Genet. Res., Camb. (1985), **46**, *pp.* 207–217
With 1 text figure
Printed in Great Britain

Virus-induced mutations in maize: on the nature of stress-induction of unstable loci*

By PETER A. PETERSON

Department of Agronomy, Iowa State University, Ames, IA 50011

(*Received 18 March 1985*)

SUMMARY

Unstable alleles, broken chromosomes and stable mutants have arisen in maize out of infected plants of Barley Striped Mosaic Virus and other viruses. Surprisingly, these same events have appeared out of progenies of these infected plants that themselves do not show any infection. These mutants showing instability have resulted from insertions that are not necessarily related. Two of these insertions (BS1 and TZ86) that have been analysed molecularly have the general characteristics of maize insertions with terminal inverted repeats and host duplication at the terminus of the transposon. In other experiments three of the unstable alleles at the *a* locus in maize (*A* locus, chromosome 3, short arm; one of genes for anthocyanin control) that arose in derivative lines of the initially treated plants are responsive to a transposable element, the *Uq* element. It was determined that the *Uq* element was not present in this initially treated plant but was present in the untreated female plant. It is proposed that the initial treatment induced events that in turn led to the mobilization of elements and that these events continue to occur in later generations. It seems that genomic events once initiated such as mobility of elements cannot be terminated despite a discontinuation of the treatment (virus) and, like a Frankenstein monster, is not responsive to its maker.

1. INTRODUCTION

Plant viruses have been identified as genome-disturbing agents. According to Brakke (1984) who has reviewed the case of transmitted virus effects on plants, research in this area began with McKinney while in graduate school in Wisconsin between 1919 and 1926. McKinney recognized differences among the yellow spots on tobacco leaves and believed that this was an expression of virus changes. In a later pursuit of this plant-virus interaction, McKinney along with Sprague and collaborators (Sprague, McKinney & Greeley, 1963), identified and provided support for a phenomenon showing deviations from expected segregation patterns identified as aberrant ratio (AR) as well as the induction of mutations after treatment of maize seedlings with Barley Stripe Mosaic Virus (BSMV). These

* Journal Paper no. J-11795 of the Iowa Agriculture and Home Economics Experiment Station, Ames, Project no. 2381.

viruses have been analysed and their relation to induced mutants considered (Jackson *et al.* 1983; Wienand, Peterson & Saedler, 1983).

After these reports (Sprague & McKinney, 1966, 1971), two different laboratories examined the AR phenomena. Samson, Brakke & Compton (1979) confirmed the deviation from the expected 3 : 1 ratio (dominant : recessive) among kernels on F2 ears of AR stocks. These authors suggested that these ratios could be accounted for by the segregation of recessive alleles complementary for colour (i.e. *AaCc* × *aacc* or *aaCc* instead of *AaCC* × *aaCC* whereby *A* and *C* are needed for colour). In further support of this thesis, Brakke, Sampson & Compton (1981) and Nelson (1981) identified the segregation of recessive alleles in AR-derived lines (*c*, *r*, Brakke *et al.*; *c*, *c2*, Nelson), which would account for the general excess of recessive phenotypes as a basis for the AR deviations.

If the AR phenomena can be accounted for by the segregation of epistatic loci, a question arises as to the origin of these mutations that gave rise to recessive alleles. From their initial report Sprague *et al.* (1963) recognized a number of seedling and endosperm mutants newly arising in their lines derived from originally infected plants.

A key feature of the BSMV-infected plants is the persistence of genome disturbance among succeeding generations of healthy plants derived from originally infected plants. This became evident with the origin, several generations after the initial treatment, of unstable mutant alleles at the *a* locus (Friedemann & Peterson, 1982; Peterson & Friedemann, 1983; and the companion paper, Pereira and Peterson).

In attempts to further examine the mutagenicity of the BSMV infection, (Mottinger 1982*a*, *b*), Mottinger *et al.* (1984*b*) induced mutants in the *Adh* and *Sh* loci. In subsequent analysis of an *Adh1* derived mutant, an insert was found and identified as *BS1*. This element has been described by Johns *et al.* and Freeling (Freeling, 1984; Johns, Mottinger & Freeling, 1985) as having a 304 bp direct repeat and occurs as a low copy number transposon. There are only 1–5 copies of *BS1* sequences in maize and teosinte. Much of the interior portion is variable according to restriction site analysis, but one segment of this interior of *BS1* is conserved.

