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Origin and diversity of mutants controlled by the *Uq* transposable element system in maize*

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SUMMARY

Three transposable element mutations, displaying an unstable phenotype at the *A* locus, were isolated from lines exhibiting Aberrant Ratio behaviour that originated from maize plants treated with plant RNA viruses. Each of these three unstable mutants is shown to belong to the *Uq-ruq* transposable element system. The two new mutants, along with the previously described *a-ruq* mutant, are nonautonomous in mutability control in that the control of mutability is governed by the segregating *Uq* regulatory element. By following the distribution of different *Uq* elements segregating in independently derived genetic lines and utilizing tests of allelism and linkage, certain lineages of *Uq*-element-containing lines are revealed.

1. ORIGINATION OF A TRANSPOSABLE ELEMENT SYSTEM AFTER VIRUS INFECTION OF MAIZE

A provocative question that has emerged in maize (*Zea mays* L.) studies has its origin in the initial studies of Sprague & McKinney (1966, 1971) in their attempt to induce mutants in maize with the induction of virus symptoms in maize plants. Out of these initial treatments, there arose several exceptional hereditary anomalies. These include, foremost, the phenomenon of Aberrant Ratio, which was represented by segregating populations exhibiting consistent and significant departures in expected Mendelian ratios out of F_2 and backcross populations. In subsequent studies, Samson Brakke & Compton (1979), Brakke, Sampson & Compton (1981) and Nelson (1981) independently showed that these aberrant ratios could be explained by the origin of mutants at epistatic loci that, because of the complementary action of genes involved, gave aberrant ratios. This indicated that new mutations originated in these treated materials.

More strikingly, descendants of virus-infected plants continue to give rise to other mutants. Mottinger, Dellaporta & Keller (1984) have identified mutations at the shrunken and bronze loci out of these descendent lines. Some of the *sh* mutants are unstable and, at the molecular level, show DNA rearrangements.

In the progeny of these original Aberrant Ratio lines originating from Wheat

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Streak Mosaic Virus infection, an unstable *a* allele (spotted expressed as colourless to colour changes) was recovered by G. F. Sprague and subsequently identified as a new transposable element system (based on nonallelism to previously described systems) called the *Uq-a-ruq* system (Friedemann & Peterson, 1982; Peterson & Friedemann, 1983). To examine other mutant *a* alleles (selected by G. F. Sprague) from Aberrant Ratio lines that developed after Barley Stripe Mosaic Virus treatment, tests were made of their homology to other transposable element systems, which are reported here.

Transposable element mutations characterized by an unstable phenotype have been induced under a number of diverse circumstances. These include the bridge-breakage–fusion cycle (McClintock, 1949), irradiation (Peterson, 1952) high chromatin loss lines (Rhoades & Dempsey, 1982), Mutator (*Mu*)-containing lines (Robertson, 1978), lines containing the regulatory element *En* (Peterson, 1978), Aberrant Ratio lines after RNA-virus treatment (Friedemann & Peterson, 1982; Mottinger *et al.* 1984), and the unstable *Adhl-S5446* mutant after BSMV treatment (Freeling, 1984).

Genetic studies on unstable mutants in maize have defined systems of transposable elements that represent specific receptor and regulatory element interactions. The genetic uniqueness of different transposable element systems has been clearly demonstrated in classical genetic tests by McClintock (1956) and Peterson (1965, 1981) since the original discovery of the two-element *Dt-a-dt* controlling element system by Rhoades (1936). Definitive genetic tests to determine transposable element systems have established the *Dt-a-dt* (Rhoades, 1936), *Ac-Ds* (McClintock, 1951), *En*(*Spm*)-*I* (Peterson, 1952), *Fcu-rcu* (Gonella & Peterson, 1977), *Bg-o2m*(*r*) (Salamini, 1981), *Uq-a-ruq* (Friedemann & Peterson, 1982), *Mrh-a-mrh* and *Mut-bz-mut* (Rhoades & Dempsey, 1982, 1983) and *Cy-bz-rcy* (Schnable & Peterson, 1984) transposable element systems.

Further aspects of the uniqueness of transposable element systems have been elucidated by molecular studies in maize (Sachs *et al.* 1983; Fedoroff, Wessler & Shure, 1983; Schwarz-Sommer *et al.* 1984; Pereira *et al.* 1985). These studies have established the similarities in the *Ac* and *Ds* elements of the *Ac-Ds* systems (Fedoroff *et al.* 1983; Behrens *et al.* 1984) and distinguished them from the *En* and *I* elements of the *En*(*Spm*)-*I* transposable element system (Schwarz-Sommer *et al.* 1984; Pereira *et al.* 1985). The main distinguishing points between these systems are the inverted terminal repeats and the host-gene related target site duplication. The two-factor interaction of receptor and regulatory elements (Peterson, 1981) has been described molecularly as *Ds* elements being defective *Ac* elements (Fedoroff *et al.* 1983) by the loss of an internal fragment that represents part of the active component of *Ac*.* Future molecular analysis of transposable element mutants from virus-induced aberrant ratio lines will help clarify the phenomenon of Aberrant Ratio and RNA virus mutagenesis.

* The recent cloning of the *En* transposable element (Pereira *et al.* 1985) has exemplified an *I* element as a defective autonomous *En* element.

2. MATERIALS AND METHODS

(i) *Source of* a *mutants*

Six newly originated *a* alleles were obtained from Dr G. F. Sprague, each independently derived after initial treatment with Barley Stripe Mosaic Virus (BSMV). The procedure involved crossing a dominant marker line (*A*, *C*, *R*, *Su*, *Pr*, *Wx*) infected with BSMV as male on a recessive female tester (*a*, *su*, *pr*, *wx*) (Cross 1) to study virus mutagenesis (Sprague, McKinney & Greeley, 1963; Sprague & McKinney, 1966). The full-coloured F_1 seed were selfed and carried on by selfing and crossing for several generations. Several mutant alleles appeared many generations after the initial treatment.

Two of the *a* alleles designated as *a-m13* and *a-m16* displayed mutability, observed as coloured spots on a colourless background. The seed from the F_2 ear obtained from Dr G. F. Sprague showed a fine low 1-spot type of pattern in the *a-m16* mutant and a low flow (Peterson, 1966) pattern in the *a-m13* mutant.

$$\frac{a\ su\ pr\ wx}{a\ su\ pr\ wx} \times \frac{A\ Su\ Pr\ Wx}{A\ Su\ Pr\ Wx} \quad \text{(BSMV-treated)} \qquad \text{(Cross 1)}$$

The *a-m13* and *a-m16* mutants appeared ten or more generations *after* the initial virus treatment. These newly arisen *a* alleles were sent to the Ames laboratory in 1978.

