50%, as determined by replicated goodness of fit tests (Sokal & Rohlf, 1969). The mutants noted showed significant ($P < 0.05$) pooled and total chi-square values. For Df(L2), Df(M), and Df(B), heterogeneity tests indicated no significant differences among the crosses made with the same mutant. The Df(L), Df(L-3) and Ab10 data gave significant ($P < 0.05$) heterogeneity chi-square values, suggesting that while the data showed a significant trend away from the Mendelian expectation, there was substantial variation from ear to ear.
[c] Standard deviation.
[d] Total number of kernels. Number in parenthesis indicates the number of families (ears), which contributed to the total kernel count.
[e] Data not available.
[f] Homozygous r-Df(M) was recovered in self crosses of r-Df(M)/Rnj- N10 plants in 24.2% of the progeny (SD = 1.4%; $n = 2107$ seeds from five ears) which is not significantly different from a Mendelian expectation of 25% ($P > 0.05$ for pooled, total and heterogeneity chi-square values).
[g] Homo zygous r-Dp(A) was recovered in self crosses of r-Dp(A)/Rnj- N10 plants in 21.4% of the progeny (SD = 3.8%; $n = 563$ seeds from three ears), which is not significantly different from a Mendelian expectation of 25% ($P > 0.05$ for pooled, total and heterogeneity chi-square values).

central euchromatin of Df(M) (Region 2) was not significantly different from the same region in progenitor Ab10, suggesting that the breakpoint is very close to the proximal edge of the knob (Table 1). Df(M) also complements a standard recessive allele of *sr2* (not shown), consistent with the interpretation that most of the proximal euchromatin is preserved in Df(M). On the other hand, Df(B) has a breakpoint within the third chromomere, and therefore lacks the entire central euchromatin, large knob, and distal tip. Df(B) has the most severe deficiency of all known Ab10 derivatives (Miles, 1970; Rhoades & Dempsey, 1985). The distal euchromatic tip of a fifth mutant, Dp(A), is two–three-fold longer than the tip on the progenitor Ab10 chromosome (Figure 2, Table 1). Because there are no known markers for the distal tip, we were unable to determine if the excess chromatin is a duplication from Ab10 or a translocation from another region of the genome. The large knob (Region 3) was also significantly longer on Dp(A) than on progenitor Ab10 (Table 1). The longer knob seen in Dp(A) chromosomes at pachytene may be due to additional DNA or to an effect of the extended tip on knob condensation.

*Genetic analysis: high transmission and viability despite large losses of chromatin*

Despite large losses of chromatin, Df(L) and Df(M) are homozygous viable with no obvious phenotypes or reduced vigor in a greenhouse or field environment. Previous work demonstrated that Df(H) and Df(K) are also homozygous viable (Rhoades & Dempsey, 1985). The much more severe deficiency, Df(F) was also reported as germinating to form plants although they were albino (due to the absence of *white2*) and died at an early stage (Rhoades & Dempsey, 1985). We have carried out a small experiment to verify the phenotype of Df(F) homozygotes. Twenty-three Df(F)/Df(F) seeds were planted along with 10 controls seeds that were homozygous for a standard recessive allele of *white2* (*w2*). All 10 *w2* control seeds germinated normally to form white plants. Of the 23 Df(F)/Df(F) plants, 14 did not germinate, eight germinated to form roots but no shoots, and a single plant germinated to form roots and a stunted white shoot.

Segregation data for the six structural mutations along with the corresponding control crosses are shown in Table 2. Progenitor Ab10 shows meiotic drive, while each of the derivatives show a complete loss of drive (Table 2). All of the deficiencies are transmitted at close to Mendelian frequencies through the female, though at levels between 42 and 47% instead of the expected 50%. These data indicate that the tip of Ab10 contains genetic information required for meiotic drive and/or other genes that have quantitative effects on normal gametogenesis and fertilization. In general, the male gametophytes of flowering plants are more sensitive to chromosomal deficiencies than the female gametophytes (e.g., Carlson, 1988). This is true for the Ab10 deficiencies as well. Male transmission was reduced for chromosomes shorter than Df(M) and was completely absent for Df(B), Df(C) and Df(I) (Table 3).

