Genetics

Genetic Analysis of Barley Stripe Mosaic Virus

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ABSTRACT

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Four barley stripe mosaic virus strains were used in pseudorecombination experiments to elucidate the roles of the individual RNA species in pathogenicity and symptomatology. Experiments with strains CV40 and CV52 indicated that RNA 1 controls pathogenicity to Rodney oats and Modjo-1 barley. Experiments with strains CV35 and CV42 supported this and further indicated that RNA 1 controls pathogenicity to Moreval and Silver King barleys. Pathogenicity to *Chenopodium amaranticolor* was determined by CV35 RNA 1, although isolates possessing CV42 RNA 1 and CV35 RNAs 2 and 3 were also pathogenic to *C. amaranticolor*. Symptom development required a more complex interaction of viral RNAs with the host. Pseudorecombinant isolates most virulent to barley in the CV52 × CV40 experiments were those with CV52 RNA 1 and CV52 RNA 2 or 3. Similarly, pseudorecombinant isolates with CV35 RNA 1 or 2 combined with CV35 RNA 3 induced more severe symptoms in barley than other RNA combinations. Homologous RNAs may be more compatible than heterologous RNAs, significantly affecting symptomatology. Symptoms on oats were more severe when RNA 3 was derived from the more virulent parental strain and milder when RNA 3 was derived from the less virulent parental strain.

Additional key words: Avena sativa, Hordeum vulgare, reassortant.

Barley stripe mosaic virus (BSMV), type member of the hordeiviruses, is a multicomponent virus (8,10) requiring seed transmission for survival (14). Although its economic importance has been reduced considerably during the past 30 years owing to cultural control practices and seed certification programs, it continues to be a major focus of research.

Much of the interest in BSMV stems from the variability in the multicomponent nature of BSMV strains. Although some strains possess as many as four RNA components (MW range ~ $0.99 - 1.43 \times 10^6$), others possess only the largest two or three electrophoretically separable components (9). Recent reports agree that BSMV is essentially tripartite (1,7). RNA 4 of the Argentine Mild and ND161 strains may be readily lost by dilution end point transfer, whereas RNA 3 of ND161, Norwich, and ND18 is stable (12). The Norwich strain requires RNAs 1, 2, and 3 to infect *Chenopodium amaranticolor* Coste & Reyn. (10). Nucleic acid hybridization studies indicate that the "bipartite" type strain actually has three distinct RNAs (7). Finally, in vitro translation products of two, three, four, and intermediate component strains also support a tripartite genome (1).

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Although in vitro translation studies show that RNA 2 contains the coat protein gene (1,3,6), other characters have not yet been linked to specific RNAs. Strains that vary in pathogenicity, symptomatology, and seed transmission have been known for some time (11,14). Genetic analysis of BSMV by constructing pseudorecombinants from these strains would clarify our understanding of the BSMV genome. We describe here pseudorecombinants constructed from four BSMV strains that enabled location of genetic determinants for pathogenicity to oats (*Avena sativa* L.), barley (*Hordeum vulgare* L., three cultivars), and *C. amaranticolor*. The roles of the various RNA species in symptom expression were also evaluated.

MATERIALS AND METHODS

Virus sources, maintenance, and purification. All virus isolates were obtained from cereal virus (CV) cultures stored in liquid nitrogen at Fargo, ND. CV35 (ND131) was designated by McKinney and Greeley (11) as the fleck blotch strain and was originally derived from a Canadian culture. CV40 (ND161) and CV42 (ND159), the eyespot oat and California moderate oat strains, respectively, were both derived from California oat strains. CV52 (ND18) was originally isolated from the type strain (CV18) by R. G. Timian. All contained three electrophoretically separable RNA components (Fig. 1). Pathogenicity differences among these isolates are summarized in Table 1. Cultivars used to distinguish these differences were Silver King (CI 905), Modjo-1 (CI 14048), and Moreval (CI 5724) barleys and Rodney (CI 6661) oats.

All isolates were propagated on Black Hulless barley (CI 666) in the greenhouse at 27 C with supplemental lighting for 16 hr a day. Seven days after inoculation, plants were harvested and virus was purified essentially as described by Lane (10).