The variegated kernels were planted in the summer of 1978. A crossing program was developed to test for the heritability of the spotted phenotype of the two mutants and their relationship to the known controlling element systems. For this, the plants originating from the variegated kernels were selfed and outcrossed to testers with the receptor allele of the *Ac-Ds*, *En-I*, *Dt-a-dt*, *Fcu-rcu*, *Bg-o2m(r)* and *Uq-a-ruq* systems. Tests to determine whether the mutability was controlled autonomously or nonautonomously were also initiated.

The strategy followed the general scheme (*a-m* identifies with *a-m13* or *a-m16*):

$$\frac{a\text{-}m}{a\text{-}m} \times \frac{a\text{-}o}{a\text{-}o}\text{no-Uq} \rightarrow \text{spotted selections} = \frac{a\text{-}m}{a\text{-}o} \qquad \text{(Cross 2)}$$

$$\times \frac{a\text{-}o}{a\text{-}o} no\text{-}Uq$$

(a) Expectation

(*a*) 50% spotted indicates an autonomous system (the *a-m* allele always segregates with its regulatory element), which is confirmed by further testcrosses.

(*b*) 25% spotted indicates independence of a regulatory element controlling mutability of the *a-m* allele.

The *a-o* no-*Uq* testers were used in the testcrosses on the expectation that the new alleles derived by a method similar to the *a-ruq* system (Friedemann & Peterson, 1982) were responsive to *Uq*, which is pervasive in most lines. After the discovery of the pervasiveness of *Uq* regulatory elements in various lines (Peterson & Friedemann, 1983) a study of their diversity and distribution was made.

Examples of changes in state of the *a-ruq* allele are also presented to show that changes in expression of mutability reside in the receptor as well as in the regulatory elements.

(*b*) *Description of phenotype*

a-m13. The selfed ear from the *a-m13* mutant exhibited kernels with a spotted flow (Peterson, 1966) phenotype with two types of coloured spots on a colourless background – a fine pattern of 1–3 coloured aleurone cells and a larger diffuse round spot pattern of about 20–50 coloured cells. Spots are predominantly at the base of the kernel and absent at the crown (Plate 1*a*).

a-m16. The initial selections included three kernels displaying fine 1–2 coloured spots on a colourless backround. The *a-m16* mutability pattern in the selfed ear consisted of an array of round spots as well as larger, irregular, coarse spots (Plate 1*b*).

3. RESULTS

(i) a-m13: *Heritability of the mutability phenotype and establishment of a 2-element system*

Kernels showing mutability (coloured spots on a colourless background, Plate 1*a*) were planted in 1978. To determine system relationships, crosses of these spotted kernels were made with the *Dt*, *En* and *Uq* systems testers such as *a-dt*, *a-m*(*r*) and *a-ruq*, respectively. One selfed ear and outcrosses to *a-dt* and *a-m*(*r*) testers (*Dt* and *E*n testers, respectively) displayed a spotted flow phenotype (Plate 1*a*). Spotted flow selections from the selfed ear and the outcrosses were tested for autonomy or independent control of the mutability system (Fig. 1).

To test the manner of mutability control, *a-o* no *Uq* testers were crossed with plants from the spotted flow selections obtained by selfing (Fig. 1). There were two segregation patterns among the resulting ears. In one set 3 out of the resulting 17 ears showed 1/2 spotted-flow phenotype, which when tested further showed one factor segregating independently and this was controlling mutability. In the other set, the remaining 14 ears (out of 17) showed all kernels spotted flow in crosses with *a-o* no-*Uq* testers. The spotted-flow kernels from these 14 ears were further backcrossed to the *a-o* no *Uq* line and showed 1/4 spotted-flow phenotype. This is illustrated in cross 3.

$$\frac{a\text{-}m13}{a\text{-}o}\,\frac{Rg}{+} \times \frac{a\text{-}o}{a\text{-}o}\text{no Uq} \rightarrow 1/4 \text{ are } \frac{a\text{-}m13}{a\text{-}o}\,\frac{Rg}{+} \text{ spotted flow} \qquad \text{(Cross 3)}$$

This indicated that a regulatory element segregating independently of the *a* locus controls mutability of the *a-m13* allele, which responds with a spotted-flow phenotype (i.e. *a-m13* + *Rg* = spotted flow). The two successive crosses allowed the segregation of factors controlling mutability.

The outcrosses of the spotted-flow selections to an *a-dt* tester also displayed heritability of phenotype (Table 1). Testcrosses with an *a-o* no-Uq tester showed independence of the regulatory element to the *a-m13* allele, and also indicated that *a-dt* did not respond to the *a-m13* regulatory element (Table 1).

Outcrosses of the spotted *a-m13* plants ((*a-m13*/*a-dt*) (*Rg*/+) designated 2811 in

