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Furovirus Diseases of Sugar Beets in the United States

Sugar beets (*Beta vulgaris* L.) comprise a major segment of the sweetener industry in the United States, outpacing sugarcane in total value by almost 50%. In 1993, 24 million metric tons (26.4 million tons) of sugar beets were harvested from 566,802 ha (1.4 million acres), at an average yield of 42 t/ha (18.7 tons per acre). Approximately 65,000 individuals were employed in the production and processing of the 1993 beet crop, which was worth over two billion dollars (35).

In the United States, sugar beets are produced in five distinct geographic regions (35). The Great Lakes region, which includes Michigan and Ohio, produces approximately 13% of the crop annually. The Red River Valley region of North Dakota and Minnesota constitutes the most intensive sugar beet production area, producing 32% of the crop on approximately 230,769 ha (570,000 acres). The Great Plains is the largest geographical region and includes Montana, Wyoming, Nebraska, Colorado, New Mexico, and Texas. This region produces approximately 22% of the sugar beet crop on 114,170 ha (282,000 acres). The Northwest region, encompassing Idaho and Oregon, produces approximately 19% of the annual sugar beet crop. This region is especially important to the U.S. sugar beet industry because all U.S. sugar beet seed is produced in Oregon. The Southwest region includes only California since Arizona ceased sugar beet production in the early 1980s. This region produced approximately 14% of the 1993 sugar beet crop and is unquestionably the most agronomically and climatically diverse, with year-round sugar beet production. Of all the major sugar beet production regions, the Southwest is the only one that has experienced a steady decline in production over the last 10 years. Although numerous factors have contributed to this decline, in a large part it has been due to rhizomania, a devastating disease of sugar beets caused by beet necrotic yellow vein virus (BNYVV), a member of the furovirus group.

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Furoviruses

Furoviruses are a defined taxonomic group of *fu*ngal-transmitted, *ro*d-shaped, single-stranded RNA viruses with divided, typically bipartite genomes (11,12,15). The name was first proposed by Shirako and Brakke (45) in 1984 and was accepted by the International Committee on the Taxonomy of Viruses in 1987 (15). Furoviruses occur in temperate regions of five continents, and several of the most important members of the group are cosmopolitan in distribution (41). The natural fungal vectors of these viruses belong to the genera *Polymyxa* and *Spongospora* (5). Soilborne wheat mosaic virus (SBWMV) is the type member of the furovirus group, which also includes beet necrotic yellow vein virus (BNYVV), beet soilborne virus (BSBV), broad bean necrosis virus (BBNV), fern mottle virus, Hypochoeris mosaic virus, peanut clump virus (PCV), Indian peanut clump virus (IPCV), Nicotiana velutina mosaic virus (NVMV), oat golden stripe virus (OGSV), potato mop top virus (PMTV), and rice stripe necrosis virus (RSNV) (15,41). Recently, sorghum chlorotic spot virus (SCSV) (32) and beet soilborne mosaic virus (BSBMV) were proposed to be furoviruses (43,50).

Furoviruses infecting sugar beet are vectored by the soilborne fungus *Polymyxa betae,* a member of the *Plasmodiophoromycetes* (1,19,20). *P. betae* is an obligate parasite and has a limited host range, primarily within the *Chenopodiaceae, Amaranthaceae,* and *Portulacaceae* (2,5,6,17). The fungus only infects primary root tissue of young roots, and the optimum temperature for infection is around 25°C (17).

The relationship between *P. betae* and BNYVV is representative of the disease cycle of most furoviruses (5). *P. betae* survives in field soil as cystosori. In the presence of a host and proper environmental conditions, cysts give rise to zoospores, which swim through free soil water until they contact a host root and encyst. Encysted zoospores produce a structure called a stachel, through which zoosporic cytoplasm enters the host cell and becomes a plasmodium. The host cell becomes infected with BNYVV if *P. betae* is viruliferous. If a nonviruliferous zoospore infects a root cell containing BNYVV, the plasmodium can incorporate the virus. BNYVV is not believed to replicate in *P. betae,* but the precise mechanism by which *P. betae* actually transmits or takes up virus particles is unknown. After a period, the plasmodium develops into a zoosporangium, which releases additional zoospores that repeat the infection cycle (5). However, some plasmodia develop into cysts, and often nearly every cell in the small feeder roots will contain a cyst. As root cells senesce, cysts (which contain BNYVV) are eventually released into the soil (Fig. 1), where they can remain viable for years without loss of virulence.

