INFORMATION TO USERS

This material was produced from a microfilm copy of the original document. While the most advanced technological means to photograph and reproduce this document have been used, the quality is heavily dependent upon the quality of the original submitted.

The following explanation of techniques is provided to help you understand markings or patterns which may appear on this reproduction.

1. The sign or "target" for pages apparently lacking from the document photographed is "Missing Page(s)". If it was possible to obtain the missing page(s) or section, they are spliced into the film along with adjacent pages. This may have necessitated cutting thru an image and duplicating adjacent pages to insure you complete continuity.

2. When an image on the film is obliterated with a large round black mark, it is an indication that the photographer suspected that the copy may have moved during exposure and thus cause a blurred image. You will find a good image of the page in the adjacent frame.

3. When a map, drawing or chart, etc., was part of the material being photographed the photographer followed a definite method in "sectioning" the material. It is customary to begin photoing at the upper left hand corner of a large sheet and to continue photoing from left to right in equal sections with a small overlap. If necessary, sectioning is continued again — beginning below the first row and continuing on until complete.

4. The majority of users indicate that the textual content is of greatest value, however, a somewhat higher quality reproduction could be made from "photographs" if essential to the understanding of the dissertation. Silver prints of "photographs" may be ordered at additional charge by writing the Order Department, giving the catalog number, title, author and specific pages you wish reproduced.

5. PLEASE NOTE: Some pages may have indistinct print. Filmed as received.

University Microfilms International
300 North Zeeb Road
Ann Arbor, Michigan 48106 USA
St. John's Road, Tyler's Green
High Wycombe, Bucks, England HP10 8HR

THE EFFECT OF ACCESSORY CHROMOSOMES ON THE RESISTANCE OF MAIZE TO VIRAL INFECTION

by

Scott Craig McGirr

A Dissertation Submitted to the Faculty of the

COMMITTEE ON GENETICS (GRADUATE)

In Partial Fulfillment of the Requirements
For the Degree of

DOCTOR OF PHILOSOPHY

In the Graduate College

THE UNIVERSITY OF ARIZONA

1 9 7 7

THE UNIVERSITY OF ARIZONA

GRADUATE COLLEGE

I hereby recommend that this dissertation prepared under my direction by Scott Craig McGirr

entitled The Effect of Accessory Chromosomes on the Resistance of Maize to Viral Infection

be accepted as fulfilling the dissertation requirement for the degree of Doctor of Philosophy

J E Endrizzi — Dissertation Director — Aug 1, 1977 — Date

As members of the Final Examination Committee, we certify that we have read this dissertation and agree that it may be presented for final defense.

J E Endrizzi	Aug 1, 1977
W P Bemis	Aug 1, 1977
R M Harris	8/22/77

Final approval and acceptance of this dissertation is contingent on the candidate's adequate performance and defense thereof at the final oral examination.

STATEMENT BY AUTHOR

This dissertation has been submitted in partial fulfillment of requirements for an advanced degree at The University of Arizona and is deposited in the University Library to be made available to borrowers under rules of the Library.

Brief quotations from this dissertation are allowable without special permission, provided that accurate acknowledgment of source is made. Requests for permission for extended quotation from or reproduction of this manuscript in whole or in part may be granted by the head of the major department or the Dean of the Graduate College when in his judgment the proposed use of the material is in the interests of scholarship. In all other instances, however, permission must be obtained from the author.

SIGNED: Scott Craig McGirr

I would like to dedicate this manuscript

to my wife, Mary

ACKNOWLEDGMENTS

I would like to express my sincere appreciation and thanks to Dr. John E. Endrizzi. The suggestions, constructive criticism and expertise that he provided were invaluable toward the completion of this research and the writing of this dissertation. His encouragement and support were given throughout the research project and for this, as well as his freely given time and enthusiastic help, I am truly grateful.

Appreciation is also given to the people that provided technical assistance. A great deal of help was given by Mr. Grant M. Ramsay in the cytological and photographic aspects of this work. Technical advice with the handling of the virus cultures was given by Dr. Merritt R. Nelson. The electron micrographs used for the verification of the virus cultures were made by Mr. Raymond E. Wheeler. Help in the design and analysis of experiments was kindly provided by Dr. Robert O. Kuehl. Assistance in the preparation of this manuscript was generously given by Dr. Robert M. Harris and Dr. William P. Bemis.

For the seed stocks they provided I am indebted to Dr. R. Thomas Ramage, Mr. Frank B. Crookham, Dr. R. J. Lambert, Dr. G. F. Sprague and Dr. Ellen Dempsey. Additional thanks is given to Dr. Myron K. Brakke and Dr. R. E. Ford for the virus cultures they provided.

Financial support of this project was given by the Arizona Foundation. I wish to express my gratitude to the Foundation personnel and to the people who support this organization.

I would like to give special thanks to my wife, Mary, who lovingly gave of her time not only to help me throughout my studies but to complement and enrich my life. Appreciation is given to my father, Dr. John I. McGirr, whose generosity, perseverance and creativity have been an inspiration to me. I cannot thank him enough for the many years of love and support he has given me. I am also grateful to my wife's family for the warmth and kindness they have always extended to me.

TABLE OF CONTENTS

LIST OF ILLUSTRATIONS

LIST OF TABLES

LIST OF TABLES--Continued

ABSTRACT

Tests were conducted to determine if the accessory (B) chromosomes, the abnormal tenth chromosome (K10) or the aberrant ratio (AR) phenomenon affect the resistance of maize (Zea mays L.) to viral infection. Genetically similar stocks of maize with and without these elements were compared to determine their effect upon the plants response to Brome Mosaic Virus (BMV), Maize Dwarf Mosaic Virus (MDMV), Wheat Streak Mosaic Virus (WSMV) and Barley Stripe Mosaic Virus (BSMV). None of the test results with BSMV were found to be conclusive. Neither the B or K10 chromosomes altered the resistance of maize to BMV and MDMV infection. However, these chromosomes were found to affect the resistance of the plant lines tested to infection with WSMV. The AR phenomenon was not found to alter the resistance of maize to infection with BMV. However, it was found to affect the resistance of the plant lines tested to both MDMV and WSMV infection.

Cross protection experiments were conducted to determine if viruses become integrated into the genome of maize and if so, whether or not the B or the K10 chromosomes would affect this process. The progeny of reciprocal crosses of healthy and viral infected plants with and without B or K10 chromosomes were compared with respect to viral susceptibility. No differences were found between the lines of maize that would be indicative of the integration of cross protective viruses.

INTRODUCTION

The intent of this study was to determine if accessory or B chromosomes of maize affect the resistance of the plant to viral infection. Since B chromosomes alter the cellular synthesis of both nucleic acids and proteins in maize (Ayonoadu and Rees 1971) viral replication within this cellular environment could be affected. The abnormal tenth chromosome (K10) of maize may be related to the B chromosome (Ting 1958) and was therefore included in the study. In order to test the hypothesis that B or K10 chromosomes directly affect the resistance of maize to viral infection, genetically similar stocks of maize with and without B or K10 chromosomes were compared with respect to susceptibility to Brome Mosaic Virus (BMV), Maize Dwarf Mosaic Virus (MDMV), and Wheat Streak Mosaic Virus (WSMV).