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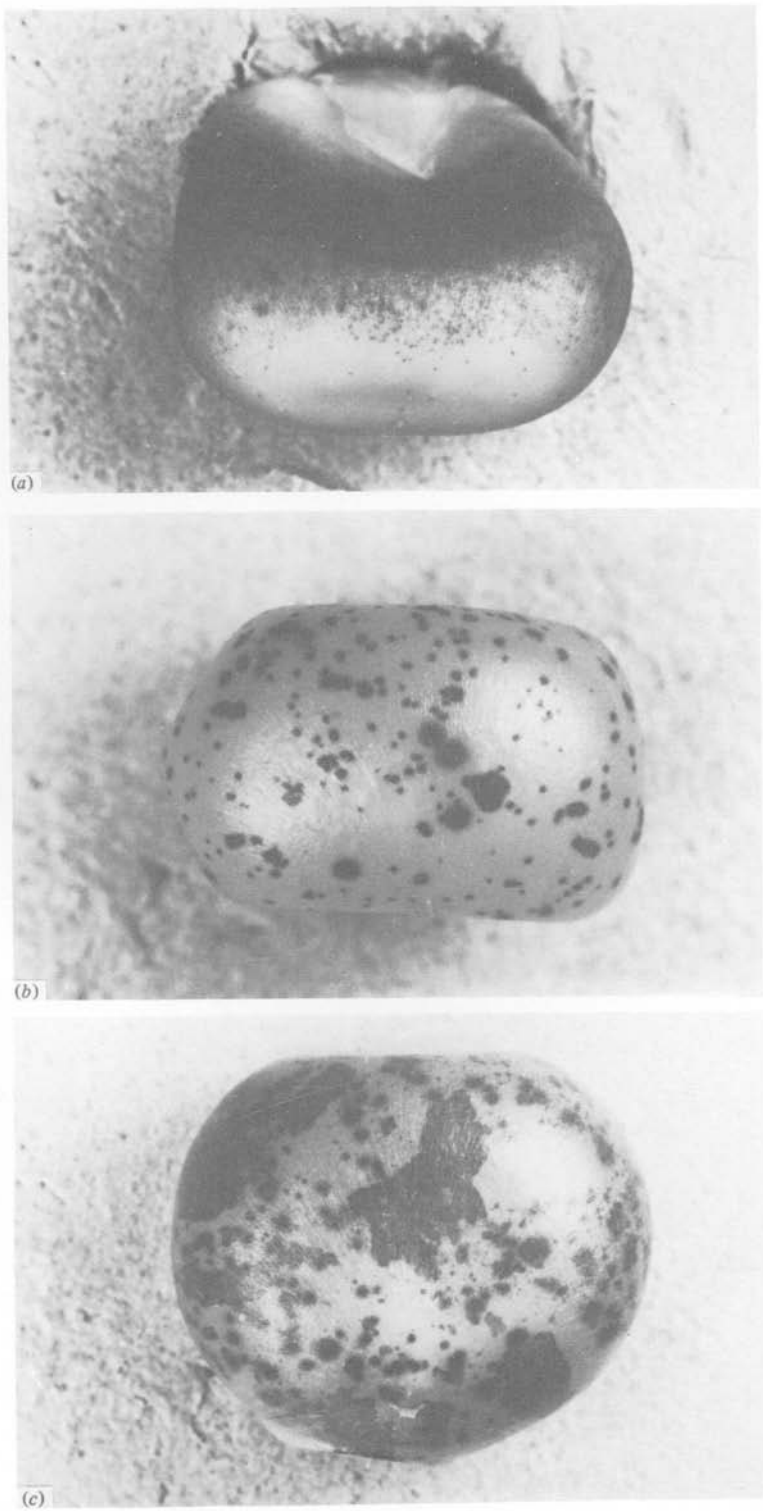

Patterns of spotting determined by the specific *Uq*-regulatory element. (*a*) The *Uq-13 a-m13* mutant displaying a spotted flow phenotype. (*b*) the *Uq-16 a-m16* mutant with an array of round and irregular spots. (*c*) *Uq* from the *Ac* tester line exhibiting very early activity (Figure 3).

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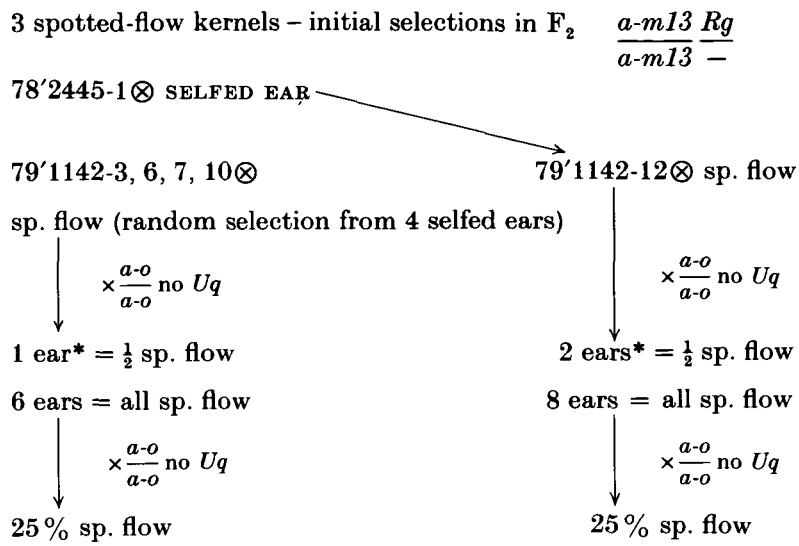

Fig. 1. Crossing scheme to illustrate independence of a regulatory element. *(1+2) three ears showing 1/2 spotted progeny out of 17 ears. ⊗ indicates selfing.

Table 1. *Progeny of the testcross of spotted-flow* a-m13 *to the* a-o *no* Uq *tester*

In the cross $\frac{a\text{-}dt\ sh2}{a\text{-}dt\ sh2} \times \frac{a\text{-}m13\ Sh2\ Rg}{a\text{-}m13\ Sh2\ Rg}$ (spotted-flow selection)

$\frac{a\text{-}m13\ Sh2\ Rg}{a\text{-}dt\ Sh2\ +}$ (spotted-flow) → ♂ on $\frac{a1\text{-}st\ Dt}{a1\text{-}st\ Dt}$

Outcross = 75 % spotted

♀ × $\frac{a\text{-}o}{a\text{-}o}$ *No Uq*

Cross '80 2811	Testcross progeny: kernel number (%) Spotted-flow (%)	Colourless (%)	Total	χ^2 (1:3)
-1/3032	58 (24·2)	182 (75·8)	240	0·088 ns
-2/3032	47 (25·8)	135 (74·2)	182	0·066 ns
-4/3034	45 (22·4)	156 (77·6)	201	0·73 ns
-8/3030	92 (25·1)	274 (74·9)	366	0·004 ns
-9/3032	90 (22·8)	303 (77·2)	393	0·92 ns
-12/3032	85 (22·6)	291 (77·4)	376	1·15 ns

ns, χ^2 not significant at 0·05 level.

Table 1) onto an *a-st Dt* line (*Dt/Dt*) used as a female displayed only 75 % spotted phenotype confirming that the *a-dt* allele was present and was proved to be responding to the *Dt* regulatory element. The results of this test, namely, the 75 % spotting, indicate that *a-dt* does not respond to the *a-m13* regulatory element but only to *Dt* in the *a-st Dt* line (Table 1).

