

***Jittery*, a *Mutator* Distant Relative with a Paradoxical Mobile Behavior: Excision without Reinsertion**

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The unstable mutation *bz-m039* arose in a maize (*Zea mays*) stock that originated from a plant infected with barley stripe mosaic virus. The instability of the mutation is caused by a 3.9-kb mobile element that has been named *Jittery* (*Jit*). *Jit* has terminal inverted repeats (TIRs) of 181 bp, causes a 9-bp direct duplication of the target site, and appears to excise autonomously. It is predicted to encode a single 709–amino acid protein, JITA, which is distantly related to the MURA transposase protein of the *Mutator* system but is more closely related to the MURA protein of *Mutator*-like elements (MULEs) from *Arabidopsis thaliana* and rice (*Oryza sativa*). Like MULEs, *Jit* resembles *Mutator* in the length of the element's TIRs, the size of the target site duplication, and in the makeup of its transposase but differs from the autonomous element *Mutator-Don Robertson* in that it encodes a single protein. *Jit* also differs from *Mutator* elements in the high frequency with which it excises to produce germinal revertants and in its copy number in the maize genome: *Jit*-like TIRs are present at low copy number in all maize lines and teosinte accessions examined, and JITA sequences occur in only a few maize inbreds. However, *Jit* cannot be considered a bona fide transposon in its present host line because it does not leave footprints upon excision and does not reinsert in the genome. These unusual mobile element properties are discussed in light of the structure and gene organization of *Jit* and related elements.

INTRODUCTION

Recent surveys of the *Arabidopsis thaliana* (*Arabidopsis* Genome Initiative, 2000) and rice (*Oryza sativa*) (Goff et al., 2002) genome sequences have revealed that plant genomes contain an abundance of transposon-related sequences. Because the biological activity of transposons has been best characterized in maize (*Zea mays*), many of the transposon sequences in *Arabidopsis* (Le et al., 2000; Yu et al., 2000) and rice (Mao et al., 2000) were identified purely by their homology to known maize transposons. The diversity of maize transposons was established more than 50 years ago by McClintock (1952) on the basis of classical functional complementation tests that enabled her to group transposons into families or systems. Several such families were recognized subsequently (Peterson, 1978, 1988). Yet, only four autonomous (self-transposing) maize transposons have been isolated and sequenced to date: *Activator* (*Ac*)

(Fedoroff et al., 1983; Muller-Neumann et al., 1984; Pohlman et al., 1984) and the related *Bergamo* (Hartings et al., 1991), *suppressor-Mutator/Enhancer* (Pereira et al., 1986; Masson et al., 1987), and *Mutator-Don Robertson* (*MuDR*) (Chomet et al., 1991; Qin et al., 1991). *Ac*, *suppressor-Mutator*, and *MuDR* are founding members of the *hAT*, *CACTA*, and *Mutator* transposon superfamilies, respectively (Kunze and Weil, 2002; Walbot and Rudenko, 2002). Not surprisingly, these are the maize transposon sequences used in the homology scans of the rice and *Arabidopsis* genomes. Multiple representatives of each sequence were found in both species. Therefore, the characterization of new autonomous elements would help to elucidate functional relationships among transposons in maize and, possibly, to identify closely related transposons in other plant species. This work deals with one such element, *Jittery*, which was activated apparently by infection with barley stripe mosaic virus (BSMV), is only distantly related to *Mutator*, and displays a most atypical transposition behavior: excision without reinsertion.

BSMV is capable of systemically infecting some susceptible maize stocks (Sprague and McKinney, 1966; Pring, 1973) and, possibly, of activating dormant transposable elements and initiating chromosome breakage-fusion-bridge cycles (Mottinger et al., 1984a). Stocks derived from plants infected with BSMV are known as aberrant ratio (AR) stocks for historical reasons. Sprague and McKinney (1966) coined the term aberrant ratio to describe departures from Mendelian expectations in the progeny of maize plants that had been infected with BSMV, wheat streak mosaic virus, or lily fleck maize virus. Subsequent genetic studies (Samson et al., 1979; Brakke et al., 1981; Nelson, 1981)

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demonstrated that many of the apparent ratio distortions in AR lines could be explained by segregation of cryptic mutations at epistatic loci. Because Sprague's original lines were homozygous dominant for all the factors being scored, new recessive mutations were apparently being produced either directly or indirectly by the viral infection.

In a series of experiments designed to isolate and characterize the new mutations arisen in AR lines, Mottinger and collaborators were able to recover mutations at the *Adh1* locus directly from BSMV-infected plants. The frequency of these mutations was 10^{-5} (Mottinger et al., 1984b), at least 100 times higher than the spontaneous mutation frequency at that locus (Freeling, 1977). The molecular analysis of one such mutation established that it had arisen by insertion of *Bs1*, the first retrotransposon described in plants (Johns et al., 1985; Jin and Bennetzen, 1989). Mottinger and collaborators also recovered a high number of new stable and unstable *sh* and *bz* mutants in AR stocks that were several generations removed from a plant inoculated with BSMV (Mottinger et al., 1984a; J. Mottinger, unpublished data) and suggested that viral infection constituted the type of genomic stress envisioned by McClintock (1984) as activating quiescent elements. Two of the unstable *sh* mutations were shown to carry insertions of 2.0 and 3.6 kb (Dellaporta et al., 1984). The recovery of different insertions from *sh* mutants that arose in AR stocks suggests a relationship between viral infection, chromosome breakage, and transposition in maize.