Further support for this present genome disturbing feature of AR stocks has been obtained by the isolation and molecular characterization of seven shrunken and one bronze mutant (Mottinger *et al.* 1984*a*). A number of the mutants, in addition to expressing marked stunting and decreased vigor, exhibit reversions and altered intermediate phenotypes.

In the BSMV-induced *sh* mutant, *sh 5586*, with a 3·6 kb insertion (Mottinger *et al.* 1984*a*), the ends are also complicated instead of simply having the traditionally found simple inverted repeats (Dellaporta, personal communication). But, like the other molecularly analysed insertion-host junction sequences, a 10 bp duplication is found at the termini of the element, though only once in the wild type.

The purpose of this paper is to present some ideas on the sequence of events that help explain the BSMV induction of unstable mutants, at least for those at the *a* locus. Support and elaboration of these concepts is given in the following paper (Pereira & Peterson, 1985).

2. RESULTS

(ii) *Sprague's original crosses*

The basic pattern in the Sprague–McKinney crossing program in the course of their virus treatments was the cross of the multiple recessive stock (*a1 su pr wx*) as a female by the virus treated male that was a multiply marked dominant stock (*A1 Su Pr Wx*) (Cross 1). (*A-a*, presence or absence of aleurone anthocyanin colour; *Su-su*, starchy or sugary seeds; *Pr-pr*, purple or red aleurone colour; *Wx-wx*, starch staining blue or red with iodine) (Sprague & McKinney, 1966). The derivatives of these parents originally used by Sprague and McKinney and their genotypes are described in Tables 1 and 2.

$$a\ su\ pr\ wx \times A\ Su\ Pr\ Wx. \qquad \text{(Cross 1)}$$
$$\text{F1}$$

The only colour allele that was recessive in this cross was the *a* allele. In subsequent progenies from these crosses other colour controlling alleles including *c1* and *c2* (Nelson, 1981) and *c1* and *r* (Samson *et al.* 1979; Brakke *et al.* 1981) were uncovered. In both investigations, it was proposed that the segregation of alleles at a second locus in addition to the *a* locus (from the original cross) could explain the excess of colourless kernels coming from crosses in Aberrant Ratio (AR) lines (to be further considered).

In other progenies, unstable *a1* alleles were isolated. These were three cases that appeared independently and proved to be *a-ruq* alleles responsive to the transposable element, *Uq* (i.e. that have a receptor element at the *a* locus that is responsive to the action of *Uq*) (Friedemann & Peterson, 1982; Pereira & Peterson, 1984).

These mutant alleles only appeared in derivative progenies after several generations of crossing (Fig. 1). It was therefore significant to determine the *Uq* content of the several lines and derivatives in these crosses.

(ii) *Distribution of* Uq

(*a*) *The original parents*

The presence of *Uq* among the original parents (Cross 1) was tested by crossing these parental lines to a tester for *Uq*, the *a-ruq* line. The cross (Cross 1) of the male parent used by Sprague and McKinney as a multiply marked male (*A*, *Su*, *Pr*, *Wx*) by the *a-ruq* line yielded coloured progeny verifying the *A/A* content of the male parent. Five F1 progenies of this cross were again crossed by the *a-ruq* line, and these yielded coloured and colourless progenies (Table 2, 1982, 3601). The colourless segregants were without spots. This result verifies that *Uq* was not present among this limited sample of the 'male' line.

In four crosses of the multiply marked 'female' parent, (*a su pr y wx*) by the *a-ruq* line, all the progenies included kernels with spots (Table 2, 1982, 3602). The appearance of nearly 100% spotted kernels among these progenies would support the contention that *Uq* was present in these lines at least in the homozygous condition, or that there were many copies of *Uq*. The presence of *Uq* in each of the plants used also supports the pervasiveness of *Uq* in this line.