Viruses have been shown to become incorporated in the chromosomes of hosts and to be subsequently inherited in an inactive state or as a provirus (Campbell 1962). While proviruses have been shown to exist in microorganisms (Campbell 1962) and animals (Huebner and Todaro 1969) they have not been shown to exist in plants. However it is thought by some researchers that proviruses do exist in maize and that the aberrant ratio (AR) phenomenon may result from viruses being integrated near marker genes (Sprague and McKinney 1966). This idea could be verified if the integrated viruses exhibit properties of cross protection. Cross protection is the phenomenon whereby the presence of

one pathogen in an organism prevents a secondary infection by a similar pathogen. In order to test the hypothesis that cross protective proviruses exist in plants exhibiting the AR phenomenon, stocks of maize having similar genetic backgrounds with and without the AR effect were compared with respect to susceptibility to BMV, MDMV, WSMV and Barley Stripe Mosaic Virus (BSMV).

If viruses capable of cross protection do become integrated into the chromosomes of maize, then the B or K10 chromosomes could affect this process and consequently the susceptibility of the plant to subsequent viral infections. The B or K10 chromosomes could affect the integration of viruses in two ways. First, recombinational events between homologous regions of host and viral genomes are believed to lead to the integration of viruses (Campbell 1962). Because both the B chromosomes (Hanson 1969) and the K10 chromosomes (Rhoades and Dempsey 1957) increase recombination in maize, they could then also enhance viral integration. Second, each accessory chromosome increases the amount of cellular DNA by 5% (Ayonoadu and Rees 1968), and the K10 chromosomes similarly increase the amount of DNA per cell. These chromosomes could increase the amount of cellular DNA available for random integration of viruses or they could contain regions of DNA homoeologous to the viral genomes and offer specific sites for incorporation. To investigate these possibilities the progeny of infected plants with and without the B or K10 chromosomes were compared with respect to viral susceptibility. Reciprocal crosses of infected and healthy plants were made to test for male and female differences in the possible inheritance of proviruses.

Accessory chromosomes of many species and particularly maize are largely heterochromatic. Since the initiation of this research, Hsu (1975) proposed the hypothesis that heterochromatin might function to protect the more vital euchromatin of cells from mutagens and viruses. This study could provide information on the validity of this hypothesis.

In the last decade a great deal has been learned about the properties of B chromosomes. However, it is still not known why they exist in natural populations when increasing numbers of B's within an organism usually decrease its viability. One view is that B chromosomes are maintained in populations exclusively by mechanisms of meiotic drive (Randolph 1941; Ostergren 1945). Another view is that a selective advantage may exist for organisms containing B chromosomes over those without them (Darlington and Upcott 1941). This study could indicate if maize plants with B chromosomes have a selective advantage against viral infection over those plants without them.

This study will indicate if accessory chromosomes directly affect the resistance of maize to viral infection. If they increase the resistance of maize to viral infection, a selective advantage could be shown for their maintenance in natural populations. It would also reveal if the abnormal tenth chromosome similarly alters the resistance of maize to viral infection. This could offer a clue to the possible evolutionary relationships of the B and K10 chromosomes. The study will also reveal if the aberrant ratio phenomenon in maize is induced by the integration of cross protective viruses. If cross

protective provirus do exist, it will reveal the effect of B and K10 chromosomes upon their viral integration. The results should indicate if these chromosomes enhance viral integration by increasing recombination or by offering additional sites for viral incorporation.

LITERATURE REVIEW

Accessory Chromosomes

Accessory (supernumerary or "B") chromosomes were first identified in sweet corn by Kuwado (1915) by virtue of differing somatic chromosome counts. In addition to sweet corn, B chromosomes have been observed by Longley (1927) in flour and dent corn and by Kozhuchow (1933) in flint corn and popcorn. Examining native Indian maize varieties Longley and Kato (1965) observed the presence of B chromosomes not only in North, Central and South American stocks but also in those from the Caribbean Islands. Maize is not alone in possessing B chromosomes. They have been reported in 608 plant species and 116 animal species (Rees, 1974).

Randolph (1928) coined the term "B" chromosome to describe the chromosomes not essential to an organism in contrast to "A" chromosomes which represent the regular members of the chromosome complement. Cytological examination of somatic metaphase cells reveals that the B chromosomes in maize are shorter and stain more intensely than A chromosomes.

While small numbers of B chromosomes have not been found to alter the phenotype of maize, large numbers do decrease vigor and fertility (Randolph 1941). Darlington and Upcott (1941) considered that B chromosomes might confer a selective advantage upon plants possessing them to compensate for the meiotic losses they observed. However, Randolph (1941) noticed an increase of B chromosomes in experimental

crosses and conjectured that a meiotic drive mechanism might be responsible for their maintenance in natural populations. It was postulated that B chromosomes of maize are parasitic in nature, functioning only in their own propagation by means of meiotic drive (Ostergren 1945).

Using A to B translocations labeled with marker genes, B chromosomes in maize were shown to undergo nondisjunction at the second pollen grain mitosis 80% of the time (Roman 1948). In the same study it was discovered that 75% preferential fertilization of the pollen containing B chromosomes over the pollen without them resulted in a selective advantage of 60% in favor of the B chromosomes. Blackwood (1956) cytologically confirmed this genetic study and found that the degree of nondisjunction and preferential fertilization varied greatly depending upon the maize variety tested. Studies with A to B translocations have shown that nondisjunction was restricted to the translocated chromosome containing the B centromere (B^A) and that it does not occur in the absence of the reciprocally translocated chromosome (A^B). It has been concluded that nondisjunction of B chromosomes occurs at the centric heterochromatic region and is regulated by the distal euchromatic region (Roman 1950; Longley 1956; Carlson 1969; Ward 1973b).

Accessory chromosomes have been found to increase the frequency of recombination at specific regions of chromosomes 3 and 9 (Hanson 1969). Recombination at specific regions of chromosome 5 has been shown to increase proportionally to the number of B chromosomes present (Nel 1969). Cytological analysis revealed an increase in the chiasma

frequency at Metaphase I of microsporogenesis in Black Mexican Sweet Corn (Ayonoadu and Rees 1968). The promotion of crossing over was found to be greater in microsporocytes than in megasporocytes on chromosomes 5 and 9 (Nel 1973). Using A to B translocations, Ward (1973c) showed that the heterochromatic region of the B chromosomes was responsible for the increased recombination of A chromosomes. He found the amount of increase in recombination to be proportional to the quantity of heterochromatin in the B chromosome present. Odd numbers of B chromosomes have been shown to enhance crossing over to a greater extent than even numbers of B chromosomes (Rhoades and Dempsey 1972; Chang and Kikudome 1974).