bz-m039 is another unstable mutation isolated from AR stocks. It produces a heavy, though finely, spotted seed phenotype and can occasionally revert to a solid purple phenotype in the germline, hence its *bz-m* designation (Figure 1). The somatic and germinal instability of *bz-m039* indicate that the mutation arose from the insertion of a transposon that is capable of subsequent excision and is therefore not a retrotransposon. The transposon at *bz-m039*, which appears to excise autonomously, has been isolated and named *Jittery* (*Jit*). Although it is distantly related to *MuDR*, the master element in the *Mutator* family, *Jit* differs in overall organization from *MuDR*. It also differs in transposition behavior from all previously characterized transposons. At least in its current host inbred line, *Jit* excises without leaving footprints and does not reinsert in the genome. This unusual transposition behavior is discussed in reference to *Jit*'s origin and structure.

RESULTS

Genetic Instability of *bz-m039*

Although the vast majority of kernels in *bz-m039* × *bz-R* testcross ears are spotted, occasional purple kernels do occur (Figure 1A). These purple kernels represent putative *Bz'* germinal revertants of the *bz-m039* mutable allele. However, more than half of them do not breed true, for example, they carry an embryo and an endosperm that are genetically discordant, indicating that the reversion events that produced them occurred postmeiotically (i.e., at one of the megagametophytic mitoses). Conversely, full *Bz'* revertant plants are occasionally seen in *bz-m039* families planted from spotted seed, providing evidence that *bz-m039* can specifically revert in the last gametophytic mitosis that gives rise

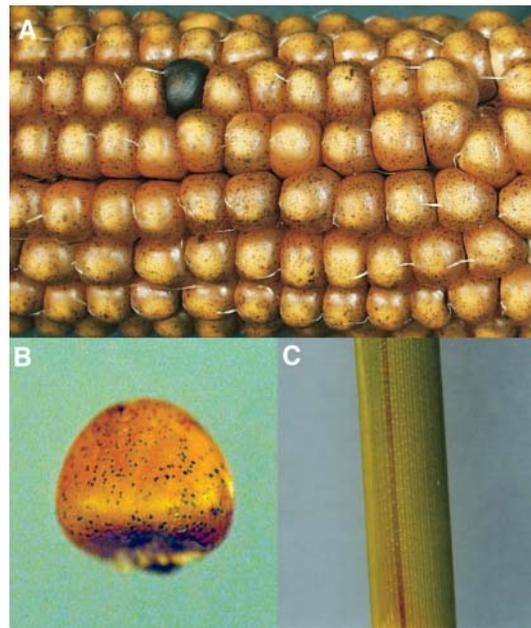


Figure 1. *bz-m039* Mutable Phenotypes.

(A) Homozygous *bz-m039* ear showing late and frequent somatic reversions in the aleurone (fine purple spots) and a putative germinal reversion (purple kernel) to *Bz'*.

(B) Close-up of a single kernel in **(A)**. The small size of most purple spots indicates that reversion events occur late in aleurone development.

(C) *bz-m039* seedling showing a fine purple stripe in the leaf sheath.

to the egg and to its sister polar nucleus (Cooper, 1938; Simcox et al., 1987). Nevertheless, concordant germinal *Bz'* reversion events occur, mostly as single kernels in the ear, at a frequency of about 1 per 1000 gametes (Table 1). These germinally transmissible reversions may arise at meiosis or at an early megagametophytic division that would generate a revertant egg nucleus and at least one matching polar nucleus.

Somatic reversion events in the aleurone occur late in development, as evidenced by the small size of most purple spots (Figures 1A and 1B). Similarly, reversion events in the sporophyte (the maize plant) are seen as fine purple stripes in the leaf sheaths (Figure 1C) or fine purple spots in the anthers (data not shown), again indicating that they occur at late developmental stages.

Jit, a New Maize Transposon

Isolation of *Jit*, the Transposon in *bz-m039*

The genetic instability of the *bz-m039* mutation suggests that an excisable transposable element has inserted in the *bz* locus. To confirm this, DNA of *bz-m039* mutants was compared with that of *Bz'* revertants by DNA gel blot analysis. A *bz* probe detected a band that was 3.9 kb larger in *bz-m039* than in its *Bz'* germinal revertants in two different digests (Figure 2; data not shown). Thus, *bz-m039* carries a 3.9-kb transposon, which was named *Jittery*. By a series of restriction digests, it was determined that *Jit* had inserted in the second exon, close to the 3' end of the

Table 1. Germinal Reversion of *bz-m039* to *Bz'*

Cross	<i>bz-m</i> Gamete Population	No. <i>Bz'</i> Selected and Tested	No. <i>Bz'</i> Confirmed	No. <i>bz-m</i>	Other ^a	Frequency of <i>Bz'</i> Germinal Reversion ^b
<i>Sh bz-m039 Wx; R-r x sh bz-R wx; R-sc</i> <i>sh bz-R wx</i>	18,070	48	16	31	1	0.9×10^{-3} [0.5 to 1.4×10^{-3}]

^aFrom pollen contamination, this individual lacked the *sh bz-R wx; R-sc* contribution of the pollen parent.