Table 1. *Pedigree history and sources of lines used and the genotypes significant to this study*

1980g		Source	Genotype	Phenotype	Reference
486	Ne	23265-1	*C2/C2 A/A*	Coloured *k*	Nelson, 1981 (Table 3)
487	Br	A4 × a16	*C/c R/R × R/r c/c*	Colourless *k*	Samson *et al.* 1979 Brakke *et al.* 1981
488	P''	19-1⊗†	*a sh2/a sh2 Uq/Uq*	Colourless, shrunken	Friedemann & Peterson, 1
489	P''	09⊗	*a-ruq/a-ruq*	Colourless, round	Friedemann & Peterson, 1
1981					
4903	80	5 × 488B-4	*c2/c2 A/A × a sh2/a sh2 Uq/Uq*	Coloured, round	
4904	'79	-5⊗	*a-ruq/a-ruq*	Colourless, round	
4905	80	6 × 489a-5	*C2/c2 A/A Uq/Uq × a-ruq/a-ruq*	Coloured, round	
4906	80	7 × 489A-2	*C2/c2 A/A Uq/Uq × a-ruq/a-ruq*	Coloured, round	
4907	80	3 × 489-3	*a a c1/c1 R1 × a-ruq/a-ruq*	Colourless, round	
4908	80	9 × 489B-1	*a a c1/c1 R1 × a sh2/a sh2 Uq/Uq*	Colourless, round	
1755			*a-ruq/a-ruq*	Colourless, round	

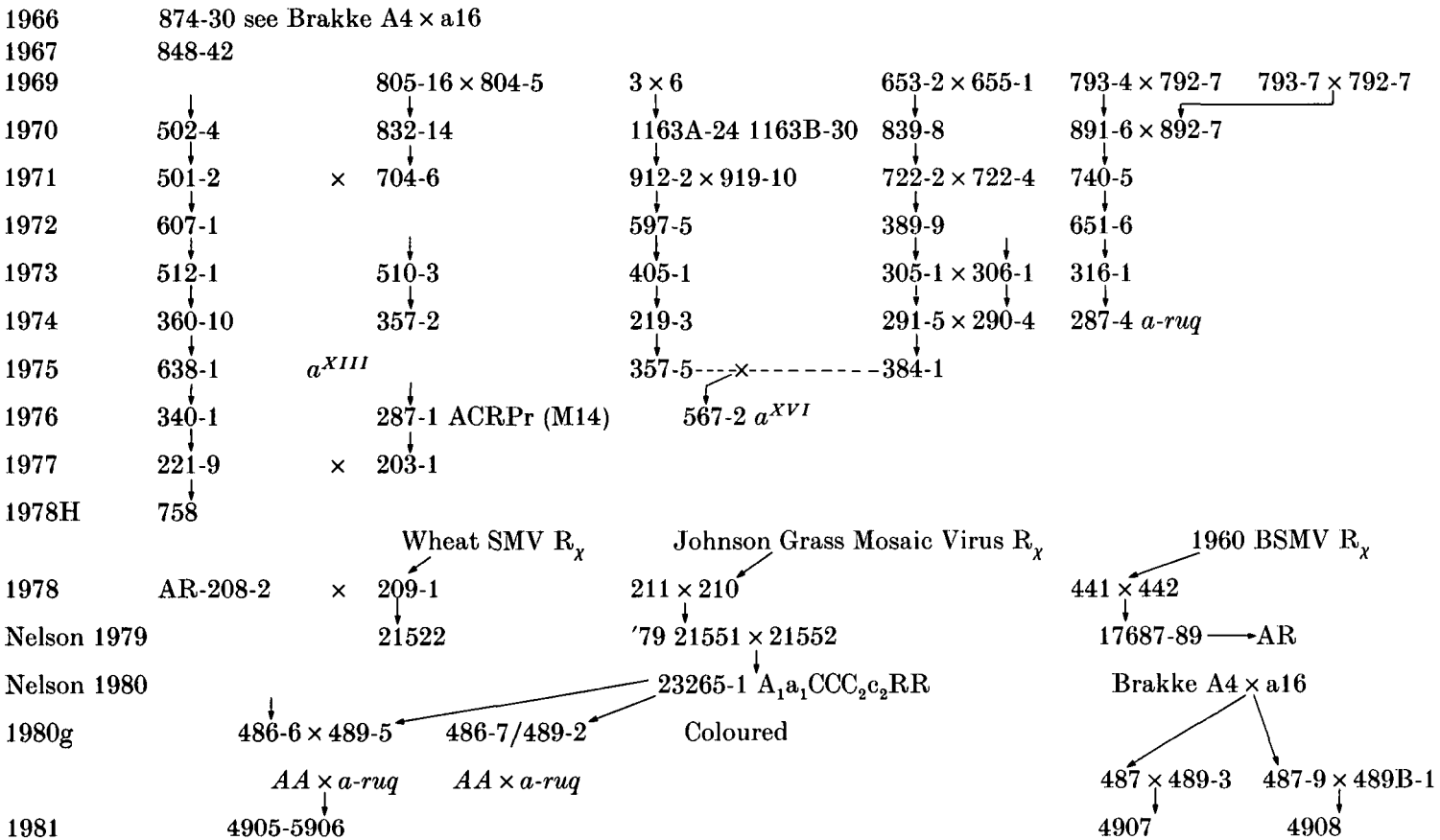
† ⊗ indicates selfing.