Extraction and purification of RNA. RNA was isolated from purified viruses as previously described (4). After ethanol precipitation, RNA was suspended in an appropriate volume of sterile PEN buffer (0.01 M sodium phosphate, 0.001 M EDTA, 0.001 M NaN_3, pH 7.0). Individual RNAs were separated by electrophoresis on either 2.4% polyacrylamide gels or 2.5% polyacrylamide/0.5% agarose gels. RNA species were separated and recovered as previously described (4).

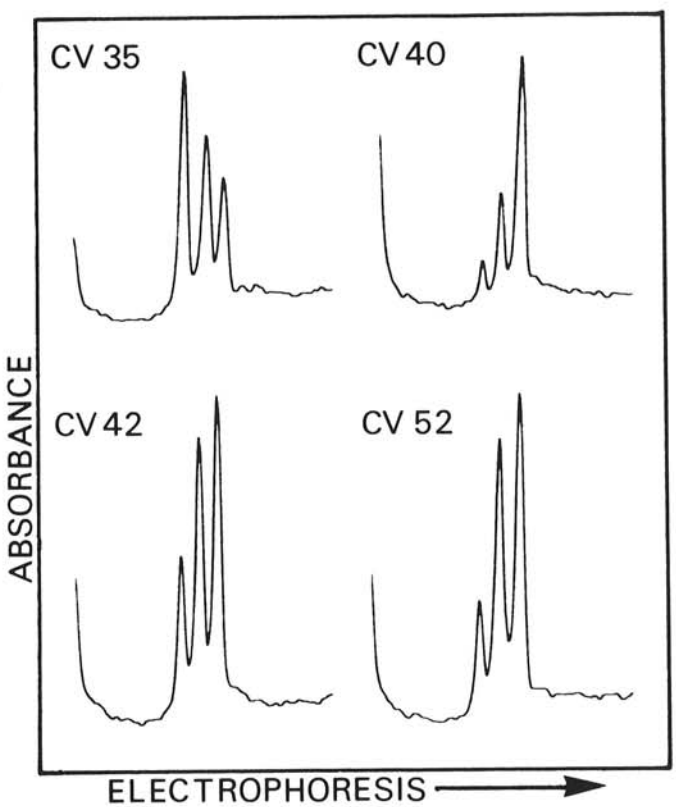


Fig. 1. RNA profiles of barley stripe mosaic virus strains CV35, CV40, CV42, and CV52 after electrophoresis on 2.8% polyacrylamide/0.5% agarose gels. Purified virus particles were disrupted and RNA modified with formaldehyde as described by Lane (10) immediately before loading the gel.

Construction of pseudorecombinants. Two sets of pseudorecombinants were constructed, CV42 × CV35 and CV40 × CV52. In each case, all possible combinations of RNAs (equal amounts of each) were spray-inoculated to Black Hulless barley (Table 2). RNA concentration was 3–5 μg/ml with 300 μg/ml fractionated bentonite (5) added. Ten infected plants representing each combination were individually triturated and again inoculated near the dilution end point to Black Hulless barley. A single infected plant then served as the source of an individual isolate (10 from each combination). Individual isolates or groups of isolates were identified according to their expected genotype. The actual genotypes of the pseudorecombinants were not absolutely proved.

Isolates were maintained in Black Hulless barley before inoculation of differential hosts. Differential hosts lacking obvious symptoms were indexed by latex agglutination tests.

RESULTS

Pathogenicity analysis of pseudorecombinants. In the first experiment, genetics of pathogenicity to Modjo-1 barley and Rodney oats were examined (Table 3). Whereas Black Hulless

TABLE 1. Variation in pathogenicity among barley stripe mosaic virus (BSMV) strains

BSMV strain	Barley cultivar				Oat cultivar	
	Black Hulless	Silver King	Modjo-1	Moreval	Rodney	*Chenopodium amaranticolor*
CV40	+[a]	−	−	−	+	+
CV42	+	−	−	−	+	−
CV52	+	+	+	+	−	+
CV35	+	+	+	+	−	+

[a] + = Infection, − = no infection.