Accessory chromosomes in all organisms should be assumed to have arisen from the regular chromosome complement (Rees 1974). Studies have shown that the nucleic acid of B chromosomes has a higher GC base composition than A chromosomes in seeds (Van Schaik and Pitout 1966) but not in leaf tissue (Pitout and Van Schaik 1968) nor in seedlings (Chilton and McCarthy 1973) of Black Mexican Sweet Corn. By examination of root meristem tissue of Black Mexican Sweet Corn, Ayonoadu and Rees (1971) revealed that while B chromosomes contained the same quantity of DNA as the average A chromosome they were 1.7 times the density at metaphase. They also found that while the amount of histone protein per cell continues to increase along with the frequency of B chromosomes, nuclear RNA and total protein content increased with the addition of up to four B chromosomes and then decreased. Nuclear RNA and total protein were disproportionately high in plants with even numbers of B chromosomes while the opposite was true of histone protein.

Abnormal Tenth Chromosome

The abnormal tenth chromosome of maize was first described by Longley (1937) as having a large terminal knob and a longer heterochromatic arm than the more common tenth chromosome. It was discovered in native Indian maize varieties from North, Central and South America and also the Caribbean Islands (Longley and Kato 1965). This abnormal tenth chromosome has since been referred to as the knobbed chromosome 10 (K10) and the more common chromosome 10 as the knobless or normal one (k10).

In plants heterozygous for the abnormal tenth chromosome (K10 k10) preferential segregation resulted in 70% of the functional megaspores receiving the K10 chromosome whereas 50% was expected with random segregation (Rhoades 1942). However, it was found that the K10 pollen competed poorly with the k10 pollen. In addition, preferential segregation occurred in several pairs of chromosomes heterozygous for knobs in the presence of the abnormal tenth chromosome (Longley 1945). Secondary centric regions or neocentromers formed between homologous chromosomes in plants heterozygous for the K10 chromosome, which may explain the preferential segregation observed (Rhoades 1952). In the presence of an abnormal tenth chromosome the degree of preferential segregation of the knobbed chromosome 9 over the knobless one quantitatively increased along with an increase in the knob size (Kikudome 1957).

Crossing over in maize was found to be increased by the presence of the abnormal tenth chromosome, but the increase was not uniform

throughout the genome (Rhoades and Dempsey 1957 and 1966). The increases in crossing over promoted by the K10 chromosome were greater in the female than the male sporocytes (Nel 1973; Ward 1973a).

In 1958, Ting hypothesized that the K10 chromosome in maize was the product of a B to A translocation. He based his conclusion on the similar appearance of the terminal heterochromatic region of the K10 chromosome to that of the B chromosomes observed at pachytene of meiosis. He also pointed out that the neocentric region of the K10 chromosome could be the vestiges of the B chromosomal centromere.

Aberrant Ratio Phenomenon

Sprague and McKinney (1966) discovered that Barley Stripe Mosaic Virus (BSMV) could infect maize plants and cause unexpected genetic ratios of some of the progeny plants. When inoculated homozygous dominant plants (AA) were used as males and crossed to homozygous recessive female plants (aa), the self-pollinated F_1 (Aa) produced F_2 progeny that would occasionally exhibit unexpected ratios other than the expected 3:1 (A_:aa). They termed this phenomenon aberrant ratio or AR and demonstrated that it occurred in the progeny of about one in two hundred systemically infected plants and could persist indefinitely. They noted that backcrosses exhibited different degrees of AR ranging from as little as 60:40 to as much as 95:5 for either genotype when 50:50 was expected. Unlike other cases of meiotic drive, male and female transmission was equally aberrant. Later Sprague and McKinney (1971) found that Wheat Streak Mosaic (WSMV) and Lily Fleck Corn Virus

(LFCV) could also induce AR but that Maize Dwarf Mosaic Virus strain-B (MDMV-B) would not.

The AR action could affect either allele of a single gene or it could switch to another previously unaffected gene. It was postulated by Sprague and McKinney (1966) that the existence of an episomal like particle, possibly viral in nature, located adjacent to the marker genes could promote the AR phenomenon. Viruses that have become incorporated into a chromosome and inherited in the inactive state are commonly referred to as proviruses. To investigate this possibility Pring (1974) analyzed maize plants infected with BSMV and observed that while a single stranded RNA similar to BSMV was present in the maize tassels of infected plants, none was detectable in the pollen or seed of these plants. Although the existence of proviruses has been documented in microorganisms (Campbell 1962) and animals (Huebner and Todaro 1969), this would be the first case reported of a provirus in plants if Sprague and McKinney's postulation was correct.

Maize Viruses

Brome Mosaic Virus (BMV), Maize Dwarf Mosaic Virus (MDMV), Wheat Streak Mosaic Virus (WSMV) and Barley Stripe Mosaic Virus (BSMV) all occur worldwide in temperate regions. They infect several monocotyledonous plants and can be readily transmitted to maize by means of mechanical inoculation with crude sap.

Brome Mosaic Virus (BMV) consists of isometric particles 25 nm in diameter (Fig. 3a, page 23) containing a single strand of RNA (Bancroft 1970). It is transmitted by nematodes (Xiphinema spp.) to

many monocotyledonous and a few dicotyledonous plants. Seedlings of most varieties of maize develop primary lessions followed by systemic necrosis and death within one to two weeks after infection.

Maize Dwarf Mosaic Virus (MDMV) consists of flexous rods 750 nm in length (Fig. 3b, page 23) which contain one piece of single stranded RNA (Pring and Langenberg 1972). There are two common MDMV strains. Strain A is identical to strain B in most respects except that the former can infect Johnsongrass and the latter cannot. MDMV is very closely related to Sugar Cane Mosaic Virus (Snazelle, Bancroft and Ullstrup 1971; Ford 1974). MDMV is transmitted nonpersistently by aphids (Messieha 1967) and infects many monocotyledonous plants. The virus has been shown to be seed born in one variety of maize at a frequency of 0.2% (Hill, Martinson and Russell 1974). Seedlings of maize do not develop local lessions but will show systemic necrosis within a week of inoculation. The virus will cause mild stunting in maize.

Tu and Ford (1970) found the concentration of MDMV to be the same in susceptible Seneca Chief and resistant Illinois A varieties of maize by using dilution end point assays. Since the former variety had systemic symptoms and the latter did not, they concluded that the mechanism of genetic resistance is in the movement of the virus rather than its multiplication. This conclusion was supported by similar experiments conducted by Jones and Tolin (1972).

A comparison was made of the degree of resistance to MDMV of double cross hybrids from eight inbred lines of maize (Zuber et al. 1973). It was concluded that resistance was inherited in a quantitative fashion and postulated that several genes with additive effects control

resistance. These conclusions were also reached by similar experiments conducted by Genter (1973).

In a study with chromosomal translocations it was determined that genes for resistance to MDMV are carried on both arms of chromosome 6 (Scott and Nelson 1971). This conclusion was supported by Findley et al. (1973), who in addition revealed that genes for resistance were located on the long arms of chromosomes 1, 2 and possibly 10 and the short arms of chromosomes 3, 7, 8 and 10.