(ii) *Relationship of the* a-m13 *controlling element system to other systems*

The specificity of a regulatory element to trigger mutability of a particular receptor element distinguishes one transposable element system from another. To test the relationship of the *a-m13* controlling element system to other known systems, the regulatory element of *a-m13* was tested for its interaction with the receptor element alleles of the known systems (*Ds*, *I*, *a-mdt*). The reciprocal test,

	Regulator							
Receptor allele	*Ac*	*En*	*Dt*	*Fcu*	*Bg*	*Uq*	*Uq-13*	*Uq-16*
C-Ds	+	−	−	−	−	−	−	−
a2-m(r)	−	+	−	−	−	−	−	−
a-m-1	−	+	−	−	−	−	−	−
a-dt	−	−	+	−	−	−	−	−
r-cu	−	−	−	+	−	−	−	−
o2-m(r)	−	−	−	−	+	−	−	−
a-ruq	−	−	−	−	−	+	+	+
a-m13	−	−	−	−	−	+	+	+
a-m16	−	−	−	−	−	+	+	+

Fig. 2. Controlling element systems: interaction of regulatory and receptor elements, (+) indicates that variegation is found (−) indicates that there is no variegation. The receptor allele is used in a tester line to test for presence of its regulatory element in a cross with a genotype to be tested.

that of the *a-m13* allele with the regulatory elements of the known systems (*Ac*, *En*, *Dt*), was undertaken to confirm the relationship. The pervasiveness of the regulatory element for *a-m13* among the standard testers required the use of testers without the segregating regulatory elements.

The results of the tests on the *a-m13* system are summarized in Fig. 2. The *a-m13* allele is triggered by a standard *Uq* of the *Uq-a-ruq* system (Friedemann & Peterson, 1982).

(iii) *Interaction of* a-m13 *with the* Uq-a-ruq *system*

For the definitive test of relationship of the *a-m13* regulatory element (*Uq-13*) with the standard *a-ruq* allele, the *Uq-13* regulatory element was extracted, independent of the *a-m13* allele and tested on an *a-ruq* line (no-*Uq*).

In the progeny of the testcross of cross 4 shown in Table 1:

$$80°2811\text{-}8/3030 = \frac{a\text{-}dt\ sh2}{a\text{-}m13\ Sh2}\ \frac{Uq\text{-}13}{+} \times \frac{a\ sh2\ +}{a\ Sh2\ +} \quad \text{(no } Uq\text{)} \qquad \text{(Cross 4)}$$

colourless shrunken selections include

$$\frac{a\text{-}dt\ sh2}{a\ sh2}\ \frac{Uq\text{-}13}{+} \quad \text{and} \quad \frac{a\text{-}dt\ sh2\ +}{a\ sh2\ +}.$$

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Table 2. *Test of the element controlling* a-m13 *mutability*

Progeny of the cross 4 $\frac{a\text{-}dt\ sh2}{a\ sh2}\frac{Uq\text{-}13}{+}$ or $\frac{+}{+}$ $\times\frac{a\text{-}ruq}{a\text{-}ruq}\frac{+}{+}\left(\frac{a\text{-}dt\ sh2}{a\ sh2}\text{derivatives of crosses in Table 1}\right)$

Cross '82 1536	Kernel no. Sp. flow	Colourless	Total	$\chi^2(1:1)$	
-1/1615	87	74	161	1·05	ns
-2/1614	0	270	270		
-3/1615	163	169	332	0·108	ns
-4/1614	0	310	310		
-5/1613	62	73	135	0·89	ns
-6/1615	0	220	220		
-7/1619	12	14	26	0·15	ns
-8/1618	0	186	186		
-9/1616	0	232	232		
-12/1614	198	178	376	1·06	ns
-13/1615	61	85	146	3·94	ns

ns, χ^2 not significant at 0·05 level.

Table 3. *Response of* a-m13 *a standard* Uq (*Cross A*) *and confirmation of* Uq (*Cross B*) *absence*

Progeny originating from the cross:

$$\frac{a\text{-}m13}{a\text{-}m13}\frac{Uq\text{-}13}{+}\times\frac{a}{a}\frac{et}{et}\frac{+}{+}(\text{no-}Uq)$$

1/2 colourless: 1/2 spotted-flow

$$\textit{colourless selections} = \frac{a\text{-}m13}{a\ et}\frac{+}{+}(\text{no-}Uq)$$

Expectation: $\frac{a\text{-}m13}{a\ et}\frac{+}{+}$

Cross A → Cross ♂ $\frac{a\text{-}o}{a\text{-}o}\frac{Uq}{Uq}$

Cross B → ♀ $\frac{a\text{-}ruq}{a\text{-}ruq}$

Progeny ears	Cross '82 4636	Spotted	Colourless	Total	$\chi^2(1:1)$
Cross A	-1/1650	189	177	366	0·39 ns
	-3/1650	240	226	466	0·14 ns
	-4/1640	229	205	434	1·33 ns
	-9/1650	124	122	246	0·016 ns
	'82				
Cross B	1617-6/4636-1	—	all	250	—
	1617-8/4636-3	—	all	226	—
	1617-3/4636-4	—	all	150	—
	1616-1/4636-9	—	all	212	—

ns, χ^2 not significant at 0·05 level.

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On the basis of an independent factor triggering *a-m13* mututability (Plate I), the colourless (nonspotted) shrunken selections are expected to have a *Uq-13* regulatory element segregating (Cross 4). In crosses of these colourless shrunken selections by an *a-ruq* tester, half the progeny ears are expected to show a spotted-flow phenotype if *a-ruq* responds to the segregating *Uq-13*. The progenies of the crosses are shown in Table 2.

Six out of eleven ears show a half-spotted flow phenotypic class (Table 2). These results confirm the hypothesis that half the colourless-shrunken genotypes had a *Uq-13* segregating to which the standard *a-ruq* responds. The variegation phenotype of spotted flow is a function of the *Uq-13* regulatory element.

Next, it was necessary to determine the phenotype elicited by the responsiveness of the *a-m13* allele to a standard *Uq*. To execute this test, *a-m13* kernels without its regulatory element *Uq-13* were selected and crossed to an *a-o Uq* tester (Table 3). In addition, crosses on an *a-ruq* line were made to confirm the absence of the *Uq-13* regulator in these colourless selections.