Beet Necrotic Yellow Vein Virus

Unlike most furoviruses, which possess bipartite, single-stranded RNA genomes, wild type isolates of BNYVV typically contain four single-stranded RNA species, although a fifth has been observed in some Japanese isolates (41). Particles of BNYVV measure 85, 100, 265, and 390 nm long and 20 nm wide. Corresponding RNA species are 1.5, 1.8, 4.7, and 6.8 kb and are 3′ polyadenylated. The coat protein is 22 kDa (11,41).

The complete BNYVV genome is 14,599 nucleotides, excluding poly-A tails (7). RNA 1 contains a single open reading frame (ORF) encoding a protein hypothesized to be involved with RNA replication (9). Six ORFs are located on RNA 2, including the 22-kDa coat protein gene

Fig. 1. Cystosorus of *Polymyxa betae* in a cell sloughing off the main root.

(11,41) and a triple gene block associated with cell-to-cell movement (10,41). RNA 1 and 2 are both required for virus infection and are always present in infected tissue (33,34). RNA 3 and RNA 4 both contain a single ORF. Genetic studies indicate that RNA 3 is involved with symptom expression on leaves of mechanically infected plants (8,28,41) and with root symptoms in natural infections (46). RNA 4 is believed to be associated with vector transmission (41). Although information concerning RNA 5 is scarce, it appears to be involved in facilitating systemic spread within the root system. RNAs 3 and 4 are always present in natural root infections but are often partially deleted or completely absent after repeated mechanical transmission (34).

Numerous studies (28,29,33,34,46,47) have shown that deletion or loss of RNA 3 and 4 can greatly affect fungal transmission and symptom expression of BNYVV. RNA 3 deletions can result in significant modifications of symptoms on local lesion hosts such as *Chenopodium quinoa*. Instead of the bright yellow lesions typical of wild type BNYVV isolates (Fig. 2), RNA 3 deletion mutants cause diffuse, pale yellow lesions or necrotic spots, depending on the nature of the deletion. When sugar beets are naturally infected by *P. betae* with BNYVV RNA 3 deletion mutants, the typical hairy root symptom associated with rhizomania does not occur even though the virus is present and replicating in the host cells (46,47). Furthermore, when wild type BNYVV is coinoculated with an RNA 3 deletion mutant onto a local lesion host, the symptom phenotype of the mutant predominates (29). These deletion mutations are the focus of extreme interest among researchers and have provided a much better understanding of the molecular aspects of disease development and vector transmission.

Fig. 2. Symptoms of beet necrotic yellow vein virus (BNYVV) on *Chenopodium quinoa*. Wild type isolates of BNYVV typically cause bright yellow local lesions that spread along leaf veins.

Rhizomania

Rhizomania, caused by BNYVV, is one of the more devastating of all sugar beet diseases. It was first identified in Italy and has since been found in the United States, Great Britain, Japan, and most of the sugar beet–growing countries of Europe (10,30). Rhizomania was first reported in the United States in California in 1984 and in Texas in 1987 (13,14). The disease was believed to be limited to these two states, but in 1992 to 1994 rhizomania was found in Colorado, Idaho, Nebraska, and Wyoming (James Gerik, *personal communication*).

Disease symptoms. Foliar symptoms of rhizomania on sugar beets in the field are obscure and easily confused with nitrogen deficiency. Irregular or circular groups of infected plants may be observed when the pathogen is initially introduced into a field, but it is common to find fields in which a large percentage of plants is infected. BNYVV-infected plants may be slightly stunted, with mildly chlorotic leaves. Foliage on severely infected plants readily wilts during the day even when soil moisture is adequate, but it regains turgor overnight. Plants infected by BNYVV often exhibit excessive crown growth, and leaves may grow in a more upright position than normal. The necrotic yellow vein symptom (Fig. 3), after which the virus was named, is extremely rare and seldom seen under natural field conditions.

Fig. 3. Systemic foliar symptoms of rhizomania (A) on a field-grown sugar beet leaf characterized by necrotic yellow veins and (B) on a new leaf from a greenhouse-grown sugar beet. Veins eventually turned necrotic and the leaf died.