Wheat Streak Mosaic Virus (WSMV) consists of flexous rods 750 nm in length (Fig. 3c, page 23) which contain one piece of single stranded RNA (Brakke 1971). It is transmitted persistently by the eriophyid mite, Aceria tulipae K. (Rosario and Del 1959) and infects many monocotyledonous and a few dicotyledonous plants. WSMV has been shown to be seed born in maize at a frequency of 0.1% (Hill et al. 1974). Maize varieties have shown a great deal of variation in susceptibility to WSMV. McKinney (1949) found the majority of the varieties he tested of sweet corn (9 out of 13 varieties tested) and field corn (7/8) to be susceptible to WSMV but the average percentage of diseased plants was low, being 25% and 31% respectively. Sill and Agusito (1955) found the majority of the varieties of field corn they tested to be susceptible (7/9) to WSMV but not sweet corn (3/7), flint corn (10/37) or popcorn (2/9). The percentage of plants diseased within the susceptible varieties was low, being 25.7%, 42.3%, 19.5% and 23.5% respectively. Seedlings of maize do not develop local lesions but will show faint systemic necrosis two to three weeks after inoculation.

Barley Stripe Mosaic Virus (BSMV) consists of three sizes of tubular rods (Fig. 3d, page 23), 111 nm x 30 nm, 128 nm x 30 nm and 148 nm x 30 nm (Harrison, Nixon and Woods 1965) which represent respectively 20%, 30% and 50% of the total RNA (Lane 1974). Chiko (1973) was unable to transmit the virus by aphids, leaf hoppers or grasshoppers in barley, and so far a vector of transmission in nature has not been found. However, the virus has been known to be transmitted by pollen in barley, and in addition, the seed of 68% of the varieties of barley in the world collection have shown transmittance of this virus (Timian 1975). The virus infects several monocotyledonous plants and a few dicotyledonous ones. BSMV has been found to induce non-Mendelian gene ratios in maize, the AR phenomenon (Sprague and McKinney 1966), and also to induce triploids and aneuploids in several varieties of barley (Sandfaer 1973). Most varieties of maize show little or no susceptibility to BSMV. No local lesions appear but systemic necrosis results in large white stripes, and in severe cases, stunting in maize.

MATERIALS AND METHODS

Seed Stocks

Seeds of Sib 67 Black Mexican Sweet Corn (Zea mays L. 2n = 20) consisting of two separate lines, one with B chromosomes (B line) and one without B chromosomes (A line) were obtained from Dr. R. J. Lambert of the Maize Cooperative in Illinois. Cytological examination of root tips were made for verification of the presence or absence of accessory chromosomes in the lines during the study. This was possible since B chromosomes in maize are somatically stable and their presence or absence can be determined by cytological analysis of root tips (Blackwood 1956). Cytology was done by pretreating the root tips in 0.02 M hydroxyquinoline, fixing them in three parts ethyl alcohol to one part acetic acid and staining with acetocarmine. Accessory chromosomes were revealed by counts of more than twenty chromosomes at metaphase of mitosis (Fig. 1b).

The seeds used in the first experiment were the Sib 67 bulk Black Mexican Sweet Corn. Five plants in the A line were analyzed and all were found to consist of the normal set of twenty chromosomes. Eight plants in the B line were analyzed and the mean number of B's per plant found was 1.9 (Table 1). For the second experiment the A and B lines were separately increased by outcrossing. Five plants in the A line were examined and all were found to consist of the normal chromosome complement. Fifteen plants in the B line were examined and the

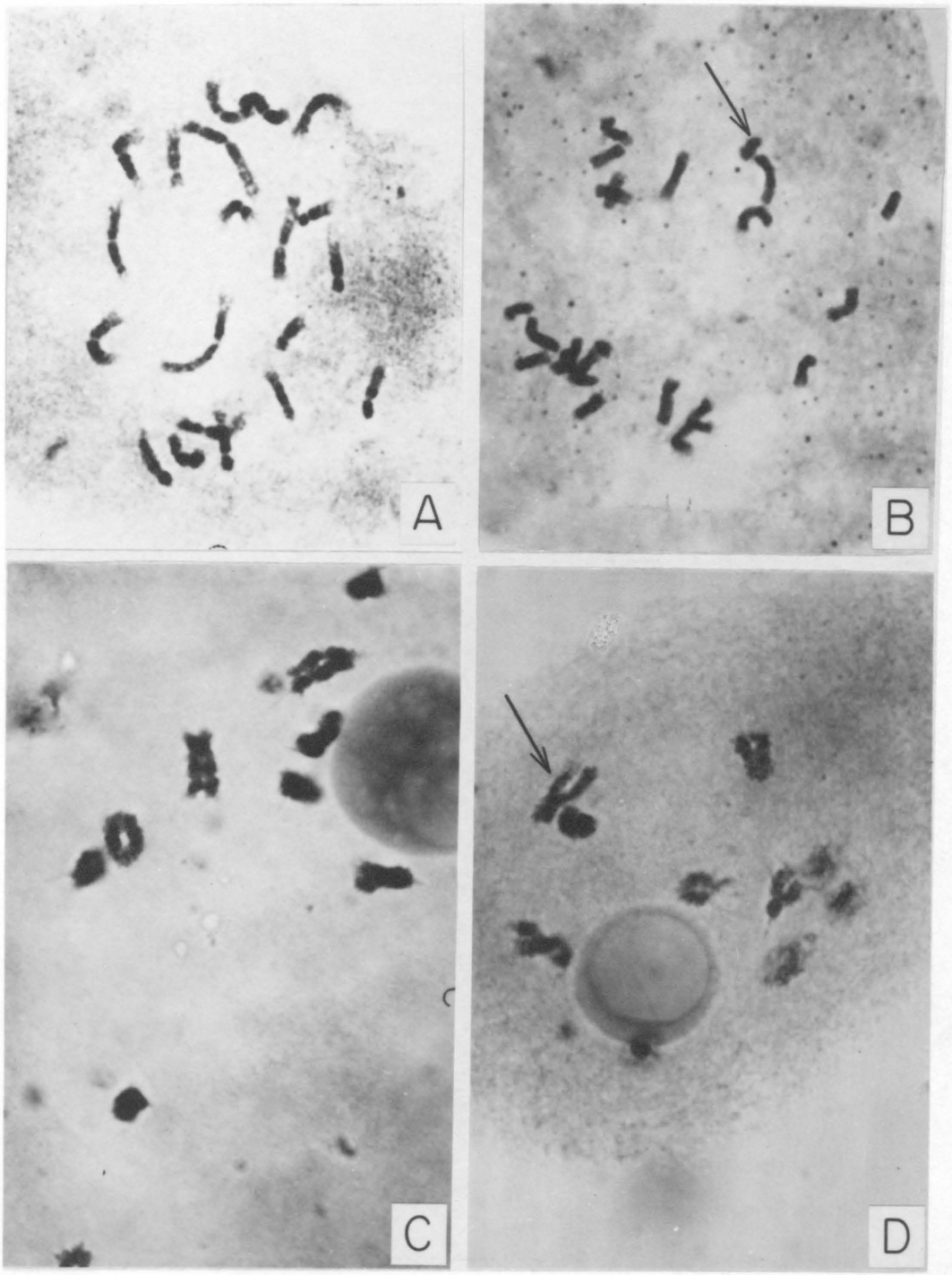

Figure 1. Mitotic metaphase of Black Mexican Sweet Corn:: (A) without B's and (B) with one B; meiotic metaphase of maize: (C) homozygous for the K10 chromosome and (D) heterozygous for the K10 and k10 chromosomes.