TABLE 2. Combinations of RNA species used to construct pseudorecombinants from barley stripe mosaic virus strains[a]

CV40 × CV52	CV42 × CV35
055	433
050	434
005	443
500	344
505	343
550	334
000	333
555	444

[a] Numbers indicate origins of RNAs 1, 2, and 3, respectively; 0, 5, 4, and 3 indicate RNA species was obtained from CV40, CV52, CV42, and CV35, respectively (e.g., 055 = CV40 RNA 1 + CV52 RNA 2 + CV52 RNA 3).

TABLE 3. Differential host reactions to pseudorecombinants constructed by exchanging RNAs 1, 2, and 3 between barley stripe mosaic virus strains CV40 and CV52

Host	RNA combination[a]							
	055	050	005	500	505	550	000	555
Black Hulless barley	9/9[b]	10/10	10/10	9/9	10/10	10/10	2/2	2/2
Modjo-1 barley	3/9	0/10	0/10	9/9	10/10	10/10	0/2	2/2
Rodney oats	9/9	10/10	10/10	0/9	2/10	0/10	2/2	0/2

[a] Numbers indicate origins of RNAs 1, 2, and 3, respectively; 0 and 5 indicate RNA species was obtained from CV40 and CV52, respectively (e.g., 055 = CV40 RNA 1 + CV52 RNA 2 + CV52 RNA 3).

[b] Number of isolates pathogenic/number of isolates tested.

barley was susceptible to all isolates, Modjo-1 was susceptible only to isolates with CV52 RNA 1 and Rodney oats was susceptible only to isolates with CV40 RNA 1.

To confirm and expand these results, pseudorecombinants were constructed from strains CV35 and CV42 (Table 4). Again, all isolates were pathogenic to Black Hulless barley. Only isolates with CV42 RNA 1 were pathogenic to Rodney oats, whereas those with CV35 RNA 1 were pathogenic to Modjo-1, Moreval, and Silver King barleys.

A few discrepancies are evident in Tables 3 and 4. Cross-contamination of the original RNA preparations is the simplest explanation. For example, one of the 433 isolates (CV42 RNA 1 + CV35 RNA 2 + CV35 RNA 3) did not infect oats but did infect Modjo-1, Moreval, and Silver King barleys (Table 4). This isolate's genotype was probably 333 rather than the expected 433. In other instances, isolates infected all differential hosts, probably because of mixed infections. Passage of these isolates through either oats or barley eliminated infectivity in the other host, indicating that these isolates were indeed mixtures.

All isolates with CV35 RNA 1 could infect *C. amaranticolor*. Surprisingly, isolates of genotype 433 were also pathogenic to *C. amaranticolor*. Their inability to infect Modjo-1, Moreval, and Silver King barleys shows they were not contaminated with CV35 RNA 1. Additional evidence supports this conclusion. One 433 isolate was successfully transferred from *C. amaranticolor* directly to oats. This isolate must have possessed CV42 RNA 1, since oat infection requires this RNA. When inoculated to *C. amaranticolor* from infected oats, these isolates still induced local lesions. Passage through oats should have removed contaminating CV35 RNA 1.

Some infectivity was also apparent with the 433 combination when RNAs were inoculated directly to *C. amaranticolor* (Table 5). Local lesion number increased (compared with that of other heterologous RNA combinations) when CV35 RNAs 1 and 3 were combined with CV42 RNA 2 and inoculated directly to *C. amaranticolor* (Table 5).

Symptomatology analysis of CV52× CV40 pseudorecombinants. Most of the isolates induced stripe mosaic-type symptoms in Black Hulless barley (Table 6). Early symptoms (10–12 days post inoculation) were expressed as a green stripe beginning at the leaf base and extending into the more chlorotic middle and distal leaf sections (Fig. 2). All isolates induced this type of symptom except those of genotype 500, which induced mostly very mild chlorosis and mosaic symptoms. By 21 days, symptoms had shifted toward essentially similar stripe mosaics on plants infected with 050, 005, 055, and 505 isolates (Fig. 3). Symptoms induced by 505 isolates, however, were most prominent and most closely resembled those induced by 555 (CV52) isolates. The stripe mosaics induced by 050, 005, and 055 isolates were more prominent than those induced by 000 (CV40) isolates and less prominent than those induced by 505 and 555 isolates. The 550 isolates caused some stripe mosaic, but green streaking with more severe chlorosis persisted as the most prominent symptom. These plants were also more severely stunted than others. Symptoms were somewhat difficult to differentiate because parental strains induced similar symptoms.