Root symptoms associated with rhizomania are variable and depend on when plants become infected (16). The most characteristic and diagnostic symptoms are observed when plants are infected early in the growing season. The tip of the taproot is killed, resulting in excessive lateral root proliferation. Subsequently, these new roots also become infected and eventually die. Lateral roots continue to develop, giving the tap root a "bearded" appearance, from which the name rhizomania (root madness) was derived (Fig. 4). Microscopic examination of these small lateral roots often reveals the presence of cystosori of *P. betae*. However, cysts of *P. betae* can be difficult to find in field-grown beets when roots are necrotic and decayed.

When soil is too cool or dry for early infection by *P. betae*, plants may become infected with BNYVV later in the growing season. Such infections are usually much less damaging. Roots are typically constricted at the point of infection, giving the root a "wine glass" appearance (Fig. 5). Root bearding (rhizomania) may be completely absent or occur only on the lower

Fig. 4. Root bearding caused by beet necrotic yellow vein virus (BNYVV).

part of the constricted portion of the root. Although sugar content is reduced in these beets, total root yield is often near normal. When roots infected by BNYVV are cut longitudinally, the central stele is frequently discolored orange or reddish brown. This discoloration of the central stele can be confused with symptoms of Fusarium root rot, caused by *Fusarium oxysporum* f. sp. *betae*. However, with *Fusarium* numerous vessels will be discolored and necrotic, while with rhizomania only the central stele is affected.

Detection and diagnosis. Several soilborne fungal pathogens and adverse soil conditions, such as hardpans, can cause symptoms that may be confused with rhizomania. Therefore, excessive lateral root proliferation is not diagnostic for rhizomania and is insufficient for predicting the presence of BNYVV. Double antibody sandwich enzyme-linked immunosorbent assay (DAS-ELISA), dot blot, and reverse transcriptase-polymerase chain reaction (RT-PCR) techniques have been developed for detection of BNYVV and other furoviruses of sugar beet (43,50). DAS-ELISA is best for rapid diagnosis of large numbers of samples. Dot blots and RT-PCR are both more specific and sensitive than ELISA, but these techniques are not yet suitable for routine processing of large numbers of samples.

Although time-consuming, one of the more accurate methods for detecting BNYVV in a particular field is to bait viruliferous *P. betae* from the soil. Sugar beet seed can be directly planted, or if fungal pathogens are present, 2- to 3-week-old seedlings can be transplanted into the test soil. Plants should be well watered, maintained at a temperature between 20 and 25°C for 6 to 10 weeks, and then harvested and tested by DAS-ELISA. If plants are not harvested within 12 to 14 weeks, excessive root necrosis can result in erratic or erroneous test results. Only small rootlets should be chosen for testing because BNYVV is unevenly distributed in the root and is seldom found in the fleshy portion of the taproot (31).

Disease loss. In California, rhizomania has been one of the primary factors responsible for a 33% reduction in acreage planted to sugar beets since 1983 (California Beet Growers Association, Stockton, CA.). The Paso Robles area of the Salinas Valley, where BNYVV was first identified in the United States, has essentially quit sugar beet production (3). Numerous other areas of central and northern California have also experienced devastating losses. These losses occurred primarily in the fall-harvested beet crop, which is planted in the spring. In 1993, winter rains greatly delayed planting in the Hamilton City area of Northern California. By the end of February, only 12% of the crop had been planted instead of the usual 80 to 90%. This meant that beets were emerging much later than normal in warm wet soils, ideal conditions for disease development. The resulting crop was a disaster, and the dramatic yield reduction was blamed directly on rhizomania (16).

When beets in California are planted in the fall, grown over winter, and harvested in the summer, disease losses from rhizomania have been much less severe. For instance, in the Imperial Valley, Holly Sugar contracts over 25,000 acres of fall-planted beets annually at the Brawley factory. In 1994, even though approximately one-third of the fields were infested with BNYVV, the factory set yield and production records. Production of a record crop in the presence of BNYVV was attributed to cool soil temperatures and limited irrigation during the early growth stages of the crop (16).