Table 1. The number of B chromosomes and centric B fragments (F) found in the A and B seed lines of Black Mexican Sweet Corn.

Seed Line	Sample No.	Number of plants with 0 to 3 B's and fragments						
		0	0 + F	1	1 + F	2	2 + F	≥ 3
A	1	5	0	0	0	0	0	0
	2	5	0	0	0	0	0	0
B	1	0	0	4	0	3	0	1[1]
	2	0	3	4	5	2	0	1[2]

1, 1 plant with 5 B's
2, 1 plant with 3 B's

average number of B's per plant was 1.1 (Table 1). In addition, several of the plants in the B line were found to contain centric B fragments, which varied in size. For the third experiment seeds were obtained from reciprocal crosses of healthy and infected plants from the B line and reciprocal crosses of healthy and infected plants from the A line. The viruses used for infection were BMV, MDMV or WSMV. BSMV was not included in this study because of its low infectivity of Black Mexican Sweet Corn.

Aleurone color in maize is controlled by the three genes Rr, Cc and Aa and all three dominant alleles must be present for pigment expression. Seed from a cross of C45-70 x C2 KYS, which was heterozygous for the abnormal tenth chromosome and the marker genes R and C, was obtained from Dr. E. Dempsey at the University of Indiana. In this seed the marker gene R for Purple aleurone was linked with the knobbed portion of the abnormal tenth (K10) chromosome and the gene r for colorless aleurone was linked with the normal chromosome ten (k10). The gene R has been shown by Rhoades (1942) to undergo approximately 1% recombination with the distal knobbed portion of the K10 chromosome (Fig. 2). Non-homologly of the distal one sixth of the k10 chromosome to the K10 chromosome has been shown by Kikudome (1957). He showed that while the SR_2 (striate) gene is located approximately 35 recombinational units distal to the R gene less than 1% recombination occurs in plants heterozygous for these two types of chromosomes.

Since the seed obtained from Dr. Demsey had the K10 R/k10 r Cc AA genotype it was crossed to a tester stock with the colorless

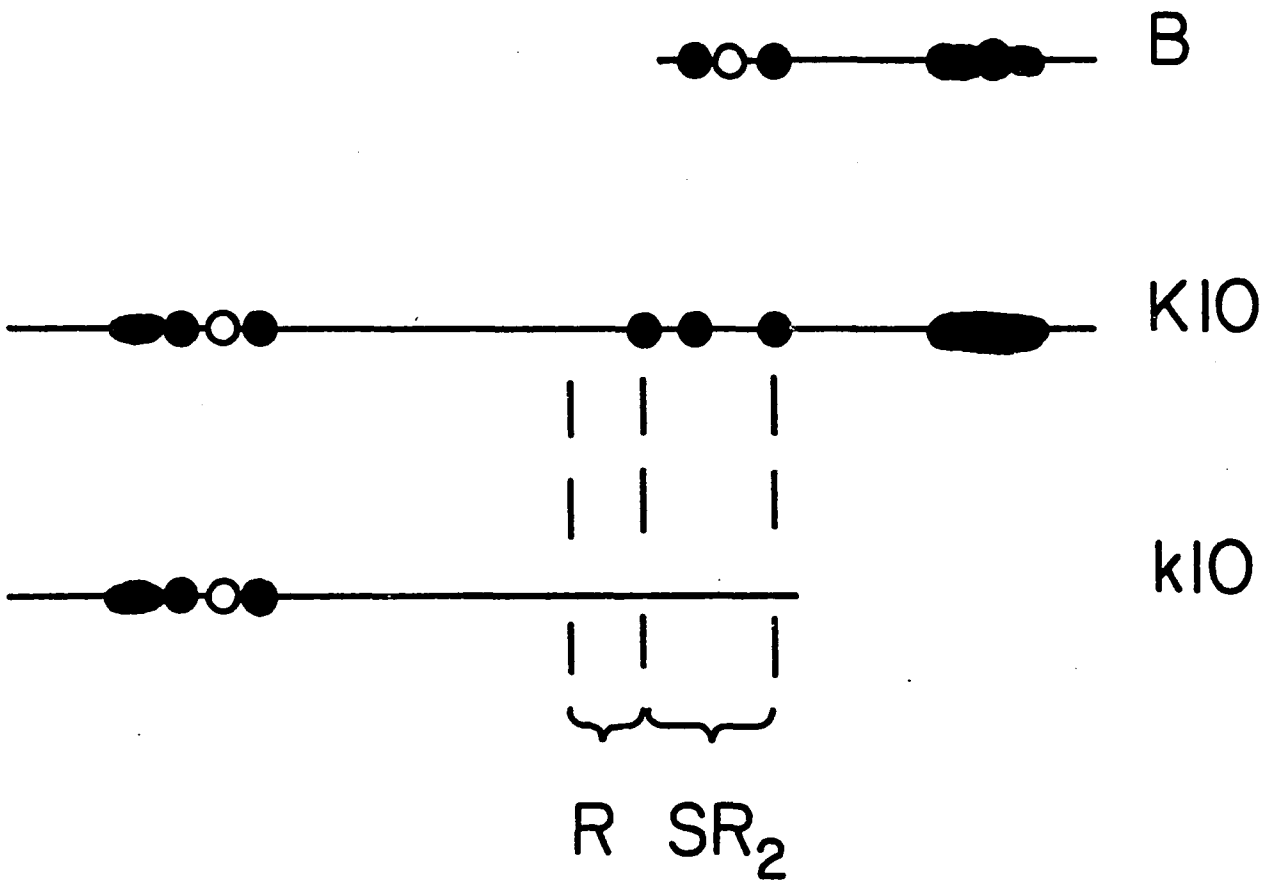

Figure 2. Cytogenetic maps of B, K10 and k10 chromosomes of maize. -- The maps were constructed from information published by Kikudome (1957), Longley and Kato (1965), and Ward (1973b).

aleurone genotype of k10 $\underline{r}^g$/k10 $\underline{r}^g$ $\underline{CC}$ $\underline{AA}$ in order to record the segregation of the $\underline{R}$ locus and thereby reveal the presence of those plants in the population with the K10 chromosome. The tester stock 73-1344 ($r^g r^g$ CC A_1A_1 A_2A_2), having colorless aleurone, was obtained from Dr. R. J. Lambert at the Maize Cooperative in Illinois. The $\underline{r}^g$ gene is similar to the $\underline{r}$ gene, the superscript refers to the green coloration of the anthers and will not be included in the subsequent discussion.