^b95% Confidence limits.

bz gene. The transposon was then isolated by PCR using *bz*-specific primers and sequenced.

Jit is 3916 bp long, has terminal inverted repeats (TIRs) of 181 bp, and causes a 9-bp duplication of its target site (Figure 3A). Curiously, the inverted repeats are not perfect: four extra bases (GCTC) occur 1 bp away from the 5' end of the element. *Jit* encodes a single putative protein of 709 amino acids (Figure 3C), which is homologous to several MURA-like (*Mutator*-like) transposases present in the Arabidopsis and rice genomes (highest scores: AAC77869, 296, $E = 2e^{-79}$; AAN65443, 296, $E = 7e^{-79}$, respectively) and to a *Mutator*-like truncated transposase encoded by a 1.3-kb insertion in tomato (*Lycopersicon esculentum*) (Young et al., 1994). Hence, this protein has been designated JITA. JITA is also homologous to the FAR1 transcriptional activator that is involved in phytochrome signaling in Arabidopsis (Hudson et al., 1999, 2003). However, it shows lower similarity to the MURA protein from the maize *MuDR* element (score: 41, $E = 8e^{-05}$) than to the above heterologous proteins. Although the putative transposases of *Jit* and *Mutator* are only very distantly related, the two elements resemble each other in the relative length of their TIR (longer than in most other excisive transposons) and in the size of their target site duplication (TSD).

Jit Excision

Jit excises autonomously, as judged from the following evidence. If *Jit* was nonautonomous and depended for its excision on a second transposon located elsewhere in the genome, stable *bz-s* derivatives carrying *Jit* at *bz* would have been expected from *bz-m039*, yet none were found. Such *bz-s* derivatives were sought in two kinds of experiments. First, rare *Sh bz Wx* seed were selected from testcrosses of *Sh bz-m039 Wx/sh bz-R wx* heterozygotes and analyzed by DNA gel blots and sequencing. The *Sh* and *Wx* flanking markers ensure that most selections are derived from *bz-m039* because of the strong positive chiasma interference in the *sh-wx* region (Dooner, 1986). Seven putative *bz-s* derivatives were obtained from a population of 18,070 *bz-m* gametes, but none were confirmed. They either retained the insertion at *bz* and failed to breed true or lacked the polymorphisms of the *bz-m039* progenitor allele, indicating that they were either mispicks or pollen contaminants in the previous generation. This experiment rules out the existence of a segregating autonomous element outside of the *sh-wx* region. Second, *bz* seed were also selected from testcrosses of *bz-m039* homozygotes after three generations of backcrossing to a *W22 bz-R* stock. Only two stable *bz* seeds were recovered from

among 3400 testcross progeny seed. Upon further analysis, one still carried the insertion at *bz* but failed to breed true. The second one represented a true stable *bz-s* mutation arisen from an aberrant excision of *Jit* from the *bz* locus. In this new mutation, *bz-s0330*, the entire *Jit* element has been lost, together with 22 bp of *bz* sequence located immediately proximal to *Jit* (5' relative to JITA), and the distal member of the TSD has been substantially altered (Figure 3B).

The present genetic data do not rule out the formal possibilities that a closely linked autonomous element is responsible for the observed mutability of *bz-m039* or that both the *bz-m039* and the *bz-R* parents carry a separable, autonomous transposon at the same location in the genome. However, the latter possibility is rendered less likely by the observation that the bronze and

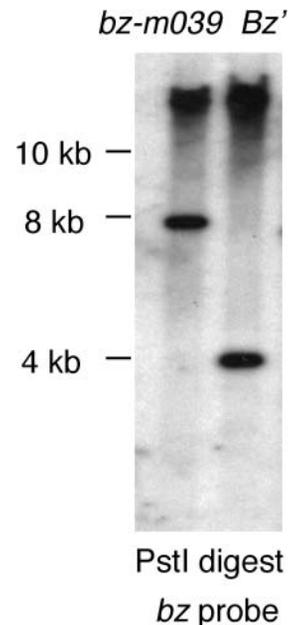


Figure 2. DNA Gel Blot Analysis of *bz-m039* and a *Bz'* Germinal Revertant.

The DNA was digested with *PstI* and hybridized to the pAGS528 *bz* probe (Ralston et al., 1988). The probe detects a band that is 3.9 kb larger in *bz-m039* than in the *Bz'* germinal revertant. The smeared signal at the top is because of the very high GC content of the *bz* probe, causing it to hybridize nonspecifically to the bulk of undigested genomic DNA, which is common in *PstI* digests. It is not seen in digests with methylation-insensitive enzymes.