The crosses of these colourless selections on the *a-ruq* tester showing all colourless kernels among the progeny confirm the absence of *Uq* (Table 3B). The crosses by the *a-o Uq* tester line displaying 50% spotted kernels show that *a-m13* is triggered by the standard *Uq* in the tester line (Table 3A). Crosses of the *a-o Uq* line on *a-ruq* (not shown) confirm the presence of homozygous *Uq* in the *a-o* Uq line. The responsiveness of the *a-m13* receptor to the standard *Uq* regulatory element and the variegation interaction of the *a-m13* regulatory elements to the *a-ruq* receptor show that *a-m13* is homologous to the *Uq-a-ruq* controlling element system. Further, the regulatory element *Uq-13 is shown to determine the spotted-flow phenotype.*

(iv) a-m16, *a 2 element system with distinct dosage relationship*

The *a-m16* allele is the other allele showing variegation. To establish the heritability of the variegated kernel phenotype, plants from the three initial spotted selections were selfed and outcrossed to the various receptor lines (testers of Regulatory elements) of several transposable element systems. The spotted phenotype showed a very marked difference with the different dosages of *Uq-16*. With two doses of *Uq-16*, i.e. when used as female, a high spot frequency as well as a coarse type pattern was evident (Plate I*b*). When used as male (with one dose of *Uq-16*) a very low frequency of fine spots, usually 1–3 spots per kernel, was observed. Also, the frequency of spotted kernels observed was significantly less than expected.

Individual plants from spotted kernels arising from the selfing of the initial spotted kernels (Table 4) were crossed both as male and female: as female to an *a-o* no-*Uq* line and as male to *a-ruq* and *a-o Uq*. The crosses of the spotted selections to an *a-ruq* line (Table 4, cross c) showed a low frequency of spotted kernels (Table 4C). The testcrosses by an *a-o* no-*Uq* line, which showed 50% spotted kernels (expect an independent *Uq-16* regulatory element), were then used to test for the heritability of the variegation phenotype. The progeny from these crosses (the spotted selections by *a-o* no *Uq*, Table 4 [cross (a)]) displayed a nonsignificant

deviation from 1/4 spotted kernel phenotype indicative of an independent regulatory element (*Uq-16*) (Table 4 A).

The cross by the *Uq* line showing 50 % spotted (Table 4, cross [b]) indicates that *a-m16* is triggered by *Uq* from the *Uq* line. These same plants used in a reciprocal cross on an *a-ruq* line (Table 4, cross [c]) displayed a lower-than-expected frequency

Table 4. *Demonstration of the response of* a-m16 Uq16 *system to the standard* a-ruq Uq *two component system*

initial spotted kernels

⊗

spotted selections

$\times \delta \frac{a\text{-}o}{s}$ No *Uq* $\qquad$ $\varphi \frac{a\text{-}ruq}{s}$ = low frequency of fine low spotted kernels

$$50\,\%\text{ spotted} = \frac{a\text{-}m16}{a\text{-}o}\,\frac{Uq\text{-}16}{+} = \text{spotted selections}$$

$$\text{Cross (a)}\ \frac{a\text{-}m16}{a\text{-}o}\,\frac{Uq\text{-}16}{+} \times \frac{a\text{-}o}{a\text{-}o}\ \text{No } Uq$$

	'82	Spotted (%)	Colourless	Total	χ^2 (1:3)
(A)	1550-1/1635	108 (22·7)	367	475	ns
	1550-11/1634	67 (23)	224	291	ns

$$\text{Cross (b)}\ \frac{a\text{-}m16}{a\text{-}o}\,\frac{Uq\text{-}16}{+} \times \frac{a\text{-}o}{ao}\,\frac{Uq}{Uq}$$

	'82 1550	Spotted (%)	Colourless	Total	χ^2(1:1)
(B)	-2/1650	213 (52·6)	192	405	ns
	-3/1650	84 46·9)	95	179	ns
	-5/1650	261 (48·9)	272	533	ns
	-6/1650	272 (49·5)	277	549	ns
	-10/1650	284/(50·5)	278	462	ns

ns = χ^2 not significant at 0·05 level.

$$\text{Cross (c) Reciprocal}\ \frac{a\text{-}ruq}{a\text{-}ruq} \times \frac{a\text{-}m16}{a\text{-}o}\,\frac{Uq\text{-}16}{+}$$

	Cross 1982	Spotted Fine low (%)	Colourless	Total
(C)	1612-5/1550-2	73 (27·9)	188	261
	1612-4/1550-3	59 (21·3)	218	277
	1620-5/1550-4	60 (12·8)	410	470
	1615-2/1550-6	54 (16·2)	280	334
	1612-3/1550-7	47 (27·6)	123	170
	1619-6/1550-8	54 (22·3)	188	242

of spotted kernels based on the expectation that *a-ruq* responds to the *Uq-16* regulatory element. The spotting pattern resulting from this cross was of a very low pattern with only 2–3 fine spots.

Because of the concern for the lower-than-expected frequency of kernels with one dose of *Uq-16*, tests were made by using spotted selections from cross c

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(Table 4C) with the *a-ruq*, *a-o* no-*Uq* and *a-o Uq* tester lines. In one series of crosses from a particular plant (shown in Table 5) originating from the cross 821550-1 × 1635 illustrated in Table 4, the response of *a-ruq* to *Uq-16* is demonstrated.

Two crosses were made with each plant by utilizing the main ear and a tiller ear to procure two tests with one plant. The testcross of the plant tiller by *a sh2* no-*Uq* tester (Table 5, cross b) indicates that the *Uq-16* regulatory element assorts

Table 5. *Series of crosses demonstrating* Uq-16 *activity and dosage relations*

Spotted selections from the cross:

$$82'1550\text{-}1/1635 \rightarrow \frac{a\text{-}m16}{a\ sh2}\ \frac{Uq\text{-}16}{+} \quad \text{(Table 4A)}$$

	Cross	Spotted	Colourless round	Colourless shrunken	Ratio	χ^2
(a)	$\frac{a\text{-}m16}{a\ sh2}\ \frac{Uq\text{-}16}{+} \times \frac{a\text{-}ruq}{a\text{-}ruq} = 83'1422\text{-}13/1516$	141	132	—	1:1	ns
(b)	$\frac{a\text{-}m16}{a\ sh2}\ \frac{Uq\text{-}16}{+} \times \frac{a\ sh2}{a\ sh2}\ \frac{+}{+} = 83'1422\text{-}13t/1529$	82	90	180	1:1:2	ns
(c)	Reciprocal cross $\frac{a\ sh2}{a\ sh2}\ \frac{+}{*} \times \frac{a\text{-}m16}{a\ sh2}\ \frac{Uq\text{-}16}{+} = 83'1531/1422\text{-}13t$	4	44	57	1:1:2	**

ns, χ^2 not significant at 0·05 level; **, χ^2 significant at 0·01 level.

independently of the *a-m16* allele, based on the 1/4 spotted kernels in the progeny. In the main plant crossed by the *a-ruq* line (Table 5A) 1/2 spotted kernels are obtained, which indicates that the *a-ruq* allele responds to the segregating *Uq-16* of the maternal parent present in two doses in the aleurone. By using the same plant in a reciprocal cross, a lower-than-expected frequency of spotted kernels is obtained, and the spotting pattern consists of a few (2–3) small spots. This differential effect between the two crosses represents a *Uq* dosage effect and is similar to the latent *Uq* activity described by Peterson & Friedemann (1983).