Symptoms induced on Rodney oats by 050, 005, 055, and 000 isolates were more easily distinguishable. Isolates with genotype 050 and 000 induced essentially the same symptoms. At 9–10 days post inoculation, plants infected with isolates of these two genotypes showed mild mosaic and chlorosis (Fig. 4). Mild spindle streak or eyespot symptoms appeared after about 21 days. The 005 and 055 isolates induced much stronger early mosaic and necrosis (especially with 055 isolates), followed by severe spindle streak/eyespot symptoms (Figs. 5 and 6). Isolates with the 055 genotype induced slightly more severe symptoms than those with the 005 genotype.

Symptomatology analysis of CV35× CV42 pseudorecombinants. Symptoms induced by the various CV35 × CV42 isolates were more easily distinguishable than those induced by the CV52 × CV40 isolates (Table 7). The CV35 and CV42 parental isolates induced more distinctive symptoms.

In Black Hulless barley, the 343, 334, 344, and 333 (CV35) isolates caused severe chlorosis and collapse of the systemically infected leaf 6–7 days post inoculation. Isolates of genotype 433 induced some collapse of the tip of the systemically infected leaf at this time also. Other isolates were not as virulent.

By 14 days post inoculation, plants infected with isolates from the 343, 433, and 333 combinations showed severe fleck mosaic and stunting (Fig. 7). Those infected with 443 isolates induced more of a stripe mosaic with some flecking, although the flecking was not as distinct as that induced by the 343 and 433 isolates (Fig. 7). Isolates of genotypes 434 and 444 caused stripe mosaics, whereas plants infected with the 334 and 344 isolates showed green streaks

TABLE 4. Differential host reactions to pseudorecombinants constructed by exchanging RNAs 1, 2, and 3 between barley stripe mosaic virus strains CV35 and CV42

	RNA combination[a]							
Host	433	434	443	344	343	334	333	444
Black Hulless barley	10/10[b]	10/10	10/10	10/10	9/9	10/10	2/2	2/2
Modjo-1 barley	1/10	0/10	2/10	10/10	9/9	9/10	2/2	0/2
Moreval barley	1/10	0/10	2/10	10/10	9/9	10/10	2/2	0/2
Silver King barley	1/10	0/10	2/10	10/10	9/9	10/10	2/2	0/2
Rodney oats	9/10	10/10	8/10	0/10	0/9	0/10	0/2	2/2
Chenopodium amaranticolor	10/10	0/10	4/10	10/10	9/9	10/10	2/2	0/2

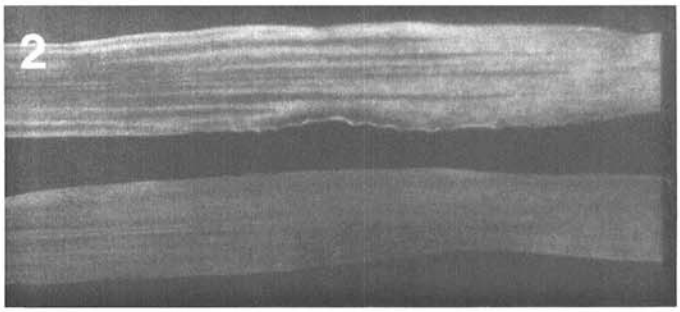
[a] Numbers indicate origins of RNAs 1, 2, and 3, respectively; 4 and 3 indicate RNA species was obtained from CV42 and CV35, respectively (e.g., 433 = CV42 RNA 1 + CV35 RNA 2 + CV35 RNA 3).
[b] Number of isolates pathogenic/number of isolates tested.