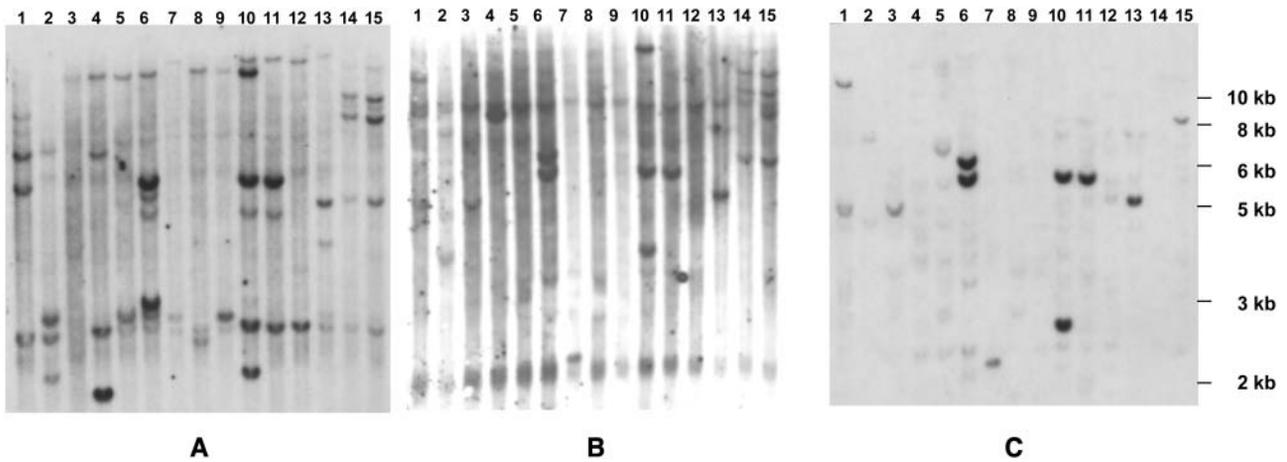


Figure 4. DNA Gel Blot Analysis of *Jit*-Related Sequences in Maize Inbred Lines and Teosinte Accessions.

Genomic DNA was digested with *Sst*I and hybridized sequentially to probes from different parts of the *Jit* element. Lane 1, *Z. mays* ssp *mexicana*, collection 107-5-3; lane 2, *Z. mays* ssp *mexicana*, collection 101-5-2; lane 3, *Z. mays* ssp *parviglumis*, collection 105-2-3; lane 4, A188; lane 5, A636; lane 6, B73; lane 7, BSSS53; lane 8, 4Co63; lane 9, H99; lane 10, M14; lane 11, Mo17; lane 12, W23; lane 13, AR stock from G. Sprague; lane 14, W22; lane 15, *bz-m039/bz-R*.

(A) *Jit* 5' end (position 1 to 471).

(B) *Jit* 3' end (position 3334 to 3916).

(C) *jitA* (position 1262 to 2098).

Characterization of *Jit* Excision Products

A general characteristic of excisive transposons is that they usually leave footprints at the site of excision. All or part of the host TSD created upon insertion is left behind, often with deletions and transversions of the bases next to the transposon termini. To determine whether *Jit* leaves footprints upon excision, the *Jit* excision products or empty sites of the 16 *Bz*' germinal revertants from Table 1 were PCR amplified and sequenced. Surprisingly, none of them had footprints. Spurious explanations, such as sample mix-ups or PCR contamination, can be ruled out because the sequence of the *Bz* progenitor allele of *bz-m039* around the *Jit* insertion site is unique and was shared by all the germinal revertants. A possible explanation of the failure to detect footprints among *Bz*' germinal revertants is that selection for *Bz* function may impose an artificial constraint on the nature of *Jit* excision products recovered because *Jit* is inserted in a part of the *Bz* glucosyltransferase that may not tolerate changes, namely, its UDP-Glc binding site.

To avoid analyzing selected excision products, unselected somatic excisions were amplified, cloned, and sequenced. Sixty plasmids, representing six excision products from each of ten different *bz-m039* homozygous plants were sequenced, and again, no footprints were found. Thus, *Jit* differs from all the plant transposons studied so far in that it does not seem to leave footprints. In the *Ac-Ds* system, +0 excision products can be more common from some positions of *Ds* in the *wx* gene than from others (Scott et al., 1996) yet are never the exclusive product. The lack of detectable footprints among *Jit* excision products has bearing on the mechanism by which the DNA double-strand break produced by excision of the transposon is repaired and, therefore, on the mechanism of *Jit* transposition.

Jit Reinsertion

The lack of a nonautonomous reporter allele does not make it feasible to determine by genetic tests if excised *Jit* elements reinsert in the maize genome (i.e., if they actually transpose). Fortunately, the low copy number of *Jit*-related sequences in *bz-m039* stocks allows reinsertion to be scored by molecular tests. To that end, DNA gel blots of selected and unselected progeny from *bz-m039* were examined for new *Jit*-hybridizing bands. The progeny included the 16 *Bz*' germinal revertants of

Table 2. Occurrence of *jitA*-Hybridizing Bands in Some North American Inbred Lines

Inbred Line	No. <i>jitA</i> -Hybridizing Bands ^a
4Co63	0
A188	0
A636	0
B37	0
B73	2 ^b
BSSS53	0
H99	0
Ill12E	0
M14	2 ^b
Mo17	1 ^b
SD purple	0
W22	0
W23	0
W64A	2 ^b

^a Hybridization to a 0.8-kb internal fragment from the *JIT*A coding sequence.

^b These lines have a 6-kb *Sst*I fragment in common.

Table 1, the stable derivative *bz-s0330*, and 50 unselected progeny from *bz-m039/bz-R* heterozygotes. Unexpectedly, no new bands were found (Figure 5; data not shown), indicating that, although it can excise, the *Jit* element in *bz-m039* does not reinsert in its current host line.