The *a-m16* allele, isolated from the independently segregating *Uq-16* regulatory element, was tested for its response to other *Uq* sources. The variegation pattern of *a-m16* was similar to that of a standard *a-ruq* in response to the original *Uq* from the standard *Uq-a-ruq* system, and to *Uq-13* by displaying a spotted-flow pattern.

The *a-m16* system was tested for interaction with the other controlling element systems. The results are summarized in Fig. 2. All other systems besides the *Uq-a-ruq* and *a-m13 – Uq-13* systems proved negative. The *a-ruq*, *a-m13* and *a-m16* alleles responded in a similar manner to different *Uq* regulatory elements.

(*a*) *Diversity of* Uq *regulatory elements*

In continuation of studies initiated on the pervasiveness of *Uq*-regulatory elements in various tester lines, the *Uq* (Figure 3) elements were compared for their ability to trigger mutability in a standard *a-ruq* allele (Friedemann & Peterson, 1982). The spotting pattern of some *Uq* elements is very distinctive. A summary

of various *Uq* sources along with the distinctive patterns of variegation induced is given in Figure 3.

(v) *Allelic nature of various* Uq *elements*

To study the origin and lineage of different *Uq* elements, their chromosomal position in relation to each other was tested. By chromosome mapping of *Uq* elements using translocation stocks (Burnham, 1966), the *Uq* from *a et* line was

	Uq source	Description of phenotype/position
1.	*Uq-a-ruq* mutant (Sprague) (*Uq-standard*)	Fine low and coarse high with 1 or 2 doses respectively (Friedemann & Peterson, 1982).
2.	*Uq-13 a-m13* mutant	Flow type, spots at kernel base, large round + numerous very fine spots (Plate 1*a*) Independent of *Uq-standard*
3.	*Uq-16 a-m16*	Very few (2–3) spots with 1 *Uq* dose, with 2 doses fine high spotted type (Plate 1*b*)
4.	*C sh bz wx Ac* line	Coarse high, many pale sectors with spots, very early and frequent events (Plate 1*c*) Independent of *Uq-standard* and *Uq-13*.
5.	$\frac{a\ sh2}{a\ sh2}$ line	Medium spotted, not very coarse, independent of *Uq-standard*.
6.	$\frac{a\ et}{a\ et}$ line (*Uq-a et*)	Fine pattern, allelic to *Uq-standard*.
7.	$\frac{a\text{-}dt\ sh2}{a\text{-}dt\ sh2}$ line	Fine pattern, allelic to *Uq-standard*.
8.	$\frac{a\ et}{a\ et}$ line with latent *Uq* (*Uq-a et* [*low*])	Latent *Uq*, very low spotting pattern and fewer spotted kernels than expected when present in 1 dose. Linked to *Uq-13*.

Fig. 3. Response of standard *a-ruq* to various *Uq* elements.

found linked to a chromosome 2-9b *wx* translocation, and independent of other *wx* translocations. To test whether two *Uq*-elements from different sources are allelic, linked, or independent, the *Uq* elements are crossed together with *a-ruq* and testcrosses made with an *a-o* no-*Uq* tester (Table 6). The phenotypic frequencies are then tested against the hypothesis of *Uq* linkage, allelism, and independence.

To illustrate the expectation of allelic and independent *Uq* elements, the cross of *Uq-standard* from the original *Uq-a-ruq* mutant with *Uq* from *a et* tester line is shown in Table 6.

The phenotypic frequencies of the progeny are not significantly different from a 1:1 ratio of spotted:nonspotted. This indicates that the *Uq* elements from the original *a-ruq-Uq* mutant (Friedemann & Peterson, 1982) and the *a et* tester line

are on the same chromosome and are possibly at the same position or linked. Results from translocation mapping link the *Uq-a et* to the 2-9b translocation and independent of other *wx* translocations. *Uq-a et* and *Uq-standard* are, therefore, possibly located on chromosome 2. In similar tests of *Uq* from the original *a-ruq-Uq* mutant and *Uq* from *a-o sh2* tester, the two *Uq* elements showed independence, i.e. on different chromosomes or at more than 50 map unit distance.

Table 6. *Test of alleles in* Uq *elements from different sources* (a et *sources and the original standard*)

Source: *a et* tester, original AR line

Cross: $\frac{a\ et}{a\ et}\ \frac{Uq\text{-}a\ et}{Uq\text{-}a\ et} \times \frac{a\text{-}ruq}{a\text{-}ruq}\ \frac{Uq\text{-}standard}{Uq\text{-}standard}$

spotted selections $\frac{a\text{-}ruq}{a\ et}\ \frac{Uq\text{-}a\ et}{Uq\text{-}standard}$ or $\frac{a\text{-}ruq}{a\ et}\ \frac{Uq\text{-}a\ et}{+}\ \frac{Uq\text{-}standard}{+}$

= allelic *Uq* elements = independent *Uq* elements

Expectation in cross × $\frac{a\text{-}o}{a\text{-}o}\ \frac{+}{+}$ (no *Uq*) × $\frac{a\text{-}o}{a\text{-}o}\ \frac{+}{+}$

50 % spotted kernels 37·5 % spotted kernels

Progeny of cross: spotted selections × $\frac{a\text{-}o}{a\text{-}o}\ \frac{+}{+}$ no *Uq*.