In the other states where rhizomania has been found, losses such as those experienced by growers in Northern California would devastate the sugar industry. Fortunately, this has not occurred. Although individual fields have suffered significant yield loss, rhizomania has not caused widespread damage, even when BNYVV was known to be present in the field. In 1993, 13 sugar beet fields in eastern Colorado were identified as positive for BNYVV. At harvest, the average yield for those fields was 51.1 t/ha (22.76 tons/acre) with a 17.08% sugar content, while the overall factory average was 44.9 t/ha (20.01 tons/acre) with a 17.08% sugar content. Likewise in 1994, the average yield from nine fields testing positive for BNYVV was 45.9 t/ha (20.46 tons/acre) with a sugar content of 15.15%. The factory average was 49.1 t/ha (21.87 tons/acre) with a sugar content of 15.64% (Western Sugar Company, Greeley, CO, *unpublished*). Results from Texas and Wyoming have been similar. In 1994, of 176 fields from Texas that were tested for the presence of BNYVV, 52 tested positive and 124 tested negative. The average yield from the positive fields was 42.9 t/ha (19.1 tons/acre) with a sugar content of 14.9%,

Fig. 5. Root constriction caused by beet necrotic yellow vein virus (BNYVV).

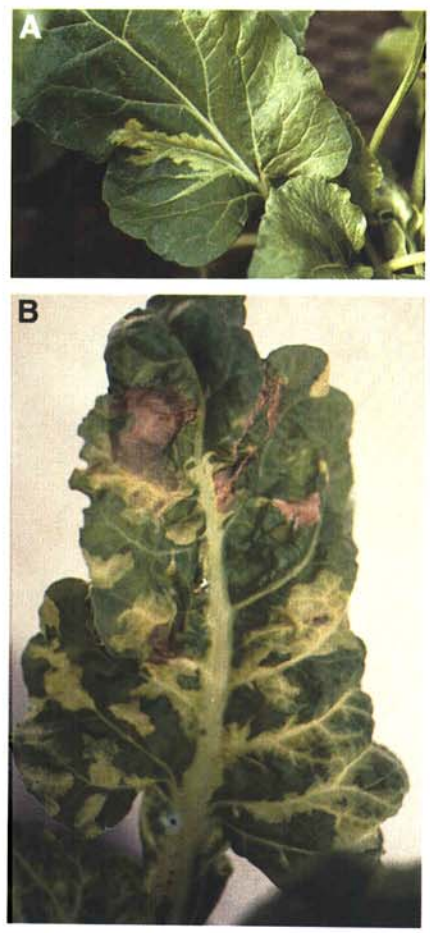


Fig. 6. Systemic symptoms on sugar beet leaves caused by beet soilborne mosaic virus (BSBMV). (A) Initially, systemic infections appear as light green bands along leaf vein. (B) On older leaves, bands and blotches may become bright yellow and then necrotic.

while the yield from the fields that tested negative was 42.4 t/ha (18.9 tons/acre) with a sugar content of 15.09%. In Wyoming, 300 samples were tested during the 1994 harvest. One hundred fifty-eight tested negative and 112 tested positive for BNYVV, but again, there was no significant difference in yields (J. S. Gerik, Holly Sugar Corporation, Tracy, CA, *unpublished*). Obviously, the presence of BNYVV in these fields had minimal impact on final yields.

The reasons sugar beet yields are sometimes unaffected in fields testing positive for BNYVV are unknown. However, as suggested for production in the Imperial Valley of California, it is likely that soil temperatures in Colorado, Texas, and Wyoming at planting time and early in the growing season are "below the threshold required for infection" (16). The soil temperature at planting time in these two states is typically between 5 and 15°C, which is well below the optimum for *P. betae* (6,21). Therefore, producers in these states will normally be able to avoid seedling infection unless they are forced to plant or replant when soil temperatures are higher.

A second reason that yields are sometimes not reduced in fields known to be infested with BNYVV could relate to pathogen distribution. *P. betae* is usually widespread in fields previously planted to sugar beets. However, within a given field the distribution of viruliferous *P. betae* can be much different than that of nonviruliferous *P. betae*. Gerik and Duffus (18) found in California that viruliferous *P. betae* was sometimes limited to small spots within a field and concluded that in such fields the viruliferous population had only recently been introduced. They also suggested that viruliferous populations of *P. betae* could replace nonviruliferous populations. If BNYVV has only recently been introduced into Colorado, Texas, and Wyoming, minimal disease losses in infested fields could be attributed to low inoculum density or limited distribution of viruliferous *P. betae*. Furthermore, disease pressure and losses could increase in these states in the future. However, with our present knowledge of disease management options, rhizomania is not likely to ever cause losses in Colorado, Texas, Wyoming, or any other state as severe as those experienced in California when the disease first hit.