Interaction between *Jit* and *Mutator*

Although *Jit* does not appear to reinsert in present stocks, it obviously did so in the AR stock in which the *bz-m039* mutation arose. A possible explanation for this is that a second, most likely unlinked, function essential for reinsertion was inadvertently lost during propagation of *bz-m039*, while the mutation was being tracked simply by its spotted seed phenotype. In this scenario, at least two genes are required for the mobilization of *Jit*: the gene in the transposon that encodes JITA, the putative transposase, and one or more genes that may or may not be carried in a mobile element. As discussed below, the analogy between this aspect of *Jit* and some recently uncovered features of the *Mutator* system is striking.

The regulatory transposon of the *Mutator* system, *MuDR*, encodes two proteins: MURA and MURB (Hershberger et al., 1995). MURA was postulated to be a transposase on the basis of weak homology to bacterial transposases (Eisen et al., 1994). Two lines of evidence support that postulate. (1) A *MuDR* deletion that retains only the MURA coding region is able to cause excision of a *Mutator1* (*Mu1*) reporter element (Lisch et al., 1999), and (2) a transgene encoding only MURA is sufficient to catalyze excision of *Mutator* elements (Raizada and Walbot, 2000). Most interestingly, in neither case was reinsertion of *Mutator* elements observed, suggesting that a second function, possibly encoded by MURB, is required for insertion. As has been pointed out (Lisch et al., 1999), although there are many Arabidopsis and rice sequences homologous to MURA in the sequence databases, no known sequences share significant similarity with MURB. Thus, MURB may be specific to the requirements of the *Mutator* system in maize.

To test if the *MuDR*-encoded MURB protein can provide the function required for the reinsertion of *Jit*, the following experiment was performed. A *Sh bz-mum1 Wx/sh bz-R wx* F1 heterozygote from a cross between a *sh bz-R wx* stock and an active *Mutator* line carrying the *bz-mum1* reporter allele was self-pollinated, and segregating *sh bz wx* F2 seed were selected. The *bz-mum1* allele carries a *Mu1* element in the *bz* locus (Brown et al., 1989) and produces spotted seeds in the presence of *MuDR*. Because of the tendency of *Mutator* elements to maintain their high copy number by replicative transposition in the germline (Alleman and Freeling, 1986; Walbot and Rudenko, 2002), most of the *sh bz wx* segregants should have multiple copies of *MuDR* in the genome. They were then crossed to a *bz-m039* stock, and the resulting F1 individuals were testcrossed with *sh bz-R wx* to isolate *Bz'* germinal revertants. Eleven concordant *Bz'* revertants were obtained from a population of 7940 *bz-m* kernels, corresponding to a frequency of 1.4×10^{-3} , which does not differ significantly from that reported in Table 1 for a non-*Mutator* stock (95% confidence limits = 0.7 to 2.4×10^{-3} ; Stevens, 1942). The revertants were characterized by sequencing of the *Jit* excision site and DNA gel blot analysis. Several new *Mu1* bands, but no new *Jit*-hybridizing bands or transposon footprints, were found in the *Bz'* revertants (Figure 5; data not shown), suggesting that MURB affects neither the excision nor the reinsertion of *Jit* in the genome.

DISCUSSION

The *bz-m039* mutable allele described here arose in an AR stock that traced its origin to a plant that had been infected with BSMV several generations earlier. Other unstable *sh* and *bz* alleles arose in those stocks (Mottinger et al., 1984a; J. Mottinger, unpublished data), suggesting that the original viral infection episode may have triggered the transposition of previously quiescent transposable elements that remained mobile for several generations but undetected until they were recovered in a reporter allele, such as *sh* or *bz*. The transposon that excises from the *bz-m039* allele

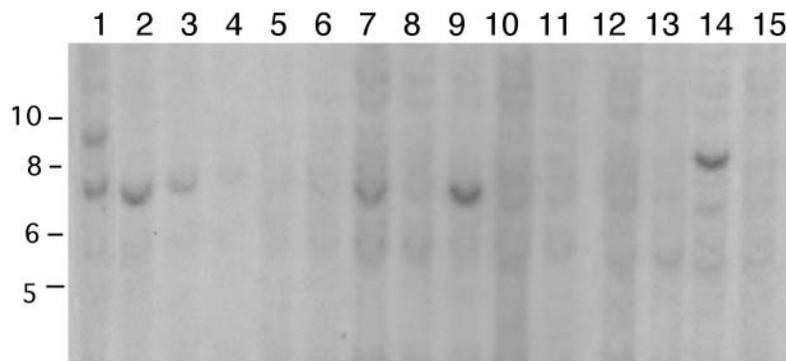


Figure 5. DNA Gel Blot Analysis of *Bz'* Revertants from *bz-m039*.