	Kernel no. (%)			
Cross: 1980	Spotted (%)	Colourless	Total	χ^2 (1:1)
1043-5/1136	352 (53·3)	308	660	ns
1043-8/1143-9	186 (51·9)	172	358	ns
1044-1/1146-6	114 (52·7)	102	216	ns
1044-4/1144-8	145 (45·7)	172	317	ns
1044-5/1146-1	61 (48·4)	65	126	ns
1044-6/1146-8	203 (55·7)	161	364	ns
1044-7/1147-6	280 (51·3)	266	546	ns
1044-9/1146/13	239 (53·6)	207	446	ns
1044-10/1151-12	114 (47·9)	124	238	ns

Other tests showed that another *Uq* from an *Ac* line (very coarse spots with pale sectors) is not allelic to *Uq-standard* or *Uq-13*. In the critical cross shown in Table 7, the progenies of cross (a) show phenotypic frequencies not significantly different from a 3:1 of spotted:nonspotted class. This demonstrates that the *Uq* from the *Ac* line is not allelic or not on the same chromosomal position as *Uq-standard* from the original *a-ruq-Uq* mutant obtained from Sprague's WSMV treated plant progeny.

Cross b in Table 7 shows that the *Uq* from the *Ac* line is not allelic to *Uq-13*, which determines the spotted-flow phenotype. Similarly, cross c in Table 7 shows that spotted:colourless phenotypic frequency is significantly different from a 1:1 ratio. Therefore, *Uq-13* is not allelic to *Uq-a et*, which has been shown previously (Table 6) to be allelic to *Uq-standard* (located on chromosome 2).

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The diversity of two closely linked *Uq* elements showing distinctly different phenotypic spotting patterns is illustrated in the flow diagram (Figure 4) and the cross shown in Table 8. The critical genotype, a heterozygote of *Uq-13* and *Uq-a et* (low) (Figure 3), is backcrossed to the *a-ruq* and *a-o* no *Uq* lines.

Table 7. *Progeny of critical cross to illustrate independent locations of* Uq *elements*

Cross (a) $\frac{a\text{-}ruq}{a\text{-}ruq}\ \frac{Uq\text{-}Ac\ line}{+}\ \frac{Uq\text{-}standard}{+} \times \frac{a\text{-}o}{a\text{-}o}\ \frac{+}{+}$ (no *Uq*)

	Kernel no.				
1982	Spotted coarse	Spotted low	Colourless	Total	% spotted
1445-2/1537-3	138	53	67	258	74
1445-5/1537-2	124	60	59	243	75·7
1445-8/1537-5	233	126	90	449	71·9

Cross (b) $\frac{a\text{-}ruq}{a\text{-}m13}\ \frac{Uq\text{-}Ac\ line}{+}\ \frac{Uq\text{-}13}{+} \times \frac{a\text{-}o}{a\text{-}o}\ \frac{+}{+}$ (no *Uq*)

	Kernel no.				
1983	Spotted coarse	Spotted flow	Colourless	Total	% spotted
1446-1/1535-7	295	118	148	561	73·6
1446-2/1535-15	175	64	64	303	78·8
1446-5/1536-4	189	82	70	341	79·4

Parents: $\frac{a\text{-}m13}{a\text{-}m13}\ \frac{Uq\text{-}13}{Uq\text{-}13} \times \frac{a\ et}{a\ et}\ \frac{Uq}{+}$

Cross (c) $\frac{a\text{-}m13}{a\ et}\ \frac{Uq\text{-}13}{+}\ \frac{Uq\text{-}a\ et}{+} \times \frac{a\text{-}o}{a\text{-}o}\ \frac{+}{+}$ (no *Uq*)

1982	Spotted flow (%)	Spotted high	Colourless	Total	Total sp (%)	χ^2 (1:1)
1532-2/1626-10	61 (17·4)	46 (13·1)	244	351	33·3	**
1532-3/1626	71 (19·6)	83 (22·9)	208	362	42·5	**
1532-4/1626	56 (19·1)	46 (15·6)	191	293	34·7	**
1532-10/1627	112 (20·5)	40 (7·3)	394	546	27·8	**

**, χ^2 test for 1:1 ratio of spotted: nonspotted significant at 1% level.

The colourless class (Table 8) is obtained by crossovers between the *Uq-a et* (*low*) and *Uq-13* (spotted flow) (see Figure 3 for the descriptions). The map distance between the two *Uq* elements calculated by doubling the colourless class frequency is 12·2 map units.

(vi) *Changes in state of* ruq *elements*

From the original *a-ruq-Uq* mutant (Peterson & Friedemann, 1982) a low activity state of the *a-ruq* allele was recovered. The *a-ruq*(*10*) allele was tested with a standard *a sh2 Uq* line. The results of the cross are shown in Table 9(A). The lower frequency of spotted kernels with a very low spotting pattern was indicative

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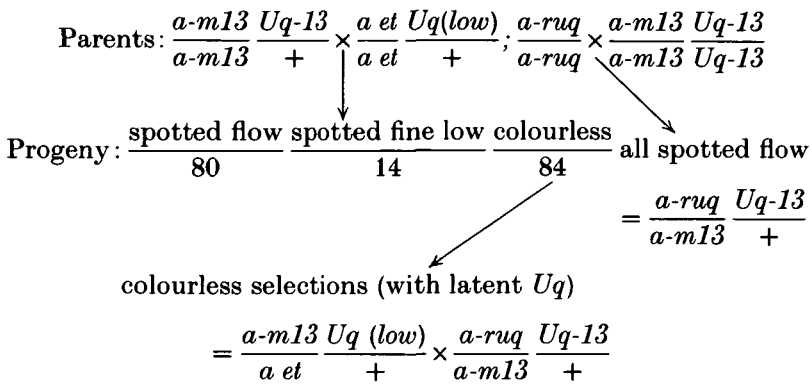

progeny phenotypic frequency

$$\frac{\text{spotted irregular}}{211}\quad\frac{\text{spotted flow}}{93}\quad\frac{\text{colourless}}{131}$$

Spotted irregular with fine background spotted selections = selections for *Uq* (*low*) and *Uq-13* (flow with fine background spots)

$$\frac{a\text{-}ruq}{a\text{-}m13}\ \frac{Uq\ (low)+}{+\ Uq\text{-}13}\ \{\text{linkage in trans}\}$$

Fig. 4. Flow diagram of cross showing *Uq* linkage.