Table 2. *...bution of* Uq *among original parents used in Spragues original crosses used in virus treatments (see ... for Pedigree history of Sprague's lines)*

			Pedigree number	Female		Male		No. of crosses		eny
				Genotype	Phenotype	Genotype	Phenotype			
1982	36	03	Sprague 81 : 353	*A C R B Pl Pr Su Wx*	Coloured	*a-ruq*	Colourless	5*	Co	*
1982	36	03	Sprague '81 : 351	*a su pr y wx*	Colourless	*a-ruq*	Colourless	4	Sp	

* 5 progenies tested in self – none with spots.

Figure 1. Pedigree sequence of G. F. Sprague's lines that gave rise to three unstable *a* alleles (*a-ruq*; *a-ruq XIII*, *a-ruq XVI*) following virus treatments. †



† The derivation of the Aberrant ratio lines derived from Dr G. F. Sprague and sent to Dr Nelson and Brakke and to this laboratory. The origin of the three *a-ruq* alleles occurred a number of generations after the original virus treatment. See the origin of a-XIII, a-XVI and *a-ruq*. Pedigrees are referable to Nelson, 1981, Table 2 and Brakke *et al.* 1981.

In the c2 *derived lines.* The *c2* mutant identified by Nelson (1981) among Sprague's 'AR' lines was tested for the presence of *Uq*. In the cross of Nelson's *c2* progeny (Table, 1, Nelson 23265-1) by the *a-ruq* line (Table 1, 1980 g 486 × 489), all the progeny were full-coloured, verifying the *A/A* content of the Nelson *c2* line. In crosses of these 2 F1's × *a-ruq* (Table 3, 1981 4905, 4906), each of the progenies of this backcross included spotted kernels. This indicates that *Uq* was present in the homozygous condition (or there are many copies) in the *c2* lines identified by Nelson (Table 3). Further, the pattern phenotype of this spotting was a 'flow' type (Peterson, 1966).

Table 3. *Test for the presence of* Uq *in designated lines from successive progenies of Sprague's original cross of* aa A2A2 CC RR su su wx wx × AA A2A2 CC RR Su Su Wx Wx

(See Fig. 1 for the pedigree history of this cross: Nelson *C2C2 AA Uq-flow/Uq-flow* × *C2C2 a-ruq/a-ruq* (80g 486 × 489) → Coloured 814905. 814904 is *a-ruq/a-ruq*.)

1981	Spots†	Phenotype	4905 Genotype
4905-1/4904	+	flow	*Aa*
4905-2/4904	+	flow	*Aa*
4905-3/4904	+	flow	*Aa*
4905-4/4904	+	flow	*Aa*
4905-5/4904	+	flow	*Aa*
4905-6/4904	+	flow	*Aa*
4905-7/4904	+	flow	*Aa*
4905-8/4904	+	flow	*Aa*
Total	8/8	—	8/8
4906-2/4904	+	flow	*Aa*
4906-4/4904	+	flow	*Aa*
4906-5/4904	+	flow	*Aa*
4906-6/4904	+	flow	*Aa*
4906-7/4904	+	flow	*Aa*
4906-8/4904	+	flow	*Aa*
4906-21/4904	+	flow	*Aa*
Total	7/7	—	7/7

† +, spots.