Genomic DNA was digested with *Sst*I and hybridized to a *jitA* probe. Lane 1, *bz-m039/bz-R* heterozygote; lanes 2 to 13, *Bz'* revertants, as *Bz'/bz-R* heterozygotes; lane 14, *bz-m039/bz-R* heterozygote; lane 15, W22 *bz-R*. The ~ 7 -kb *jitA*-hybridizing band seen in the *bz-m039/bz-R* heterozygote in lane 1, but not in lane 14, and in some of the *Bz'* revertants was apparently present in the original *bz-m039* stock. It segregates independently of the *bz* locus and is found in some *bz-m039* lineages only (cf. Figure 4).

to produce the spotted aleurone and striped plant phenotypes shown in Figure 1 is *Jit*, a novel maize element assigned to the *MuDR* superfamily of plant transposons (Walbot and Rudenko, 2002) based on the homology of their putative transposases.

Jit is a 3.9-kb transposon predicted to encode a single 709-amino acid protein. Because this protein is distantly related to the MURA transposase encoded by *MuDR* (Eisen et al., 1994; Hershberger et al., 1995), it has been named JITA. JITA is much more closely related to the MURA protein of *Mutator*-like elements (MULEs) from *Arabidopsis* and rice. However, its closest homolog in the GenBank databases is the *Arabidopsis* FAR1 transcriptional activator involved in phytochrome signaling, prompting Hudson et al. (2003) to speculate that some transcription factors could have evolved directly from immobilized transposases that retained their ability to bind DNA.

Jit and *MuDR* share several structural features, including their relatively long TIRs (~200 bp), the size of their TSD (9 bp), and the homology, if distant, of their respective transposases. However, *Jit* differs from *MuDR* in that it encodes a single protein. In this, it resembles all the other MULEs in the sequence database, which do not encode a MURB homolog. *Jit* also differs from *Mutator* elements in genomic abundance. Based on hybridization criteria, sequences highly similar to JITA are absent from most lines of maize examined or are present in just one or two copies, even in the active *bz-m039* lines. Sequences homologous to the *Jit* ends are detected in all maize lines, but the number of bands hybridizing strongly to an end probe is much lower for *Jit* (two to six) than for *Mu1* (~40) (Chandler et al., 1986).

The biggest difference between *Jit* and maize *Mutator* elements is in their genetic behavior. *bz-m039* reverts germinally to *Bz'* at a frequency close to 1 in 1000 (Table 1). By contrast, mutable alleles harboring *Mutator* elements rarely revert germinally, their average frequency of germinal reversion being $<10^{-5}$ (Walbot and Rudenko, 2002). *Mutator* elements leave typical transposon footprints upon excision; *Jit* excises without leaving footprints in either somatic or germinal tissues. The most striking difference is in the actual transposition of the elements. *Mutator* elements are bona fide transposons that move to new locations in the genome, by either replicative (Lisch et al., 1995) or excisive (Raizada et al., 2001) transposition. *Jit* can excise at high frequency either at meiosis or in the megagametophyte, but it does not reinsert in the genome. The genetic data suggest that *Jit* excision is an autonomous property of the element. Yet, technically, *Jit* cannot be considered an autonomous transposon in its current host line. Instead, *Jit* properly should be called an autonomous excision: an element capable of specifying its own chromosomal removal but not its reinsertion. Clearly, an element that behaves like *Jit* in the current *bz-m039* lines would be an evolutionary dud: it would neither generate genetic diversity nor propagate itself, and its failure to reinsert would result in its quick elimination from the genome. Although *Jit* does not transpose in the stocks analyzed, it did transpose recently when it inserted in *bz*. A second function required for reinsertion, but not excision, may have been present in the stock where *bz-m039* arose. This function could have been easily lost by segregation in a subsequent generation because the mutation was tracked from one generation to the next by its spotted or excision phenotype.

Recent studies with the *Mutator* system have revealed that certain *MuDR* derivatives that lack the MURB function behave essentially like *Jit* in being capable of excision but not of reinsertion (Lisch et al., 1999; Raizada and Walbot, 2000). The simplest interpretation of this observation is that whereas MURA alone is required for excision, both MURA and MURB are required for transposition (Lisch, 2002; Walbot and Rudenko, 2002). The parallels between the two systems are compelling enough to postulate the existence of a JITB function for *Jit*. The JITB protein would be responsible for both the reinsertion of *Jit* and for the formation of footprints. It could do this, for example, by binding to the receptor site, enabling the formation of staggered cuts nine bases apart, and to the donor site, blocking the default annealing of the complementary single strand ends generated upon excision, which would then be variably processed by DNA repair enzymes to produce the typical footprints seen with other transposons. In this view, the moderate target site sequence preference exhibited by *Mutator* elements (Raizada et al., 2001; Dietrich et al., 2002) could be a property of MURB. The B function of members of the *MuDR* superfamily would parallel the function of TnsD or TnsE, proteins that determine target site preference and are essential for the transposition of *Tn7* in *Escherichia coli* (Craig, 2002). Interestingly, given the total lack of MURB-related sequences in the databases, no sequences similar to TnsE are found among *Tn7* relatives in other bacteria. In the absence of JITB, JITA would still bind to sites in the *Jit* TIRs, analogous to the MURA binding sites found in the *MuDR* TIRs (Benito and Walbot, 1997), and make staggered cuts in the DNA immediately adjacent to the transposon ends. However, the resulting double-strand break would be subsequently repaired without footprint formation by the action of an endogenous ligase present in all maize lines, and, in the absence of a cleaved receptor site, *Jit* would fail to reinsert in the genome. If the above model also applies to *Mutator*, one would predict that the somatic excisions of *Mu1* observed in the absence of MURB (Lisch et al., 1999; Raizada and Walbot, 2000) would also lack footprints, but this issue was not addressed in those studies.