*Ab10 may be inherently unstable: other cytological evidence from a non-*Mu *background*

In a strain heterozygous for Ab10 and two other small knobs (Hiatt, Kentner & Dawe, 2002) we unexpectedly observed three instances where the large knob of Ab10 had broken and separated from the main body of the 10th chromosome (Figure 3). Although we could not determine where the breakpoints were on the fragments, their overall size and the fact that they did not hybridize the TR-1 probe (which labels the three chromomeres of Ab10), is consistent with breakage within the central euchromatin. It is unlikely that the breakage events were induced by *Robertson's*

*Table 3.* Male and female segregation ratios for all available Ab10 deficiencies

| Ab10 deficiency[a] | Male segregation (%) | Female segregation (%) |
|---|---|---|
| Df(B)[b] | 0 | 42 |
| Df(C)[c] | 0 | Variable reduction |
| Df(I)[c] | 0 | Variable reduction |
| Df(F)[c] | 26 | 50 |
| Df(H)[c] | 35 | 50 |
| Df(K)[c] | 35 | 50 |
| Df(M)[b] | 50 | 47 |
| Df(L)[b] | 50 | 44 |
| Df(L-2)[b] | NA[d] | 44 |
| Df(L-3)[b] | NA[d] | 44 |

[a] Plants heterozygous for N10 and the indicated Ab10 deficiency were test crossed to N10 homozygotes. The deficiencies are listed from the most severe deletion, Df(B) to the least, Df(L).
[b] Data taken from Table 2.
[c] Data modified from Rhoades & Dempsey (1985), Table 1.
[e] Data not available.

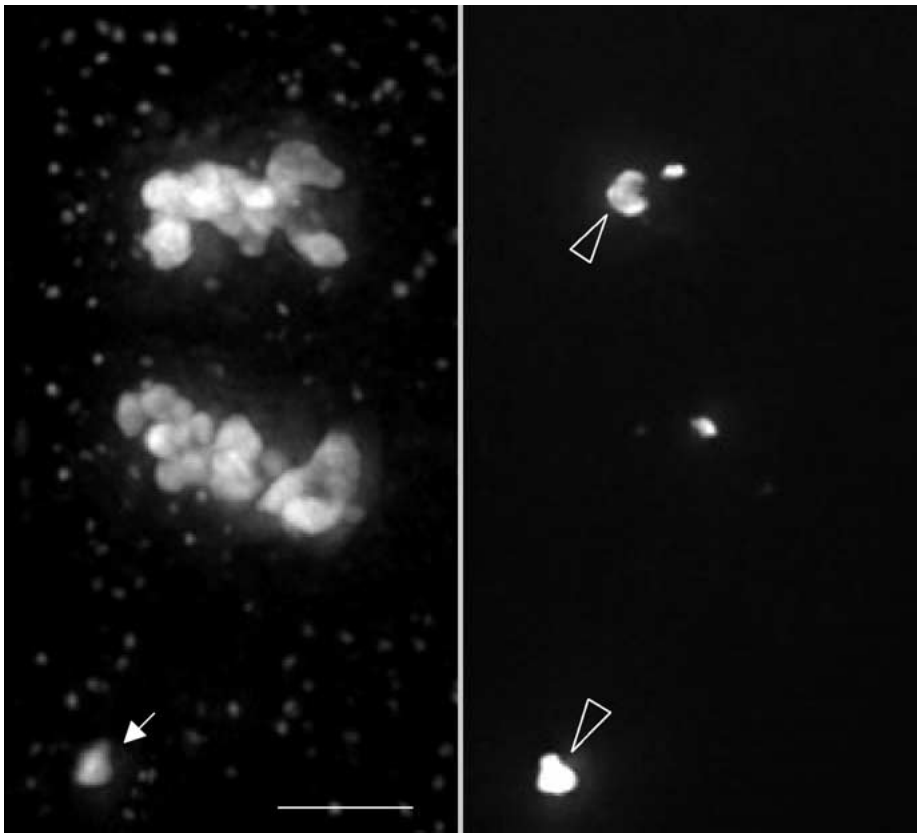


*Figure 3.* Cytological evidence that Ab10 is inherently unstable. The images are from a partial projection of an anaphase II cell. On the left are the chromosomes, as shown by DAPI staining. An acentric chromosomal fragment is observed in the lower left corner (arrow). The image on the right shows labeling for the 180 bp knob repeat. The acentric fragment contains the large knob signal from the Ab10 chromosome (empty arrowhead). This cell was also hybridized with a TR1 repeat probe that identifies the Ab10 chromomeres; no TR1 staining was evident on the acentric fragment (not shown). The data suggest that the Ab10 chromosome was broken within the central euchromatin. Two other cells containing acentric fragments with the same staining pattern were also found. The bar represents 5 μm.