TABLE 5. Infectivity to *Chenopodium amaranticolor* of RNA mixtures derived from barley stripe mosaic virus strains CV35 and CV42

RNA combination[w]	Number of lesions[x]
334[y]	6 cd[z]
344	9 c
343	20 b
443	0 d
433	5 cd
434	0 d
333	45 a
444	0 d

[w] Each inoculated at a concentration of 1 μg/ml.
[x] Average per half-leaf (four half-leaves per repetition, two repetitions).
[y] Numbers indicate origins of RNAs 1, 2, and 3, respectively; 4 and 3 indicate RNA species was obtained from CV42 and CV35, respectively (e.g., 334 = CV35 RNA 1 + CV35 RNA 2 + CV42 RNA 3).
[z] Numbers followed by the same letter are not significantly different, $P \leq 0.1$ (least significant difference test).

Figs. 2–8. Examples of symptoms produced on plants infected with pseudorecombinants constructed from barley stripe mosaic virus strains CV52 and CV40 (Figs. 2–6) or CV35 and CV42 (Figs. 7 and 8). **2,** Typical green streak produced on Black Hulless barley 10–12 days after inoculation. Bottom leaf is healthy. **3,** Typical stripe mosaic symptom on Black Hulless barley about 21 days after inoculation. **4,** Mild chlorosis and mosaic produced on the second leaf and start of the spindle streak/eyespot symptom produced on the third leaf of Rodney oats 10–12 days after inoculation with the 050 and 000 isolates. Bottom leaf is healthy. **5,** As Figure 4, except more severe symptoms produced after inoculation with 005 