Table 8. *The diversity of two closely linked* Uq *elements illustrated from the cross*

Progeny of cross (a) $\frac{a\text{-}ruq}{a\text{-}m13}\ \frac{Uq\text{-}low+}{+\ Uq\text{-}13}\ \frac{a\text{-}ruq}{s}\ \frac{+}{+}$

1983	Spotted irregular	Spotted flow	Colourless	Total	% colourless
1444-6/1519	197	186	28	411	6·8
1444-12/1517	166	160	28	354	7·9
1444-13t/1519	214	167	22	403	5·4

Progeny of cross (b) $\frac{a\text{-}ruq}{a\text{-}m13}\ \frac{Uq\ (low)+}{+\ Uq\text{-}13} \times \frac{a\text{-}o}{a\text{-}o}\ \frac{+}{+}$ (no *Uq*)

1983	Spotted irregular	Spotted flow	Colourless	Total	% colourless
1444-3/1536-3	213	188	18	419	4·3
1444-4/1539-10	147	160	25	332	7·5
1444-8/1539-9	72	59	3	134	2·2
1444-13/1536-7	178	160	23	361	6·3
			147	2414	6·09

Progeny of a cross of two *Uq* elements linked in trans onto an *a-ruq* line and to an *a-o* no *Uq* line.

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that the *a-ruq(10)* allele was not triggered frequently. A progeny test of spotted selections is shown summarized in Table 9(B), in crosses to *Uq-13* and *Uq-16* sources. The response to these *Uq* sources is also a low frequency of spotted kernels and a low spotted pattern in ten ears. One exceptional ear shows 25% spotted kernels, which would be expected if the *a-ruq(lo)* allele state changed to the standard *a-ruq* active state. These series of crosses demonstrate that the receptor

Table 9(A). *Response of* a-ruq (lo) *allele to standard* Uq

Cross	Round: Spotted low	Round: Colourless	Round: Shrunken: colourless
82'4626-1/1650 = $\frac{a\text{-}ruq\ (lo)}{a\ sh2} \times \frac{a\ sh2\ Uq}{a\ sh2\ Uq}$	11 (4·3)	120	126

Table 9 (B). *Progeny of spotted low selections (from* 82'4626-1/1650) *in crosses to* Uq-16 *and* Uq-13

Spotted low selections	Number of ears	Spotted kernels (%)
$\frac{a\text{-}ruq(lo)}{a\ sh2}\frac{Uq}{+} \times \frac{a\text{-}o}{a\text{-}o}\frac{Uq\text{-}16}{+}$	8	5
	1	25
$\times \frac{a\text{-}o}{a\text{-}o}\frac{Uq\text{-}13}{Uq\text{-}13}$	2	5
Total	11	

component of the *a-ruq* allele has changed from a high-activity to a low-activity state and back to the high-activity state again. Other derivatives of *a-ruq* that include a high-activity state have also been recovered, which show a very high rate of germinal changes to nonresponsive pale and colourless alleles in the presence of *Uq-standard*.

4. DISCUSSION

Unstable mutants at the *A* locus, derived from Aberrant Ratio lines induced by treatment of two different RNA plant viruses, Wheat Streak Mosaic Virus (WSMV) and Barley Striped Mosaic Virus (BSMV), induced lines yielding the *a-ruq*, *a-m13* and *a-m16* mutants. These three mutable alleles, each independently derived (with separate pedigrees) from the Aberrant Ratio lines some 5–12 generations after the initial induction of Aberrant Ratio after virus treatment, are triggered by a *Uq*-regulatory element segregating independently and thus belonging to the *Uq-a-ruq* transposable element system previously defined (Friedemann & Peterson, 1982). The involvement of two different RNA plant viruses, both inducing Aberrant Ratio, and subsequently inducing transposable element mutations at the *A* locus characterized genetically as *ruq* elements under the control of *Uq* regulatory elements, might indicate a common phenomenology of transposable element mutation induction in the Aberrant Ratio lines.

The specific *Uq* regulatory element that corresponds to each of the three mutants

determines the mutable phenotype of each mutant. *Uq*-13 triggers a phenotype identified as spotted-flow and *Uq-16* a spotted pattern with a lower frequency of spots than the standard *Uq* (Friedemann and Peterson), in each of the three unstable alleles *a-ruq*, *a-m13* and *a-m16*. The three mutants are each nonautonomous or two-element systems, comprising a *ruq* receptor and *Uq* regulatory element. Thus far, it has not been possible to find an autonomous *Uq* controlled system. As outlined by Peterson (1985) in the accompanying paper, *Uq* was present in one of the original parents in the initial cross of tester parent crossed by the symptom-expressing treated plant. Thus, it is axiomatic that the treatment enhanced the movement of the active *Uq* to the *A* locus in succeeding progenies of these initial parents because these spotted kernels were not observed in the F_1 (Sprague, personal communication). This suggests that the virus infection induces Aberrant Ratio phenomenon by a mechanism presently not understood, which subsequently increases the *ruq* element insertions at the *A* locus.

The diversity of the different *Uq* regulatory elements was followed to give a clue to their origin, in assorted tester lines and the original *ruq* mutants. The standard *Uq* from the *Uq-a-ruq* mutant is not allelic to the *Uq-13*, which determines a flow phenotype, and both show independent assortment with the *Uq* from the *Ac* line, which determines a very coarse-spotted phenotype (Fig. 1*c*). This indicates independent origins of the *a-ruq* and *a-m13* mutants, and mobility of the *Uq* element to different chromosomal positions, which are maintained in diverse tester lines. Linkage of the two *Uq* elements, each with a characteristically different phenotype, indicates transposition to a position on the same chromosome, with a simultaneous change in state determining a different pattern of mutability. The change in state of the *ruq* element has been observed from a normal *ruq* to a *ruq*(*lo*) state characterized by a lower frequency of spots per kernel as well as a lower number of spotted kernels than expected. The reversion of *ruq*(*lo*) state to the normal *ruq* state was also observed, signifying the response of the *ruq* element to the *Uq* regulatory element by a change of state.

Aberrant Ratio lines with segregating *Uq* elements could be utilized for transposable element mutagenesis of the *Uq-ruq* system. In normal non-Aberrant Ratio lines (without virus treatment) segregating for *Uq*, no *ruq* element mutations have been observed, indicating the necessity of using Aberrant Ratio lines, though the control crosses of non-Aberrant Ratio lines resulting from virus treatment have not been tested for such mutant inductions. The molecular isolation of the *A1* gene (O'Rielley *et al.* 1985) offers the opportunity of studying the nature of the *ruq* insertions recovered from the aberrant ratio lines.

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