In the c *lines* (*Brakke*). In similar crosses of Brakke's lines by *a-ruq* that were also progenies out of Sprague's 'AR' lines (Table 4, 4907), *Uq* was found in three of the six progenies (Table 4). In these lines, *Uq* was present in the heterozygous condition because only half of the progenies in this limited sample showed the presence of *Uq*. The *Uq* gave a 'flow' pattern similar to, but not as striking as, that in the *c2* derived lines. In all tests for *Uq*, a common *a-ruq* line was used, and thus, any differences in patterns are assignable to *Uq*. The other tested plant from the Brakke lines lacked *Uq* (Table 4, 4908).

(*b*) *The* c2 *allele, a test of its response to* Uq

Because the three tested *a*-unstable mutants (there were others) that originated in these AR lines were responsive to *Uq*, crosses were made of this *c2* allele (Table

Table 4. *Tests for the presence of* Uq *in Abnormal Ratio lines*

(Brakke (80g 487, Table 1) *C1 C1 R* − × *a-ruq/a-ruq* → Coloured, round 81 4807, 4808 × *a-ruq/a-ruq* (4904).)

1981	Spots
4907-3t × 4904	−
-4 × 4904	−
-5⊗†	+ slight flow
-6 × 4904	+ slight flow
-7 × 4904	−
-9 × 4904	+ slight flow
4908-1-1⊗	−
-2/4904	−
-3⊗	−
-4⊗	−
-5⊗	−
-6⊗	−

−, no spots.
+, ⊗ indicates selfing.

Table 5. *Test of the response of mutant alleles originating in BSMV infected plants or their progeny to* Uq

(The cross: *C2/c2 A/a* × *a sh2/a sh2 Uq Uq* → Coloured kernels 1981 4903. 1755 is *a-ruq/a-ruq*.)

	Presence of spots*		Segregation†		
		Cross × *a-ruq*	Round		
1981	Selfs	↓	Coloured	Colourless	Shrunken: colourless
4903-1⊗‡	0	.	110	38	48
-2/1755	.	+ flow	.	.	.
-2/on 1755	.	+ flow	.	.	.
-3⊗	0	.	.	.	.
-3t⊗	0	.	.	.	.
-4/1755	.	+ flow	.	.	.
-5⊗	0	.	209	51	76
-5t⊗	0	.	125	45	48

* + indicates the presence of spots.
† These 9 : 3 : 4 ratios are consistent with a tested *A Sh/a sh C2/c2* genotype.
‡ ⊗ indicates selfing.

3, 814905) with *Uq*-containing lines to test the responsiveness of *c2*. (At the time the crosses were initiated, it was not known that a *Uq-flow* was present in these lines as determined by the test of *c2* × *a-ruq*). Nevertheless, F2 progenies were obtained and none showed *c2* instability (Table 5, selfs). To verify that *Uq* was present in these F1 plants, two of the F1 sibs were crossed with the *Uq* tester (*a-ruq*), and spots were present on the kernels in each instance, verifying the presence of *Uq* (Table 5, crosses with 1755). The segregation ratios of the selfs verify the segregation of both the *c2* and *a* (linked to the closely associated *sh2*) alleles in a 9 : 3 : 4 ratio (Table 5).

https://doi.org/10.1017/S0016672300022667 Published online by Cambridge University Press

(*c*) *Allelism between* Uq-flow *and* Uq-st

There is a distinct and readily distinguishable phenotypic pattern elicited by the two *Uq* alleles, *Uq-flow* originating from the *c2* lines (Table 1, 81 4905, 4906 and Tables 3 and 4) and *Uq-st* (Friedemann & Peterson, 1982). In crosses of the *Uq* heterozygotes *Uq-c2/Uq-st* onto an *a-ruq* (Cross 2),

$$\frac{a\text{-}ruq}{a\text{-}ruq}(\text{no } Uq) \times \frac{A1\ Sh2}{a1\ sh2}\ \frac{Uq\text{-}c2}{Uq\text{-}st} \qquad \text{(Cross 2)}$$

the resulting progeny illustrate a close linkage of the two *Uq*'s. The expectations of the alternative of the independence of the two *Uq*'s are not compatible with these results (Table 6). More correctly, the cross should be written as in Cross 3.