isolates. **6,** As Figure 4, except more severe symptoms produced after inoculation with 055 isolates. **7,** Range of symptoms produced on Black Hulless barley 14–15 days after inoculation with pseudorecombinants of CV35 and CV42. Top leaf is healthy, followed in descending order by leaves from plants infected with isolates of 343, 334, 344, 434, 443, 433, 444, and 333. **8,** Range of symptoms produced on Rodney oats about 14 days after inoculation with pseudorecombinants of CV35 and CV42. Top leaf is from a plant infected with an isolate of 434, followed in descending order by leaves from plants infected with isolates of 443 and 433; bottom leaf is healthy. Plants infected with 444 appear similar to those infected with 434.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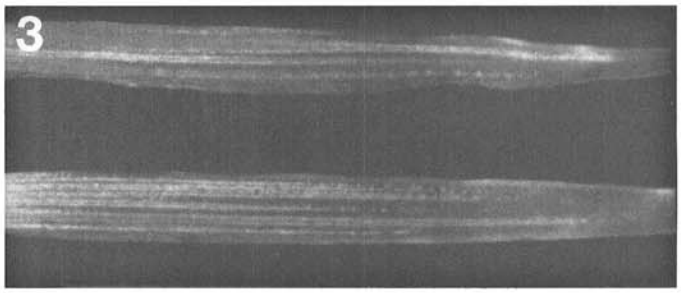
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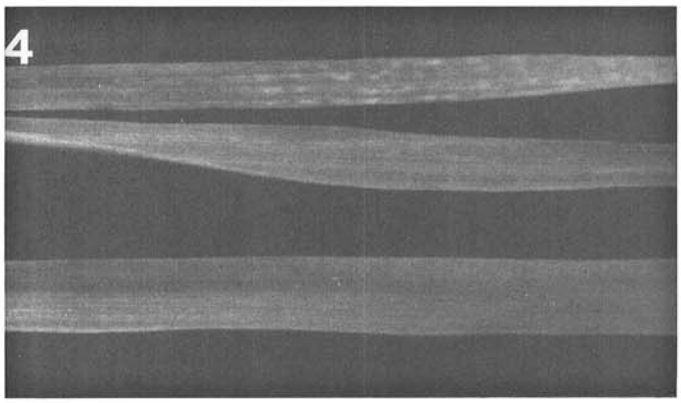
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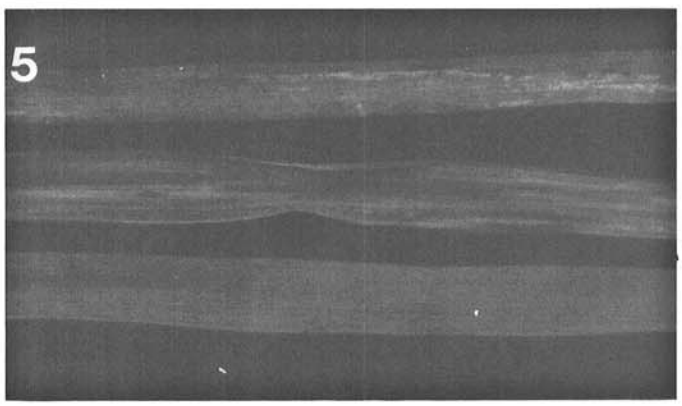
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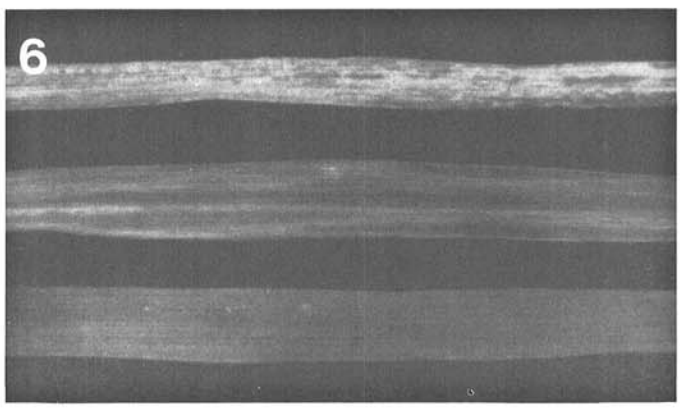
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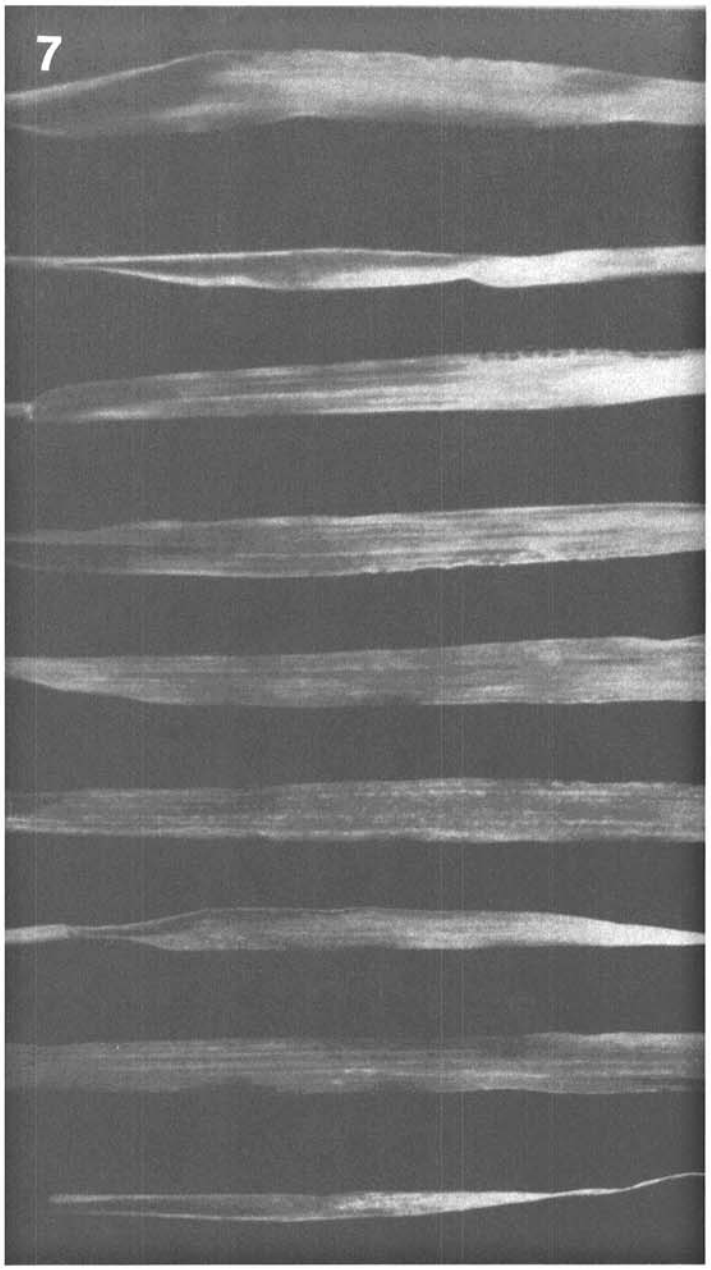
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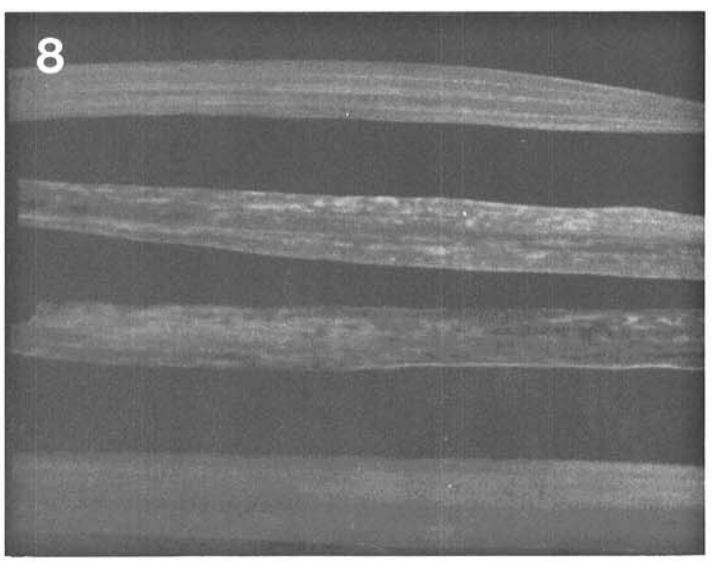
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extending from the leaf base upward into the chlorotic tips (Fig. 7).