*mutator*, since to our knowledge *Mu* is not active in the Knobless Wilbur's Flint background used.

## Discussion

We have recovered a surprisingly large number of chromosomal aberrations in a screen for mutations that reduce meiotic drive of the maize Ab10 chromosome. From approximately 11,340 progeny, five Ab10 deletions were identified [(DF(B), Df(L), Df(L-2), Df(L-3) and Df(M)] and a duplication [Dp(A)]. Here we will discuss the possible factors contributing to the apparently high levels of breakage, the unexpectedly high transmissibility of the deletions, and the implications for the evolution of meiotic drive systems.

### *The mechanism of chromosome breakage*

Each of our Ab10 deletions were identified in lines with active *Robertson's Mutator* elements. Previously, *Mu* was shown to cause chromosome breakage on the short arm of chromosome 9. Using the *yellow-green-2* locus (*yg2*) as a marker, Robertson and Stinard screened over 779,000 seedlings for deletions (Robertson, Stinard & Maguire, 1994). One hundred and twenty-five yellow green seedlings were identified and 18 were confirmed to have deletions on the short arm of chromosome 9. While impressive, this rate of this chromosome breakage ($2 \times 10^{-5}$) is more than 20 times lower than the rate of breakage we found on Ab10 (7/11340, or $6 \times 10^{-4}$). We also found three instances in a non-*Mu* background where the terminal portion of Ab10 had broken from the remainder of the chromosome at meiosis II (Figure 3). These data

suggest that *Mu*-induced chromosome breakage is not the only source of deletions in our mutant screen.

One possibility for the source of breakage events is aberrant chromosome segregation caused by the structure of Ab10. For instance, chromosome breakage could be caused by pairing and recombination within the inverted region of Ab10. An expectation from this model is that pachytene inversion loops would be observed in heterozygous strains. However, inversion loops have never been observed in heterozygous Ab10/N10 plants, though they may occur at low frequencies. Another more likely scenario is that the knob DNA plays role in destabilizing the chromosome. Heterochromatin is slow-replicating, and knob heterochromatin is the last DNA to complete replication in the maize genome (Pryor et al., 1980). Perhaps as a consequence, knob heterochromatin is correlated with failure of sister chromatid separation, anaphase bridge formation, and presumably chromosome breakage (Fluminhan, Aguiar-Perecin & Santos, 1996; Fluminhan & Kameya, 1997). Five of the mutations described here appear to have occurred either within a knob [Df(B)] or immediately flanking a knob [(Df(M) and the Df(L) class], consistent with the idea that knob heterochromatin had a causal role in the breakage events. Under this model, Dp(A) may have arisen through a breakage-fusion-bridge (BFB) cycle, which is initiated by chromosome breakage and can cause multiple rounds of deficiency and/or duplication events (McClintock, 1941b).

*Large Ab10 deletions are tolerated*

Cytologically detectable deletions are rarely recovered in diploid plants such as maize. The presumed reason for the paucity of transmissible deletions is that much (~60–70%) of the genome is expressed during the haploid gametophyte stage of plant development (Mascarenhas, 1989). Deleterious alleles are genetically exposed at this stage and are likely to be eliminated. In maize the only known cytologically detectable deletions are flanking the centromere or at the very ends of chromosomes (McClintock, 1941a, 1944; Rhoades & Dempsey, 1953; Chao et al., 1996). These regions are known to be rich in heterochromatin and poor in mapped genes (e.g., Davis et al., 1999). Deficiencies in gene-rich, interstitial regions have been identified, but they are cytologically undetectable and generally not transmitted through the male (Stadler & Roman, 1948; Carlson, 1988).