Test crosses were made and the progeny grown from purple and colorless seeds were examined cytologically to confirm the presence or absence of the K10 chromosome. Plants grown from purple seed should have the heterozygous genotype of K10 $\underline{R}$/k10 $\underline{r}$ and contain the abnormal tenth chromosome, whereas plants grown from colorless seed should have the homozygous genotype of k10 $\underline{r}$/k10 $\underline{r}$ and not contain the abnormal tenth chromosome. Cytological analysis was done by collecting tassels, fixing them in three parts ethyl alcohol to one part acetic acid and staining the sporocytes in acetocarmine. The K10 chromosome was evident at Metaphase I of meiosis because of the unequal length of a bivalent pair (Fig. 1d).

For the first experiment involving the abnormal tenth chromosome, seed was collected from test crosses of healthy plants. For the second experiment seeds were also obtained from reciprocal crosses of healthy plants of the test stock to MDMV or WSMV infected plants heterozygous for the K10 chromosome. It was not possible to recover seeds from plants treated by BMV or BSMV because plants inoculated with BMV

died at an early stage of growth and plants inoculated with BSMV did not become systemically infected.

Two lines of maize with the aberrant ratio phenomenon and two genetically similar lines without it were obtained from Dr. G. F. Sprague at the University of Iowa. The two aberrant ratio lines were designated AR_1 (74: 302-9 x 303-5) and AR_2 (74: 310-8 x 311-7) while the other two lines with normal ratios were designated NR_1 (74: 342-2 x 343-1) and NR_2 (74: 322-4 x 323-7). The AR lines obtained from Sprague had been derived from AA plants (red aleurone) that had been inoculated with BSMV and crossed as males to aa plants (colorless aleurone).

The seed increases for the experiment, involving the AR phenomenon, were made by backcrossing heterozygous plants (Aa = red aleurone) to recessive plants (aa = colorless aleurone). The seed increases were scored with respect to seed color ratios to verify the AR and NR lines. The normal ratio lines showed the expected gene ratio of 1 Aa: 1 aa and the aberrant ratio lines showed unexpected gene ratios (Table 2).

Virus Stocks

The different kinds of viruses used in this study were Brome Mosaic Virus (BMV), Maize Dwarf Mosaic Virus (MDMV), Wheat Streak Mosaic Virus (WSMV) and Barley Stripe Mosaic Virus (BSMV). These viruses can be readily transmitted to maize by means of mechanical inoculation with crude sap. These viruses are common to grasses such as maize and some of them may become integrated as proviruses in maize

Table 2. Genotypic ratios obtained from backcrosses of two normal ratio lines (NR_1 and NR_2) and aberrant ratio lines (AR_1 and AR_2) of maize.

Plant	NR_1 Aa : aa	NR_2 Aa : aa	AR_1 Aa : aa	AR_2 Aa : aa
1	51:27*	37:33	25:70*	110:188*
2	21:12	13:12	14:84*	112:185*
3	27:28	78:99	54:89*	127:117
4	121:128	9:16	97:164*	92:162*
5	114:143		25:50*	79:125*
6	151:138[1]		55:104*	148:241*
7			7:25*	91:310[1]*
8			20:91*	130:210[1]*
9			91:202[1]*	
10			224:321[1]*	
Totals	485:476	137:160	612:1,200	889:1,538

1. Seed from several ears.

*Significantly different from the expected 1:1 gene ratio at the .01 level.

chromosomes as indicated by induction of the AR phenomenon. While WSMV and BSMV induce AR, MDMV has been found not to (Sprague and McKinney 1971) and BMV has not been tested.

Maize Dwarf Mosaic Virus strain B isolated from maize in Iowa was obtained from Dr. R. E. Ford at the University of Illinois. The strain B will not infect Johnsongrass while strain A will. The virus culture was maintained in Golden Gross Bantam Sweet Corn, a variety donated by the Crookham Seed Company in Idaho.

Brome Mosaic Virus type strain, Wheat Streak Mosaic Virus strain 530 and Barley Stripe Mosaic Virus mild Argentina strain were all obtained from Dr. M. K. Brakke at the University of Nebraska. The BMV and WSMV cultures were maintained in Sieta Cerros Wheat which was obtained from the University of Arizona stock. BSMV was propogated in the Moore variety of Barley which was obtained from Dr. R. T. Ramage at The University of Arizona.

Verification of the virus stocks was made by examination of the crude sap from infected host plants under an electron microscope (Fig. 3) by Mr. Raymond E. Wheeler at The University of Arizona.

Experimental Procedures

All experiments were conducted in greenhouses at The University of Arizona. Plants were grown in thirty-two ounce styrofoam cups. The potting mixture was one part sterilized soil, one part peat and two parts vermiculite. Rapid Grow Fertilizer was applied periodically to plants to maintain optimum growth. Cygon and Galecron insecticides were used when necessary to control insects.