Occasionally, other isolates, including those with 333 and 444 genotypes, also showed some degree of striping. Green streaks extended from the leaf base into the more chlorotic leaf tips in plants infected with 333 isolates. If plants infected with 444 isolates showed striping, on the other hand, the basal end of the leaf was more chlorotic than the distal end.

The 434 and 444 isolates induced mild stripe mosaic symptoms in oats 2–3 wk after inoculation (Fig. 8). Those with genotype 443 induced mostly a fleck mosaic (Fig. 8, second leaf), and those with genotype 433 induced the most severe symptoms, also of the fleck-mosaic type (Fig. 8, third leaf).

DISCUSSION

Pseudorecombination studies such as these have an inherent risk of RNA contamination as a source of error. Although this risk could not be completely eliminated, the overall evidence clearly demonstrated the importance of RNA 1 for pathogenicity to certain hosts. CV35 or CV52 RNA 1 was essential for infection of barley cultivars Modjo-1, Moreval, and Silver King, whereas CV40 or CV42 RNA 1 was essential for infection of Rodney oats. Pathogenicity to *C. amaranticolor* was also determined by RNA 1, with the exception of the combination CV42 RNA 1 + CV35 RNAs 2 + 3. No clear explanation for this exists. Considering the complexity of the interaction and the involvement of multiple RNAs in symptom development, this may not be so unusual.

All RNAs had some degree of influence on symptom development. Isolates with CV52 RNA 1 combined with either CV52 RNA 2 or 3 induced more severe symptoms on Black Hulless barley than did other heterologous isolates in the CV52 × CV40 experiments. With pseudorecombinants of CV35 × CV42, symptoms were more severe when induced by isolates with CV35 RNA 1 or 2 and CV35 RNA 3. Apparently, no single RNA dominated symptom expression in Black Hulless barley. In infected oats, isolates with RNAs 2 and 3 from the more virulent of the parental strains (CV35 or CV52) induced the most severe symptoms.

The fact that certain combinations of homologous RNAs significantly affected symptom expression could indicate that compatibility is involved. While all RNA combinations were sufficiently compatible for infection of a susceptible host (e.g., Black Hulless barley), perhaps two RNAs donated from the same parent are more compatible with each other, significantly affecting symptom intensity, if not symptom type.