Disease control. Until the recent development of disease-tolerant germ plasm, cultural and chemical methods of disease control were the only means of reducing losses to rhizomania. Planting as early as possible, when soil temperatures are too cool for infection by *P. betae,* has been very effective in delaying the onset of disease. Since only juvenile or primary root tissue is susceptible to infection by *P. betae*, early planting allows establishment of the tap root before infection occurs (17). Although later infections will reduce sugar content, acceptable root yields may still be produced. Likewise, planting a field with transplants instead of seeding may result in increased sugar beet root yields (19). This method has been used in Japan but has not gained acceptance in the United States. Other cultural practices that have been suggested to reduce the incidence and severity of rhizomania or to limit the spread of the pathogen include limiting irrigation duration and increasing frequency, avoiding introduction of infested soil into clean fields, and lengthening crop rotations (4,16,23).

Despite the use of good cultural practices, losses to rhizomania can still be excessive, and additional control measures are often warranted. Soil fumigation and use of tolerant germ plasm have both been used successfully in reducing losses to rhizomania (22,39). The use of either of these control measures usually results in significantly improved yields. However,

Fig. 7. Close-up of typical symptoms caused by beet soilborne mosaic virus (BSBMV).

Fig. 8. Yellow vein symptom caused by beet soilborne mosaic virus (BSBMV) similar to that caused by beet necrotic yellow vein virus (BNYVV) (compare with Fig. 3).

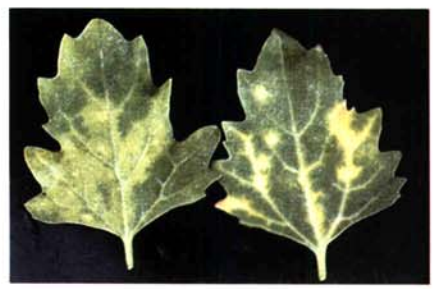


Fig. 9. *Chenopodium quinoa* infected by beet soilborne mosaic virus (BSBMV) and beet necrotic yellow vein virus (BNYVV). Leaf on left was mechanically inoculated with BSBMV and leaf on right with BNYVV.

Fig. 10. Typical symptoms of rhizomania possibly caused by beet soilborne mosaic virus (BSBMV). In 1994, numerous roots exhibiting rhizomania-like symptoms tested positive for BSBMV but negative for beet necrotic yellow vein virus (BNYVV) by ELISA.

Fig. 11. Symptoms on *Chenopodium quinoa* following inoculation by beet soilborne mosaic virus (BSBMV) and beet necrotic yellow vein virus (BNYVV) alone and in combination. The leaf on the left was inoculated with BSBMV, the leaf on the right with BNYVV, and the center leaf with a mixture of the two.

the level of tolerance in existing germ plasm may not be sufficient to produce a profitable sugar beet crop without fumigation, especially in fields where inoculum density of viruliferous *P. betae* is high or where other soilborne pathogens are present (20,22,39). Therefore, the use of tolerant cultivars and fumigation is generally recommended for growers forced to plant in infested fields. Still, even with this combination, producers should proceed with caution.

Harveson and Rush observed a cultivar × fumigation interaction in a field infested with multiple soilborne pathogens (22). Some cultivars with good tolerance to rhizomania were highly susceptible to fungal root rot pathogens such as *F. o.* f. sp. *betae* and *Aphanomyces cochlioides*. Even with fumigation, these cultivars were devastated by the fungal pathogens. This suggested that even if viruliferous *P. betae* is present in a field, it might be better to focus disease management practices on the primary fungal pathogens if inoculum density of viruliferous *P. betae* is low (20). It also pointed out the need to develop a simple method for quantifying viruliferous *P. betae* in field soils.

Beet Soilborne Virus

Beet soilborne virus (BSBV) was first identified in sugar beet roots from England in 1982 (27) and has since been found in Finland, Sweden, Germany, Belgium, and the United States (26). BSBV is rod-shaped and vectored by *P. betae* but has no serological relationship to BNYVV. Two serogroups of BSBV have been identified in Europe: Ahlum and Wierthe. The Wierthe serotype has only been found in Germany (36).