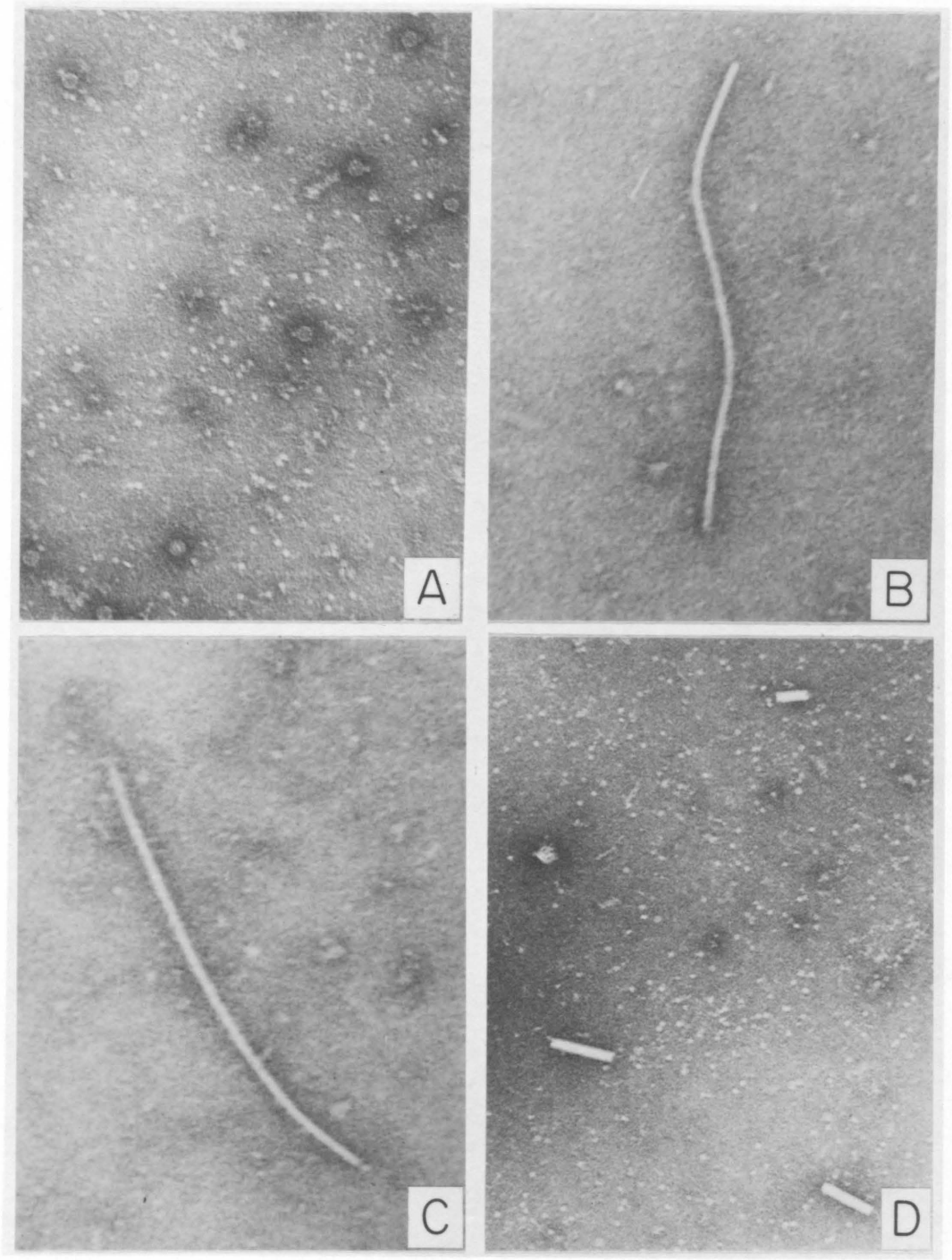

Figure 3. Electron micrographs of virus particles, X 97,000. -- (A) BMV, (B) MDMV, (C) WSMV, (D) BSMV.

The seeds of some of the varieties of maize used contained Rhizopus fungus spores and were therefore sterilized prior to planting. This was done by soaking the seed for six hours in water and then soaking them for ten minutes in a solution of 0.25% sodium hypochloride (5% bleach), followed by rinsing with water. In addition, a solution of Captan's fungicide was applied to the seed at the time of planting.

Plants were inoculated at the two to three leaf stage. Inoculum was made by grinding the systemically infected leaves of source plants in water (1:1 w/v) with a mortar and pestle. A small amount of carborundum was added to the crude sap to act as an abrasive. Cotton swabs were used to apply the inoculum to the leaves of the plants.

In the first experiment the viral susceptibility of two lines of Black Mexican Sweet Corn, one with B chromosomes (B line) and one without them (A line), of otherwise similar genetic background were compared. The experiment was set up in a split-plot randomized complete block design. Each block was randomly divided into four virus treatment plots which were in turn divided randomly into subplots containing the A and B lines of maize. The plots and subplots consisted of 50 and 25 plants respectively. The design included treatments with BMV, MDMV, WSMV and a control. Five replications of the complete blocks were made, consisting of a total of 1000 plants.

The second experiment with Black Mexican Sweet Corn was set up in a split-plot randomized complete block design similar to the first one. However, three replications of the complete blocks, instead of five, were made involving a total of 600 plants.

A third experiment with Black Mexican Sweet Corn was conducted to determine the effect of accessory chromosomes upon the viral resistance of the progeny of plants previously infected by viruses. The plants used in the experiment were derived from reciprocal crosses of healthy and infected plants in the B line and also from reciprocal crosses of healthy and infected plants from the A line. The viruses used for both the initial infection of the parent plants and the subsequent infection of the progeny of these plants were BMV, MDMV and WSMV. For this analysis a four factor experiment was designed. A group of 1,800 plants was partitioned according to (a) the three parental virus treatments (600 plants/virus); (b) the sex of the treated parent (300 plants/sex); (c) the three progeny virus treatments (100 plants/treatment); and (d) the two seed lines (50 plants/line).

In the first experiment involving the abnormal tenth chromosome, sibling groups of sweet corn plants with this chromosome (K10 k10) and without it (k10 k10) were compared with respect to viral susceptibility. The experiment was set up in a split-plot randomized complete block design. Groups of the K10 k10 and the k10 k10 plants were randomly assigned to subplots of 25 plants per subplot. The design included four treatments consisting of the three viruses BMV, MDMV and WSMV and a control. Four replications of the complete blocks were made which involved a total of 800 plants.

Another experiment was conducted to determine the effect of the K10 chromosome upon the viral resistance of the progeny of plants previously infected by MDMV and WSMV. Reciprocal crosses of healthy

and infected maize with the K10 chromosome and reciprocal crosses of healthy and infected maize without this chromosome were made. The progeny of these reciprocal crosses involving the virus treatments MDMV and WSMV were then inoculated by the same two viruses as well as BMV. A four factor experiment was designed consisting of a total of 800 plants. In it plants were partitioned according to: (a) the two parental virus treatments (400 plants/virus); (b) the sex of the treated parent (200 plants/sex); (c) the three progeny virus treatments (100 plants/treatment); and (d) the chromosome type (50 plants/type).

An experiment was conducted to compare the viral susceptibility of genetically similar maize lines with and without the aberrant ratio (AR) phenomenon. Four lines of maize, two exhibiting normal gene ratios and two exhibiting aberrant gene ratios were used. The experiment was set up in a split-plot randomized complete block design. Each block was randomly divided into five treatment plots with 200 plants per plot which were divided into subplots containing the four lines of maize with 50 plants per subplot. These were further divided into sub-subplots containing 25 plants per sub-subplot that were derived from seeds with red or colorless aleurones. The design included five treatments consisting of the four viruses BMV, MDMV, WSMV and BSMV and a control. Two replications of the complete blocks were made which consisted of a total of 1000 plants.

The susceptibility of the maize plants treated with the different viruses was judged by three criteria or parameters: the post inoculation time of systemic necrosis, the percentage of plants showing

necrosis and plant fresh weights. Characteristic leaf necroses induced by the viruses used in this study are shown in Figure 4. Scoring for systemic leaf necrosis was done at five day intervals after inoculation. The time of necrosis was estimated by converting the percentages of necrotic plants at the various time intervals to probit and using the least squares method of analysis to determine the time at which 50% of the necrotic plants ($P.D._{50}$) developed symptoms. Plant fresh weights of both treated and untreated plants were measured with a Mettler scale at the conclusion of the experiments. This was done by severing the plant at the ground level and weighing the above ground portion of the plant. Experiments with B chromosomes, K10 chromosomes and the AR phenomenon were concluded 50, 40 and 30 days respectively after inoculation.