Several Maize Lines Can Be Considered as Possible Sources of JITB

The Original AR Line Where *bz-m039* Arose

This stock shows a strong hybridizing band with *jitA* and TIR probes (Figure 4, lane 13) and has been confirmed by sequencing to carry the *Bz* progenitor of *bz-m039* (Z. Xu, unpublished data). The *bz-m039* mutation is currently being crossed back into this line to test for *Jit* reinsertion after excision.

Maize Inbred Lines with *Jit* Homologs

A few maize inbreds carry sequences that hybridize strongly to a *jitA* probe (e.g., M14, Mo17, and B73; Figure 4). If the A and B functions are present together in other elements of the *Jit* family, as they are in *MuDR*, these inbreds are also potential sources of the JITB function. These lines are also being tested for *Jit* transposition. Another possible source of JITB are lines carrying

a two-element mutable system designated *rMrh/Mrh* (Shepherd et al., 1989). *Mrh* was postulated on genetic grounds to transactivate the excision of the *rMrh* insertion from the *a1-mrh* mutable allele. This insertion is only 246 bp long, but its 80-bp TIRs are 76% identical with *Jit*'s TIRs over the first 50 bp and, like *Jit*, *rMrh* is flanked by a 9-bp duplication of the target site. Thus, *rMrh* and *Jit* may belong to the same family of mobile elements. It is not known though if excised *rMrh* elements can reinsert in the genome.

Mutator Lines

The JTB function conceivably could have been provided by MURB, a protein that has been implicated in the reinsertion of *Mutator* elements. This possibility was tested by crossing *bz-m039* into an active *Mutator* line and looking for new *jitA*-hybridizing bands among *Bz'* revertants. However, no new *jitA*-hybridizing bands or transposon footprints were found, indicating that MURB cannot provide the reinsertion function missing in *Jit*.

The putative gene encoding JTB may or may not be carried in a mobile element. None of the MULEs that are the closest *Jit* relatives encode a B function, yet these elements can move, at least in *Arabidopsis ddm1* mutants (Singer et al., 2001). Lisch (2002) has summarized other evidence that suggests that these elements are or recently have been active in various angiosperm species, including the identification of apparently complete MULEs in rice, barley (*Hordeum vulgare*), sorghum (*Sorghum bicolor*), and lotus (*Lotus corniculatus*); the occurrence of ESTs corresponding to MULEs in EST databases, and the identification of nearly identical copies of several elements in species with almost completely sequenced genomes. If a B function is required for MULE transposition in those species, the gene encoding it would have to be carried in a different transposon or be an integral component of their genomes.

Although the hypothesis of a missing B function is attractive because of the parallels between *Jit* and *Mutator*, there are other plausible explanations for *Jit*'s puzzling genetic behavior. The presence of four extra bases in the 5' TIR, just one nucleotide away from the end, is unique to *Jit* and may affect its reinsertion properties. This difference between 5' and 3' TIRs is not seen in *rMrh*, the only other known transposable element with TIRs related to *Jit*, or in two different recently isolated maize *Jit*-like sequences: one from the inbred B73 (GenBank Genome Survey Sequence section, accession number CG254112) and one isolated by PCR from the AR stock (Z. Xu, unpublished data). The difference conceivably could have arisen from a DNA repair error in *Jit*'s last transposition event into *bz*. The isolation and characterization of other *Jit*-homologous elements from different sources, particularly from the progenitor stock of *bz-m039*, should shed light on this point. In this model then, JITA would be the only function required for transposition, and *Jit*'s inability to reinsert would be simply the consequence of a TIR structural defect. Even if other elements encoding proteins with high similarity to JITA turn out to have *Jit*-like imperfect TIRs and to behave like *Jit*, there may be other MULEs in the maize genome without such imperfection, perhaps encoding more distantly related A proteins that require only an A function for trans-

position. The *Mrh* element, which has only been defined genetically, would be a candidate for such a MULE in maize.

If a B function is required for *Jit* reinsertion, what could be its origin? In the simplest situation, it would be present together with the A function in truly autonomous members of the transposon family. However, no MULEs in either monocots or dicots encode a second protein. Only *MuDR* does. This would suggest that *mudrB* may be a host gene that was acquired by *MuDR* during a defective DNA repair event to close the gap generated by transposon excision. Fragments of several maize genes have been found in different members of the *Mutator* family (Lisch, 2002), supporting the possibility of host gene acquisition by transposons. If the B protein binds to target DNA to confer the weak degree of target site specificity seen with *Mutator* elements, it may have evolved quickly, possibly explaining the lack of MURB-type proteins in other plants. The appropriation of a host gene by a transposon to increase its spread would be the counterpart of the proposed co-option of a transposase gene as a transcription factor by a host (Hudson et al., 2003) and would lend support to the view that transposon-host relationships are much more mutualistic than previously envisioned (Kidwell and Lisch, 2001).