At odds with most of the published work in maize, we find that very large deficiencies are tolerated at the end of the long arm of Ab10. Previous work (Rhoades & Dempsey, 1985; and verified here) established that seeds homozygous for the Df(F) deletion, which includes roughly half of the region occupied by the drive system, can be recovered as feeble plants. A rough calculation based on analysis of cytological data (Dawe et al., 1994; Dawe & Cande, 1996) suggests that Df(F) is missing roughly 1% of the euchromatin in the cell. This equates to ~30 cM assuming that the level of recombination in the distal portion of the drive system is roughly the same as recombination in the proximal portion (see Rhoades & Dempsey, 1985 for distance estimates). We cannot assess whether the more severe Ab10 deletions, Df(B), Df(C) and Df(I), are homozygous-viable because they are not transmitted through the male. Nevertheless, these deficiencies are transmitted at high levels through the female (Tables 2 and 3), and lack approximately 1.5% of the euchromatin in the cell. A deficiency of this size correlates to roughly a third the size of the *Arabidopsis* genome (Arabidopsis Genome Initiative, 2000) and more than twice the size of any other transmissible deletion in maize (Carlson, 1988; Chao et al., 1996). The high transmissibility of the Ab10 deficiencies suggests that much of the genetic information in the drive system is not essential for plant growth.

*Implications for evolution of meiotic drive: information-poor regions of the genome are most susceptible*

A common denominator in the best-studied drive systems is the tendency for the various *cis*- and *trans*-active functions to become linked together by structural polymorphisms such as inversions (Lyttle, 1991). This allows multiple drive loci and positive modifiers of the drive loci to evolve in concert without being separated by recombination (see also Hiatt, Kentner & Dawe, 2002). However, suppressed recombination also allows deleterious alleles to build up within the linkage group/haplotype, which can limit the spread of the meiotic drive system (Lyttle, 1985; Ardlie, 1998). One might anticipate that successful meiotic drive systems are more likely to evolve in information-poor regions of the genome that have a minimal impact on the fitness of the organism. Consistent with this idea, the majority of known drive systems appear to be located in gene-poor regions of the genome such as pericentromeric regions, dispensable chromosomes,

or as with Ab10, at the end of a chromosome where there are relatively few genes.

In general, gene content/unit length is lowest in the regions flanking centromeres (e.g., Arabidopsis Genome Initiative, 2000; International Human Genome Sequencing Consortium, 2001). The mouse *t*-haplotype, the *Drosophila Segregation Distorter* and *Sex Ratio* systems, and the mosquito *Male Drive* system, are all localized in pericentromeric regions (Lyttle, 1991). Dispensable chromosomes are another particularly fertile ground for the evolution of meiotic drive systems. Many plants and animals have dispensable B chromosomes, which have been shown to accumulate by meiotic drive (Zimmering, Sandler & Nicoletti, 1970). These are particularly well studied in grasshoppers, where the B chromosomes are highly polymorphic and composed primarily of repetitive DNA (Camacho et al., this volume). B chromosomes are likely to have evolved from alien (extra) chromosomes introduced from other species (Östergren, 1945; John et al., 1991). The potential for alien chromosomes to demonstrate preferential segregation has been uncovered several times in plants (Cameron & Moav, 1957; Maguire, 1963 and references therein). As might be expected, dispensable chromosomes sustain large deletions without detriment to the organism (Carlson, 1988; Hu & Quiros, 1991).

The Ab10 meiotic drive system evolved along with structural polymorphisms in a terminal section of chromosome 10. Much of this genetic information, occupying upwards of 30 cM, is not essential for growth. These data suggest that the meiotic drive system lies in chromatin with relatively little information content, either as a result of low gene density or high genetic redundancy. In fact there are relatively few known markers in the region of chromosome 10L where the major components of the drive system map (roughly position 120 on the Davis et al., 1999 map; Mroczek & Dawe, unpublished). These data are consistent with the view that there are relatively few genes at the end of chromosome 10L, but more thorough sequence information will be required to make firm conclusions. Interestingly, many of the knobs on the other nine maize chromosomes lie at a considerable distance from the ends of chromosomes, in regions that are almost certainly densely populated with genes (Buckler et al., 1999; Hiatt, Kentner & Dawe, 2002). These secondary knob sites exploit the *trans*-acting factors on Ab10 and do not have the structural polymorphisms typical of complete meiotic drive systems. As a result, secondary knobs are less likely to occur in linkage disequilibrium with deleterious alleles.

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