Effect of Temperature on Susceptibility of Normal and Aberrant Ratio Corn Stocks to Barley Stripe Mosaic and Wheat Streak Mosaic Viruses

Myron K. Brakke and Richard Samson

Research chemist, Agricultural Research, Science and Education Administration, U.S. Department of Agriculture and also professor, Department of Plant Pathology, University of Nebraska; and student, University of Nebraska, Lincoln 68583.

Cooperative Investigations, Agricultural Research, Science and Education Administration, U.S. Department of Agriculture and Department of Plant Pathology, Nebraska Agricultural Experiment Station, Lincoln 68583. Published with the approval of the director as Journal Series Paper 6019 of the Nebraska Agricultural Experiment Station.

Accepted for publication 22 January 1981.

ABSTRACT

Brakke, M. K., and Samson, R. 1981. Effect of temperature on susceptibility of normal and aberrant ratio corn stocks to barley stripe mosaic and wheat streak mosaic viruses. Phytopathology 71:823-824.

Corn was more susceptible to systemic infection by barley stripe mosaic virus at 25 C than at 35 C, but cultivar Black Hulless barley was equally susceptible at both temperatures. Some corn lines were more susceptible to wheat streak mosaic virus at 35 C than at 25 C, but other lines were equally susceptible at those temperatures. Plants of corn stocks with a virus-induced mutation, aberrant ratio, were susceptible to infection by the inducing virus.

Aberrant ratio (AR) is a virus-induced mutation caused in corn by barley stripe mosaic virus (BSMV), wheat streak mosaic virus (WSMV), and corn lily fleck virus (5–7). AR has been reported only in progeny from virus-infected plants of one multiply marked dominant stock that was selected because it was susceptible to BSMV. Because infected plants set few kernels, they were used as male parents to pollinate silks of a corresponding recessive stock (5), which happened to be resistant to BSMV and WSMV.

The AR effect is manifested as a distortion in expected segregation ratios. The phenomenon has persisted in ten generations of the progeny of AR stock. One possible explanation of the AR effect is that it results from persistence of the virus genome in these plants; however, no virus has been detected in AR plants (3,5).

The purpose of this research was to investigate the susceptibility of plants of AR stocks to infection by BSMV and WSMV to find whether the failure to recover virus may be caused by the inability of the plants to support multiplication of virus to detectable levels.

MATERIALS AND METHODS

AR corn (*Zea mays* L.) stocks and related lines obtained from G. F. Sprague (4) included the original male and female lines that were used to establish AR; a control F_1 cross from plants of the above lines that were not infected with virus; 67:120, a line showing AR in the "a" phenotype derived from a BSMV-infected male parent; and 74:130, a line showing AR at the "su" phenotype derived from a WSMV-infected male parent. Cultivar Golden Cross Bantam sweet corn was obtained from commercial sources and inbred Ohio 28 was from the Ohio Agricultural Research and Development Center, Wooster.

A mixture of three isolates of WSMV recovered from field corn in 1972 and strains ND18 and Argentine Mild of BSMV were used. No differences were noted between the two BSMV strains; hence a single set of results will be presented. Both strains have been used to induce AR.

Except as noted otherwise, plants were grown in controlled environment growth chambers with 8 hr of darkness and 16 hr of cool-white fluorescent light (20,000 lm/m^2) per day. Plants were inoculated by rubbing leaves with extracts of BSMV-infected barley or WSMV-infected wheat ground in water (1 g of leaves in 5 ml) with 1% Celite. Infections were usually judged by symptoms, but many recovery attempts were made by inoculating barley (*Hordeum vulgare* L. 'Black Hulless') for BSMV and wheat (*Triticum aestivum* L. 'Michigan Amber') for WSMV.

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. § 1734 solely to indicate this fact.

RESULTS

BSMV rarely infected corn plants of any of the lines at 35 C, but at 25 C it infected plants of all except the female line (Table 1). Black Hulless barley was equally susceptible to BSMV at both 25 and 35 C. On the other hand, WSMV infected plants of all the lines at 35 C but did not infect plants of Golden Cross Bantam at 25 C, and infected only one of 10 plants of the female line at 25 C.