METHODS

Plant Materials

The *bz-m039* mutation arose as a single spotted (*bz-m*) seed among a majority of purple (*Bz*) seed in an ear from a cross of an AR *Sh Bz Wx* stock as female with a *sh bz-R wx* pollen tester. The AR stock was obtained originally from G. Sprague and was 15 generations removed from a plant infected with BSMV. The mutant was backcrossed twice to a W22 *sh bz-R wx* line and used in genetic tests. The *bz-mum1* allele was obtained from Don Robertson and introduced into a W22 background. *bz-R* is the stable reference allele for the *bz* locus. The teosinte plants trace their origin to seed collections made by Jerry L. Kermicle as follows: *Zea mays* spp *mexicana*, collection 107-5 from Copandaro, Michoacán; *Z. mays* spp *mexicana*, collection 101-5 from Cocotitlán, Chalco, Mexico; *Z. mays* spp *parviglumis*, collection 105-2 from Erendira, Michoacán.

Bz' germinal revertants were isolated from hand pollinated crosses of a *Sh bz-m039 Wx/sh bz-R wx; R-r* heterozygous female parent with a *sh bz-R wx; R-sc* male parent. To protect against pollen contamination, no stocks carrying the *Bz* progenitor of *bz-m039* were grown in the same nursery, and a *bz-R* tester stock with a unique *R* allele was used as male parent. In the presence of all the complementary factors required for anthocyanin pigmentation, the *R-r* allele present in the female parent specifies red plant parts (roots, coleoptile, auricle, anthers, etc.), whereas the *R-sc* allele present in the male parent confers green plant parts, so the latter serves as a pollen contamination marker. In addition, ~700 bp surrounding the *Jit* empty site in the putative *Bz'* revertants were sequenced. Several single nucleotide polymorphisms distinguish the haplotype of the *Bz* progenitor allele of *bz-m039* from that of other *Bz* alleles. All the *Bz'* revertants that segregated the *R-sc* allele had the unique haplotype of the *Bz* progenitor, providing further evidence of their bona fide nature.

Nucleic Acid Extraction, Blotting, and Hybridization

Leaf DNA was isolated by a urea extraction procedure (Greene et al., 1994). DNA concentration was established by comparing aliquots of

sample DNA to 1 μ g λ DNA on an agarose gel stained with ethidium bromide. Restriction digested DNA (10 μ g) was resolved on 0.8% agarose gels and then transferred to Hybond N+ membranes (Amersham Pharmacia, Uppsala, Sweden). 32 P-labeled probes were generated with Ready-To-Go DNA labeling beads (Amersham Pharmacia). The *bz* probe was pAGS528 (Ralston et al., 1988). The primers used for generating *Jit* probes, with their location in the *Jit* sequence indicated in parentheses, were as follows: Jit1F (1 to 24) and Jit3R (451 to 471) for the 5' end, Jit3334F (3334 to 3363) and Jit3'R (3893 to 3916) for the 3' end, and Jit1262 (1262 to 1285) and Jit2098R (2075 to 2098) for *jitA*. Hybridization and membrane washing followed established protocols (Church and Gilbert, 1984).

PCR Amplification and Sequencing

A fragment containing the entire *Jit* insertion was amplified by PCR using *bz* primers C (5'-CTCAACACGTTCCCAGGC-3') and F (5'-CGACAGAC-TATCTCCACGA-3'). PCR was performed following the protocol of Qiataq (Qiagen USA, Valencia, CA). The reaction consisted of 1 \times PCR buffer, 1 \times Q-buffer, 1 mM total deoxynucleotide triphosphates, 400 nmoles of each primer, and 2 units of rTth polymerase (Perkin-Elmer, Foster City, CA). The reaction was denatured at 95°C for 3 min and then cooled to 80°C and held for addition of polymerase. The reaction continued with 37 cycles of 30 s of 94°C denaturation, 30 s of 62°C annealing, and 4 min of 72°C extension. The last 20 cycles autoextended the extension time by 20 s per cycle. After the reaction, a clear sharp band was visible. The band was excised from a gel and purified with a BIO101 glass-bead kit (Q-Bio, London, UK). The PCR product was then cloned into a pGEM-T Easy vector (Promega, Madison, WI) and sequenced on an ABI377 DNA sequencer (Applied Biosystems, Foster City, CA). Fragments were analyzed using the Lasergene99 (DNASar, Madison, WI) software suite. The final *Jit* sequence was used as a query against the sequence databases.

Somatic and germinal *Jit* excision sites were amplified, respectively, using the genomic DNAs of *bz-m039* and *Bz'/bz-R* revertants as templates and primers Bz-C and Bz-3R (5'-AAACCTCTGAACAGCAA-GACGACC-3'). These primers are located 470 bp upstream and 270 bp downstream, respectively, of the *Jit* insertion site. The sequence corresponding to primer C is deleted from the *bz-R* allele present in the *Bz'/bz-R* heterozygotes (Ralston et al., 1988), so only the *bz* fragment from the *Bz'* revertant allele is amplified in the reaction. The PCR products were cloned into pGEM-T Easy and sequenced.

Sequence data from this article have been deposited with the EMBL/GenBank data libraries under accession numbers AF247646, AAN65443, AAC77869, M76798, and CG254112.

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***Jittery*, a *Mutator* Distant Relative with a Paradoxical Mobile Behavior: Excision without Reinsertion**

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