A role for RNA 3 in moderating the number of local lesions produced on *C. amaranticolor* could be implied by the fact that local lesion number on *C. amaranticolor* was enhanced when RNAs 1 + 3 of a strain (CV35) that infected *C. amaranticolor* were combined as inoculum with RNA 2 from a strain (CV42) that did not infect *C. amaranticolor*. Further investigation is required, however. Preliminary evidence from our laboratory indicates that such isolates (e.g., 343) may be present in higher titer than others (e.g., 344 and 334) within infected barley. The increase in the number of local lesions produced could have arisen from increased aggressiveness of the isolate rather than a specific function of RNA 3 per se.

Symptomatology data also support the hypothesis that RNA 3 influences aggressiveness. RNA 3 alone was sufficient to change the symptom or symptom severity on oats. In all cases where RNA 3 was donated from the more virulent parental strain (CV35 or CV52), symptoms on oats were more severe than when RNA 3 was donated from the less virulent parental strain (CV40 or CV42). Observations of Black Hulless barley and oats in the CV35 × CV42 experiments indicated that CV35 RNA 3 was associated with the appearance of fleck-type mosaic symptoms. The influence of CV52 RNA 3 on symptoms was more obvious in oats than in Black Hulless barley.

The importance of RNA 3 in symptom expression as demonstrated here also reaffirms the tripartite nature of the BSMV genome. Although specific functions were attributable to RNAs 1 and 3, no function was found that could be attributed solely to RNA 2, except for its previously reported role in coat protein production (1,3,6). Much of the information on RNAs 1, 2, and 3 has been shown to be unique (2,7,12,13). Our data, along with Lane's (10), add biological evidence of specific and separate functions for each RNA species.

Combining this type of genetic data with nucleotide sequencing data has great potential for providing insight into the mechanisms of pathogenicity and symptom development. Further, the future

TABLE 6. Symptoms[a] produced on Black Hulless barley and Rodney oats approximately 3 wk after inoculation with pseudorecombinants constructed from CV52 and CV40

	RNA combination[b]							
Host	055	050	005	500	505	550	000	555
Black Hulless barley	Stripe mosaic	Stripe mosaic	Stripe mosaic	Mosaic	Stripe mosaic	Stripe or streak	Stripe mosaic	Stripe mosaic
Rodney oats	Mosaic; necrosis; spindle streak and eyespot	Mosaic; spindle streak and eyespot	Mosaic; necrosis; spindle streak and eyespot	...[c]	...	...	Mosaic; spindle streak and eyespot	...

[a] Stripe mosaic = mosaic pattern extending parallel to veins to form broken stripe; stripe or streak = solid or unbroken band of chlorotic or green area extending up leaf; mosaic = general mosaic not limited to stripe pattern.
[b] Numbers indicate origins of RNAs 1, 2, and 3, respectively; 0 and 5 indicate RNA species was obtained from CV40 and CV52, respectively (e.g., 055 = CV40 RNA 1 + CV52 RNA 2 + CV52 RNA 3).
[c] No infection.

TABLE 7. Symptoms[a] produced on Black Hulless barley and Rodney oats 2–3 wk after inoculation with pseudorecombinants constructed from CV35 and CV42

	RNA combination[b]							
Host	433	434	443	344	343	334	444	333
Black Hulless barley	Fleck mosaic; severe stunting	Stripe mosaic	Stripe mosaic; fleck mosaic	Green streak	Fleck mosaic; severe stunting	Green streak	Stripe mosaic	Fleck mosaic; severe stunting
Rodney oats	Fleck mosaic	Stripe mosaic	Fleck mosaic	...[c]	...	...	Stripe mosaic	...

[a] Fleck mosaic = sharply defined mosaic pattern distributed randomly over leaf surface and not in a stripe pattern; green streak = solid green band extending up leaf into lighter green area; stripe mosaic = mosaic pattern extending parallel to veins to form broken stripe.
[b] Numbers indicate origins of RNA 1, 2, and 3, respectively; 4 and 3 indicate RNA species was obtained from CV42 and CV35, respectively (e.g., 433 = CV42 RNA 1 + CV35 RNA 2 + CV35 RNA 3).
[c] No infection.

use of RNAs derived from DNA clones rather than of electrophoretically separated RNAs can eliminate the inherent risk of RNA contamination as a source of error.

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