BSMV and WSMV were recovered from all symptomatic corn plants that were tested by inoculation to barley and wheat plants, respectively. No virus was recovered from symptomless plants. Attempts were made to recover BSMV from 30 symptomless plants of the AR stock (67-120) derived from a BSMV-infected pollen

TABLE 1. Susceptibility of some corn lines to wheat streak mosaic virus (WSMV) and barley stripe mosaic virus (BSMV) at 25 C and 35 C

	Virus used for inoculation					
	BSMV		WSMV		None	
Plant	25 C	35 C	25 C	35 C	25 C	35 C
Corn						
AR(BSMV) *a* phenotype	34/100[a]	1/93	16/19	12/16	0/97	0/77
AR(WSMV) *su* phenotype	5/16	0/17	7/22	12/13	0/17	0/12
F_1, non-AR	2/11	0/10	4/12	8/12	0/13	0/11
Female parent line	0/10	0/6	1/10	9/10	0/11	0/8
Male parent line	8/10	0/9	6/10	11/11	0/8	0/10
Ohio 28	2/6	0/5	7/7	6/6	0/5	0/5
Ohio 28[b]			14/47	20/37	0/11	0/11
Golden Cross Bantam	10/40	1/39	0/9	12/13	0/34	0/43
Golden Cross Bantam[b]			0/42	27/42	0/21	0/21
Barley						
Black Hulless	75/105	52/81			0/89	0/104

[a]Numerator is number of plants with mosaic symptoms and denominator is number of plants inoculated. Combined results of four experiments.

[b]Combined results of three experiments in which the plants at 25 C were in the greenhouse with an average temperature of 25 C and the plants at 35 C were in a growth chamber.

TABLE 2. Effect of incubation of Golden Cross Bantam sweet corn plants at 35 C after inoculation with barley stripe mosaic virus on subsequent development of symptoms at 25 C

Number of days at 35 C	Expression of symptoms at 25 C	
	Inoculated	Uninoculated
0	27/37[a]	0/17
1	29/51	0/17
2	20/34	0/15
3	7/47	0/36
7	0/25	
14	0/33	
1[b]	25/37	0/12
1[c]	9/12	0/17
2[b]	24/24	

[a]The numerator is the number of plants with mosaic and the denominator is the number that were inoculated. Combined results of two experiments.
[b]These plants were at 25 C in the light for 1 day and were then moved to 35 C for 1 or 2 days before being returned to 25 C.
[c]These plants were at 25 C in the light for 2 days before being incubated at 35 C for 1 day.

TABLE 3. Effect of incubation of Golden Cross Bantam sweet corn plants at 25 C after inoculation on subsequent development of symptoms of barley stripe mosaic virus

Time after inoculation at 25 C in dark	Expression of symptoms at		
	Inoculated		Uninoculated
	25 C	35 C	25 C
0 day	15/36[a]	0/36	0/24
2 day	17/36	1/44	0/36
3 day	34/43	3/35	0/19
4 day	18/34	4/17	0/24

[a]Numerator is number of plants showing mosaic and denominator is number inoculated. Combined results of three experiments.

parent and to recover WSMV from 20 symptomless plants of the AR stock (74:130) derived from a WSMV-infected pollen parent. Some of these plants had been inoculated manually, and some had not, but virus was recovered from none of them. Correlation between symptoms and the ability to recover virus was perfect.

The effect of the length of exposure to high temperature on the infection of corn by BSMV was investigated by temperature-shift experiments. Plants kept for as long as 3 days at 35 C starting immediately after inoculation still showed symptoms after being placed at 25 C, although the percentage of plants showing symptoms was less with longer incubation at 35 C (Table 2). Plants kept for 7 or 14 days at 35 C after inoculation did not show symptoms when subsequently kept at 25 C. Exposure of plants to 25 C for 1 or 2 days after inoculation, then to 35 C for 1 or 2 days, before placing them at 25 C did not prevent development of symptoms (Table 2). Only a few plants kept in the dark for 2, 3, or 4 days at 25 C showed symptoms of BSMV when they were subsequently moved to 35 C (Table 3).