The genome of BSBV is bipartite and the RNAs are not polyadenylated. Thus, BSBV is more similar to the majority of furoviruses than to BNYVV. BSBV causes no obvious symptoms on sugar beet, but some researchers have reported reduced seedling growth in greenhouse studies (31).

In the United States, BSBV has not been reported from field-grown beets but has only been identified by baiting the virus from soil (37). It is apparently widespread throughout the U.S. sugar beet–growing areas but causes no obvious economic loss. For these reasons, the report of BSBV in the United States has generated little interest among researchers. Most research on this virus is presently being conducted in Germany and Great Britain.

Beet Soilborne Mosaic Virus

A complex of viruses infecting sugar beets was identified in Texas in 1988. These viruses were reported to be morphologically similar to BNYVV and transmitted by *P. betae* (38). Individual isolates from the complex were recovered and given designations such as TX7 and TX8, and polyclonal antisera were prepared to purified virions. The isolates were described as serologically identical to each other and distinct from BNYVV. However, there was some cross-reactivity with BNYVV antiserum (49,50).

In 1991–92, a disease survey revealed that TX7 serotypes were widespread throughout the sugar beet–growing areas of Texas (23). Since 1992, they have also been frequently identified in California, Colorado, Idaho, Nebraska, and Wyoming. TX7 serotypes are extremely common in Colorado, Texas, Nebraska, and Wyoming, and were sometimes confused with BNYVV in ELISA tests when rhizomania was first identified in these states (16).

The name beet soilborne mosaic virus was first used in 1993 to describe viral isolates within the TX7 serotype (49). However, because of the many similarities between BSBMV and BNYVV, there has been some speculation that BSBMV could possibly be a strain of BNYVV (42). Partial characterization of BSBMV has shown BSBMV and BNYVV are more similar to each other than to other members of the furovirus group (25,42; G. B. Heidel, C. M. Rush, T. L. Kendall, S. A. Lommel, and S. K. Manohar, *unpublished*). BNYVV and BSBMV are both vectored by *P. betae*, and both have at least four polyadenylated RNA species. They have similar particle lengths, host ranges, and coat protein molecular weights (25; G. B. Heidel, C. M. Rush, T. L. Kendall, S. A. Lommel, and S. K. Manohar, *unpublished*). Wisler et al. also reported partial serological cross-reactivity to BSBMV when using antiserum made from whole BNYVV capsid protein or the 42 K protein from BNYVV RNA 2 ORF 3 (49,50). BSBMV differs from BNYVV primarily in serological reactivity and in symptom expression on various host plants. Indeed, one of the primary reasons for designating BSBMV as a different virus from BNYVV was that isolates of BSBMV were not associated with rhizomania-like disease symptoms (50).

Symptom expression and disease development. Symptoms caused by BSBMV can be extremely variable (44; G. B. Heidel, C. M. Rush, T. L. Kendall, S. A. Lommel, and S. K. Manohar, *unpublished*). Plants exhibiting systemic foliar

Fig. 12. Symptoms on *Beta maritima* following inoculation with beet soilborne mosaic virus (BSBMV) and beet necrotic yellow vein virus (BNYVV) alone and in combination. (A) Bright yellow local lesions caused by BNYVV. (B) Necrotic spots caused by BSBMV. (C) The BSBMV symptom phenotype dominated on dual inoculated leaves.

symptoms are easiest to find in the field during September and October. Foliar symptoms occur at a much higher frequency than those caused by BNYVV, but the overall incidence of systemic infection by BSBMV is still very low. Common foliar symptoms of BSBMV include light green or yellow blotches and bands that follow primary leaf veins (Fig. 6). The bands can become bright yellow but are typically broader than the yellow-vein symptom caused by BNYVV (Fig. 7). Occasionally, systemically infected leaves exhibit a mottled or mosaic pattern or symptoms that are very similar to the vein banding symptom of BNYVV (Fig. 8). It is unknown whether the variety of foliar symptoms in sugar beets caused by BSBMV is due to environment, sugar beet cultivar, or genetic variation among viral isolates, but a similar degree of symptom variation can be observed on *Chenopodium quinoa.*

During the early stages of our research with BSBMV, most isolates produced diffuse, pale yellow local lesions on *C. quinoa*, easily differentiated from the bright yellow local lesions produced by BNYVV (Fig. 9). However, as more isolates were collected, it became apparent that symptom variation was not unusual. A variety of symptom phenotypes have been observed, and some of these have been indistinguishable from those caused by BNYVV (44). Symptom phenotype of BSBMV has been observed to change after repeated mechanical inoculations, as observed with BNYVV deletion mutants (28,33,34). Studies are underway to determine whether observed variation in symptoms is due to genetic variation in the viral genome or environmental conditions. These studies of symptom variation on *C. quinoa* may reveal valuable information that could relate to observed symptom variation on sugar beet roots.