$$\frac{a\text{-}ruq}{a\text{-}ruq}(\text{no } Uq) \times \frac{A1\ Sh2}{a1\ Sh2}\ \frac{Uq\text{-}c2+}{+\,Uq\text{-}st}. \qquad \text{(Cross 3)}$$

Table 6. *Relation of* Uq-flow (c2 *source*) *to* Uq-st (a-ruq *source*)*: Linkage of* Uq-c2 *and* Uq-st

The cross: Coloured [*C2c2 A1a1*]

$$\times\ Uq\text{-}c2 \times \frac{a1\ sh2\ Uq\text{-}st}{a1\ sh2\ Uq\text{-}st}$$

$$\downarrow$$

$$\frac{a\text{-}ruq}{a\text{-}ruq} \times \text{Coloured}\ \frac{A\ Sh\ Uq\text{-}c2}{a\ sh\ Uq\text{-}st}$$

$$\downarrow$$

	Flow	Spotted (General + crown)	Flow + crown-general	cl.	T	Recomb
Exp. if indep.	22	20	1	4	47	10·6 %
	12	12	12	12	—	—

cl, colourless, no spots.

3. DISCUSSION

The induction of virus infection (BSMV, WSMV or Johnson Grass MV) in plants to induce mutation such as initiated by Sprague *et al.* (1963) is effective in the induction of a number of events, including mutations, the 'AR' phenomena (Sprague and McKinney, 1966, 1971), chromosome breakage (Sprague *et al.* 1963; Mottinger *et al.* 1984*a*), and transposon movement (Friedemann & Peterson, 1982; Delaporta *et al.* 1984, personal communication; Johns *et al.* 1985).

The 'AR' phenomena ('Aberrant Ratio') that describes aberrant segregation patterns in conventional backcrosses (Sprague & McKinney, 1966, 1971) has been demonstrated by two independent investigators to be due to the presence of recessive mutations (likely induced by the virus treatment). These mutants are epistatic to the colour alleles used in the backcrosses utilized by Sprague & McKinney (1966, 1971) and thus lead to a deviation from the expected Mendelian segregation (Nelson, 1981; Brakke *et al.* 1981). These investigators identified these loci that had mutated (*c1*, *c2* and *r*). It is obvious that these mutants are recently induced because the original parents in the cross (Cross 1) did not harbour these

alleles since a direct cause and effect with the virus treatment is not possible. The presence of these loci in homozygous or heterozygous condition would distort the expected ratios based on a single locus such as $Aa \times aa$.

The induction of one *Adh-1* mutant by pollen from infected plants with BSMV has uncovered a mutant with an insert identified as *BS-1* (Johns *et al.* 1985). This insert occurs in low copy number (1–5) in most lines of maize and its relatives and is not homologous or related to several transposons that moved into the shrunken gene in lines that were made unstable by BSMV infection. Johns *et al.* (1985) suggested that BSMV infection mobilizes unrelated transposons as a response to environmental stress.

The mobilization of transposons and mutation induction continues in progenies of originally infected plants that themselves do not show infection. These progenies include 'AR' lines. With the use of 'AR' lines, Mottinger isolated seven shrunken mutations from these lines (Mottinger *et al.* 1984*a*) and a number of these behaved as unstable alleles. One of these, *sh-5586* (Tz-86) has been characterized by Dellaporta *et al.* (1984). Like other transposons in maize, the (Tz-86) insert in the *sh* gene has caused a 10 bp duplication as a direct repeat at the termini of the element.

Among these same 'AR' progenies, there appeared unstable *a* alleles. These have been identified as *a-ruq* alleles that are responsive to *Uq* elements (Friedemann & Peterson, 1982; Peterson & Friedemann, 1983; Pereira & Peterson, 1985). What is evident in the examination of Fig. 1 is that these three unstable *a* alleles appeared several generations after the initial infection among plants not showing virus infection. The mobilization of transposons continued for several generations after the initial infection as is evident with these mutants and with the *sh* mutants from the 'AR' lines (Mottinger *et al.* 1984*a*).