Young, emerging leaves of BSMV-infected plants kept at 25 C were free of symptoms on nearly half of the plants as they became older. All of the young leaves were symptomless on plants kept at 35 C for 1 wk or more after they had become systemically infected at 25 C. Pring (3) also reported that BSMV did not remain fully systemic in corn at 25 or 32 C.

DISCUSSION

These data show that plants of AR corn stocks are susceptible to the viruses originally used to induce AR. No uninoculated plants of AR stocks developed virus symptoms and no infectious virus was recovered from them. Their susceptibility to inoculation shows that they were not resistant to virus. If these AR plants had contained seedborne virus, the virus should have multiplied to detectable levels and symptoms should have developed. The evidence strengthens previous conclusions (3,5) that AR plants do not have seedborne virus.

Failure to infect all of the plants of AR stocks probably was due to inefficient inoculation or too low a concentration of virus, although the possibility of segregation for resistance in these populations has not been eliminated. Genes for resistance could have been inherited from the female line. Conceivably, resistance could also derive from the AR phenomenon itself, if this phenomenon results from presence of the virus genome in a condition analogous to that of temperate bacteriophage in lysogenic bacteria. Such bacteria are resistant to reinfection (2). However, there is no evidence suggesting that AR is caused by a mechanism similar to lysogeny, and no phenomenon similar to lysogeny is known in plants. We have attempted to test for such a phenomenon by screening 2,945 wheat plants from seed harvested from wheat infected with WSMV for susceptibility to WSMV. All were susceptible (*unpublished*; seed collected by C. L. Niblett and E. Kerr).

The question whether every plant in an AR stock carries the mutation is pertinent to the question whether the uninfected plants of the present experiments might carry AR-mediated resistance to virus. The data of Sprague and McKinney (5,6) suggest that not all plants of AR stocks express the mutation. Samson et al (4), however, showed that probably all of the plants of the particular AR stocks used in the present experiments carry and express the mutation. In some cases, the mutation, which is postulated to be a gene inactivation, was not expressed in reciprocal crosses between plants of AR stocks, but was expressed in crosses to tester plants.

The failure of BSMV infection to develop in corn at 35 C is a property of this particular host-virus combination. Corn supported WSMV infection at 35 C, and in some cultivars better than at 25 C. BSMV infected barley at 35 C, even though barley grows very poorly at this temperature and its chloroplasts do not develop normally (1). BSMV did not seem to be rapidly inactivated in corn at 35 C, but survived for at least a few days and resumed multiplication when the temperature was lowered. The high temperature may inhibit movement of the virus or some other step essential to development of systemic infection, rather than virus multiplication.

LITERATURE CITED

1. Feierabend, J., and Mikus, M. 1977. Occurrence of a high temperature sensitivity of chloroplast ribosome formation in several higher plants. Plant Physiol. 59:863-867.
2. Luria, S. E., and Darnell, J. E., Jr. 1967. General Virology. 2nd ed. John Wiley & Sons, New York.
3. Pring, D. R. 1974. Barley stripe mosaic virus infection of corn and the "aberrant ratio" genetic effect. Phytopathology 64:64-70.
4. Samson, R. G., Brakke, M. K., and Compton, W. A. 1979. Evidence for gene inactivation in the virus-induced aberrant ratio phenomenon in maize. Genetics 92:1231-1239.
5. Sprague, G. F., and McKinney, H. H. 1966. Aberrant ratio: an anomaly in maize associated with virus infection. Genetics 54:1287-1296.
6. Sprague, G. F., and McKinney, H. H. 1971. Further evidence for the genetic behavior of AR in maize. Genetics 67:533-542.
7. Sprague, G. F., McKinney, H. H., and Greeley, L. W. 1963. Virus as a mutagenic agent in maize. Science 141:1052-1053.