It is usually impossible to visually identify a field-grown sugar beet that is infected by BSBMV unless foliar symptoms have developed. When beets with BSBMV foliar symptoms are collected, roots typically appear healthy with no noticeable abnormality of any kind. However, during the 1993 and 1994 growing seasons, some beet roots with classic symptoms of rhizomania (i.e., stunting, constriction, and bearding) tested negative by DAS-ELISA for BNYVV and positive for BSBMV (44) (Fig. 10). We are presently trying to determine whether the BSBMV isolates from these roots are truly capable of causing rhizomania-like symptoms or whether BSBMV came in after BNYVV and in some way displaced it. In preliminary greenhouse studies, certain isolates of BSBMV have significantly reduced growth of infected sugar beet seedlings compared to noninfested controls (24).

Conclusions and Future Research

The prospect that some isolates of BSBMV may be virulent is of the utmost concern to sugar beet growers and industry alike. Considering the widespread distribution of BSBMV throughout the western sugar beet–growing states, this concern is justified. The degree of variation in virulence among BSBMV isolates, the response of BNYVV-tolerant varieties to BSBMV, and how or whether BSBMV interacts with BNYVV and other soilborne pathogens must be elucidated as soon as possible.

From a more optimistic viewpoint, most isolates of BSBMV may have minimal effects on sugar beets. It has recently been shown that *P. betae* does not move as rapidly through the soil as once thought (21,48). The widespread distribution of BSBMV throughout the western sugar beet–growing states implies that this virus has been around for a long time. Therefore, instead of having a detrimental effect on sugar beet production, some BSBMV isolates may be beneficial and, in some way, interfere with infection by BNYVV. BSBMV could compete for recognition sites in the host or vector or even possibly confer resistance somewhat akin to natural cross-protection. Prillwitz and Schlosser (40) reported that BSBV could interfere with infection by BNYVV and reduce symptom severity. In greenhouse experiments, sugar beets were inoculated with BSBV and later challenged with BNYVV. Protected plants had lower BNYVV titers, and taproot weights were increased by 50% compared to plants infected with BNYVV alone. Since BSBMV is more closely related to BNYVV than BSBV, it is possible that BSBMV might also affect infection by BNYVV. Preliminary studies in our lab have shown that when BSBMV and BNYVV are coinoculated onto *C. quinoa* or *Beta maritima,* BSBMV interferes with BNYVV symptom expression, and the BSBMV symptom phenotype predominates (42) (Figs. 11 and 12). A similar response was observed between BNYVV RNA 3 deletion mutants and wild type isolates of BNYVV (29). When the two are inoculated onto test plants, the diffuse pale yellow local lesion of the mutant predominates and the mutant inhibits development of the bright yellow lesion caused by the wild type virus. Although BSBMV has not been shown to interfere with infection by BNYVV under field conditions, the results of these studies are encouraging, and the interactions between BSBMV and BNYVV warrant further study.

In the United States, much of the future

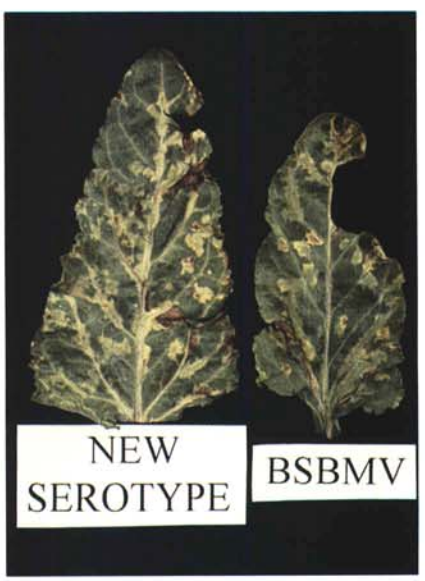


Fig. 13. Foliar symptoms produced by beet soilborne mosaic virus (BSBMV) and another closely related but serologically distinct virus isolate.