Further support for the general mobilization of elements that continues even in the absence of virus-infected plants comes from the analysis of the two parents used in the initial cross by Sprague *et al.* (1963). The original cross (Cross 1) for the alleles in question could more correctly be written as (Cross 4).

$$a^0\ a^0 \times A\ A \text{ (infected plant).} \qquad \text{(Cross 4)}$$

According to the tests with the *Uq* tester (*a-ruq*), the original cross was as is shown in Cross 5.

$$a^0\ a^0\ Uq\ Uq \times A\ A \text{ (no } Uq\text{) infected plant.} \qquad \text{(Cross 5)}$$

Because the *a-ruq* alleles that appeared several generations later must have originated from the *A* alleles of the infected plants (the a^0 allele of the kernels parent is a null, nonresponsive *a* allele), the induction of the *a-ruq* was caused by events initiated in the original infected plant (Cross 1, male parent) but not consummated until several generations later by the mobilization of the *Uq* elements present in the uninfected female parent (Cross 1, female parent).

With this general revelation of parent content with respect to the various alleles in question, the sequence of events in the origin of the unstable alleles may be considered. The general scenario of Receptor–Regulatory element relationship is illustrated by two cases of Transposon Systems interaction (*Ac Ds*, Federoff,

Wessler & Shure, 1983; *En* (Spm), Schwarz-Sommer *et al.* 1984; Pereira *et al.* 1985). This two-system interaction is explained by the insertion of a defective Regulatory element that is homologous to the active Regulatory element in sequence arrangement of its inverted repeat structure. Because of this, it follows that the initial mobilization promoted by the virus infection affected the transposition of the *Uq* elements present in the F1 of the cross (Cross 1) and in subsequent progenies of plants. At some point, a defective *Uq* was inserted into the *A* locus and subsequently behaved as an unstable allele only in the presence of *Uq*. In this way a transposable element system was established of a receptor allele (*a-ruq*) responsive to the Regulatory element, *Uq*. It is curious that of the four *ruq*-receptive alleles, all are receptive types and are not autonomously acting (Friedemann & Peterson, 1982; Oberthur & Peterson, 1984; and Pereira & Peterson, 1985). This is unlike the *En* element that gives rise to autonomously acting elements at loci where it inserts (Peterson, 1961, 1963, 1976, 1978, 1981).

There is at least one other alternative to the origin of the *a-ruq* alleles. Possibly, the active *Uq* mobilized defective *Uq*'s that became inserted as defective receptor elements.

The events initiated by the virus infection continue to induce new events (transposon movement) even in the absence of infected plants. As noted by Rhoades (1943) in describing the effect of the Iojap gene in the origin of cytoplasmic mutants, the transposing events, like a Frankenstein monster, are no longer under the control of its maker.

REFERENCES

BRAKKE, M. K. (1984). The aberrant ratio phenomenon and virus induced mutations in maize. *Annual Review Phytopathology* **22**, 77–94.

BRAKKE, M. K., SAMPSON, R. C. & COMPTON, W. A. (1981). Recessive alleles found at *R*, *C* loci in maize stocks showing aberrant ratio at the *A* locus. *Genetics* **99**, 481–485.

DELLAPORTA, S. L., CHOMET, P. S., MOTTINGER, J. P., WOOD, J. A., YU, S.-M. & HICKS, J. B. (1984). Endogenous transposable elements associated with virus infection in maize. *Cold Spring Harbor Symposium Quantitative Biology* **49**, 321–328.

FEDOROFF, N., WESSLER, S. & SHURE, M. (1983). Isolation of the transposable maize controlling elements *Ac* and *Dc*. *Cell* **35**, 235–242.

FREELING, M. (1984). Plant transposable elements and insertion sequences. *Annual Review of Plant Physiology* **35**, 277–298.

FRIEDEMANN, P. & PETERSON, P. A. (1982). The *Uq* controlling-element system in maize. *Molecular and General Genetics* **187**, 19–29.

JACKSON, A. O., DAWSON, J. R. O., COVEY, S. N., HULL, R., DAVIES, J. W., MACFARLAND, J. E. & GUSTAFSON, G. D. (1983). Sequence relations and coding properties of a subgenomic RNA isolated from barley stripe mosaic virus. *Virology* **127**, 37–44.

JOHNS, M. A., MOTTINGER, J. & FREELING, M. (1985). A low copy number, *copia-like* transposon in the maize genome. *EMBOJ.* **4**, 1093–1102.

MOTTINGER, J. (1982*a*). Mutations of *Adh* induced by barley stripe mosaic virus. *Maize Genetics Cooperation News Letter* **56**, 89–90.

MOTTINGER, J. (1982*b*). More on aberrant ratio. *Maize Genetics Cooperation News Letter* **56**, 84–89.

MOTTINGER, J. P., DELLAPORTA, S. L. & KELLER, P. B. (1984*a*). Stable and unstable mutation in aberrant ratio stocks of maize. *Genetics* **106**, 751–767.

MOTTINGER, J. P., JOHNS, M. A. & FREELING, M. (1984*b*). Mutations of the *Adh1* gene in maize following infection with barley stripe mosaic virus. *Molecular General Genetics* **195**, 367–369.

NELSON, O. E. (1981). A re-examination of the aberrant ratio phenomenon in maize. *Maydica* **26**, 119–131.

OBERTHUR, E. & PETERSON, P. A. (1984). *Uq* controlled mutable allele at the *c* locus. *Maize Genetics Cooperation Newsletter* **58**, 45.

PEREIRA, A. & PETERSON, P. A. (1984). *a-m13* and *a-m16*, additional *Uq*-responsive mutants. *Maize Genetics Cooperation Newsletter* **58**, 6.

PEREIRA, A. & PETERSON, P. A. (1985). Origin and diversity of mutants controlled by the *Uq* transposable element system in maize. *Genetical Research* **46**, 209–226.

PEREIRA, A., SCHWARZ-SOMMER, Zs., GIERL, A., BERTRAM, I., PETERSON, P. A. & SAEDLER, H. (1985). Genetic and molecular analysis of the Enhancer (*En*) transposable element system of *Zea mays*. *European Molecular Biology Organization Journal* **4**, 17–23.

PETERSON, P. A. (1961). Mutable *a1* of the *En* system in maize. *Genetics* **46** (7), 759–771.

PETERSON, P. A. (1963). Influence of mutable genes on induction of instability in maize. *Proceedings of the Iowa Academy of Science* **70**, 129–134.

PETERSON, P. A. (1966). Phase variation of regulatory elements in maize. *Genetics* **54**, 249–266.

PETERSON, P. A. (1976). Basis for the diversity of states of controlling elements in maize. *Molecular and General Genetics* **149**, 5–21.

PETERSON, P. A. (1978). Controlling elements: the induction of mutability at the *A2* and *C* loci in maize. In *Maize Breeding and Genetics* (ed. D. B. Walden), pp. 601–631. New York: John Wiley.

PETERSON, P. A. (1981). Instability among the components of a regulatory element transposon in maize. *Cold Spring Harbor Symposia on Quantitative Biology* **45**, 447–455.

PETERSON, P. A. & FRIEDEMANN, P. D. (1983). The Ubiquitous controlling-element system and its distribution in assorted maize testers. *Maydica* **28**, 213–249.

RHOADES, M. M. (1943). Genic induction of an inherited cytoplasmic difference. *Proceedings of the National Academy of Sciences* **29**, 327–329.

SAMSON, R. G., BRAKKE, M. K. & COMPTON, W. A. (1979). Evidence for gene inactivation in the virus-induced aberrant ratio phenomenon in maize. *Genetics* **92**, 1231–1239.

SCHWARZ-SOMMER, Z., GIERL, A., KLOSGEN, R. B., WIENAND, V., PETERSON, P. A. & SAEDLER, H. (1984). The *Spm* (*En*) transposable element controls the excision of a 2-hb DNA insert at the *wx m-8* allele of *Zea mays*. *European Molecular Biology Organization Journal* **3**, 1021–1028.

SPRAGUE, G. F. & MCKINNEY, H. H. (1966). Aberrant ratio: An anomaly in maize associated with virus infection. *Genetics* **54**, 1287–1296.

SPRAGUE, G. F. & MCKINNEY, H. H. (1971). Further evidence on the genetic behavior of AR in maize. *Genetics* **67**, 533–542.

SPRAGUE, G. F., MCKINNEY, H. H. & GREELEY, L. (1963). Virus as a mutagenic agent in maize. *Science* **14**, 1052–1053.

WIENAND, U., PETERSON, P. & SAEDLER, H. (1983). Hybridization studies of barley stripe mosaic virus cDNA clones to virus induced maize mutants. *Maize Genetics Cooperation News Letter* **57**, 157–158.