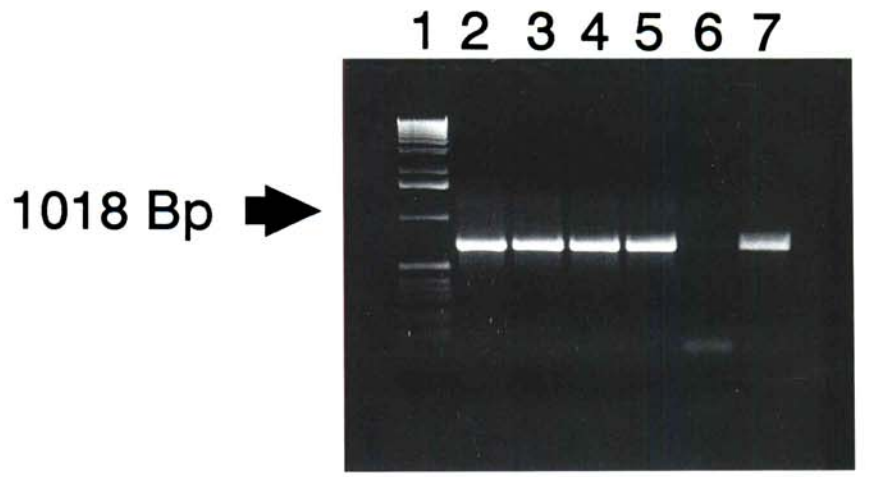
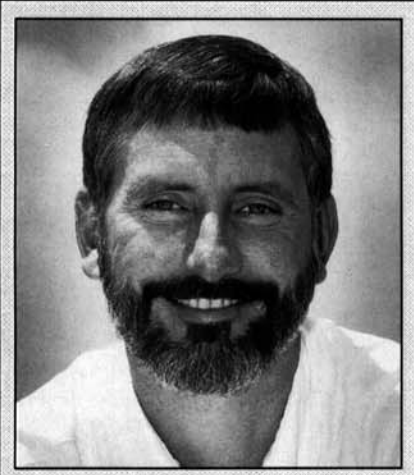


Fig. 14. Reverse transcriptase-polymerase chain reaction (RT-PCR) products amplified from nucleic acid extracts of beet soilborne mosaic virus (BSBMV) or the new serotype using primers developed for BSBMV. The isolates used to produce products in Lanes 2, 3, and 7 tested positive for BSBMV by ELISA. Those in Lanes 4 and 5 tested negative, and Lane 6 was the healthy control.



Charlie Rush

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Gretchen Heidel

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research on furoviruses of sugar beets will, of necessity, be very applied. However, there are numerous aspects of these viruses that require more fundamental investigation. For instance, the true taxonomic relation of BSBMV to BNYVV is an important question that must be resolved. There are undeniably major differences between these two viruses but also major similarities. The fact that both have quadripartite genomes and are polyadenylated separate them from all other furoviruses. Therefore, it might be appropriate to place these two in a distinct subgroup of the furoviruses.

During the 1994 growing season, several new viral isolates were recovered that produce foliar symptoms on sugar beet and *C. quinoa* that are indistinguishable from symptoms caused by BSBMV (Fig. 13). These viruses are quadripartite; the coat protein is approximately 22 kDa; and when used as templates in conjunction with primers generated specifically for BSBMV, they produce a product of the expected size for BSBMV (44) (Fig. 14). However, these new isolates are serologically distinct from BNYVV and BSBMV. Considering the known variability among isolates of BSBMV in symptom expression, it is not surprising that serotype variability also exists. It is conceivable that the quadripartite furoviruses of sugar beet in the United States constitute one large heterogeneous population that varies, among other things, in serotype, pathogenicity, and virulence. A continuum may exist with one end of the spectrum represented by highly virulent BNYVV types, the other end by avirulent BSBMV types, and an unknown number of variants in between. On the other hand, BNYVV, BSBMV, and the new serotype may represent truly distinct but related populations with significant levels of intrapopulation variation. Whatever the case, these viruses present a unique opportunity for study of variability and population dynamics of furoviruses.

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