All the experiments were set up in randomized complete block designs in an east to west direction in a greenhouse in order to reduce the weight variability that would occur as a result of differences in light intensity. An analysis of variance was made on the data obtained for the disease variables examined. The data on the percentage of diseased plants were transformed to arcsin in order to normalize it prior to analysis. A 5% level of significance was chosen prior to the experiments. F tests were used to judge the significance of main effects and interactions in the analysis of variance. Within treatment comparisons were made between plants with and without the B chromosomes, the K10 chromosomes and the AR effect. This was done by using least

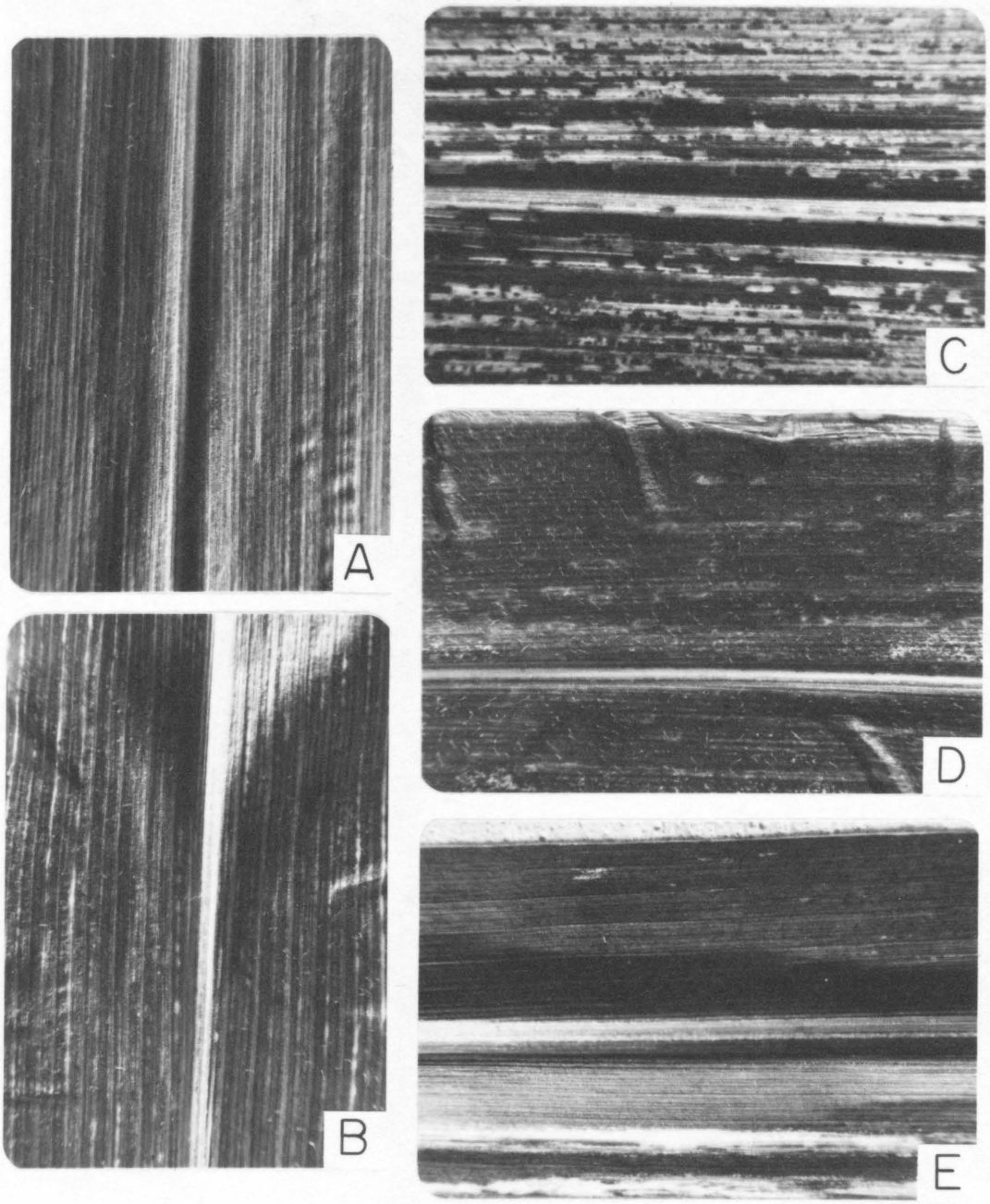

Figure 4. Photographs of sections of Black Mexican Sweet Corn leaves illustrating leaf necrosis. -- (A) control, (B) BMV, (C) MDMV, (D) WSMV, (E) BSMV.

significant differences which were determined at the .05 and .01 level by taking the product of the standard deviation of the mean differences and tabulated t values.

RESULTS

A series of experiments were conducted to determine if accessory (B) chromosomes, the abnormal tenth (K10) chromosome or the aberrant ratio (AR) phenomenon affect the resistance of maize to viral infection. The viruses used were Brome Mosaic Virus (BMV), Maize Dwarf Mosaic Virus (MDMV), Wheat Streak Mosaic Virus (WSMV) and Barley Stripe Mosaic Virus (BSMV). A control treatment was included, and the criteria of disease susceptibility were the time of systemic necrosis, the percentage of plants showing necrosis and plant fresh weights, as discussed in Materials and Methods.

B Chromosomes and Susceptibility

In the first experiment the viral susceptibility of two lines of Black Mexican Sweet Corn, one with B chromosomes (B line) and one without them (A line), of otherwise similar genetic background were compared. The majority of plants in the B line had one or two accessory chromosomes (Table 1). The experiment included treatments with BMV, MDMV, WSMV and a control.

The data are summarized in Table 3 and the results of the two factor analyses of variance of the experimental variables are presented in Table 4. No significant differences in susceptibility to BMV or MDMV were found between the A and B lines. However, a significant difference at the .01 level was found in susceptibility to WSMV between the two lines with respect to the time of systemic necrosis. The

Table 3. Viral susceptibility of Black Mexican Sweet Corn with (B line) and without (A line) accessory chromosomes: the figures are based on 125 plants.

Virus Treatment	Onset of Necrosis (days)		Percent with Necrosis		Mean Weight (grams)	
	A line	B line	A line	B line	A line	B line
BMV	6.3	6.8	99.3	98.6	52.4	49.8
MDMV	6.7	6.0	97.9	97.9	55.6	51.8
WSMV	27.0	31.4*	35.7	30.0	58.4	59.6
Control	-	-	2.9	2.9	62.6	57.6

*Significant at the .01 level.

Table 4. Two factor analyses of variance of the data from the experiment summarized in Table 3.

Source	Onset of Necrosis (days)		% with Necrosis (arcsin)		Mean Weight (grams)	
	df	MS	df	MS	df	MS
Block	4	2.83	4	11.47	4	183.46
V (virus)	2	1,728.34*	2	8,578.64*	3	183.69
Error V	8	5.01	8	42.47	12	47.13
C (chrom.)	1	14.7 *	1	24.30	1	65.03
VC	2	17.78*	2	3.70	3	18.02
Error C	12	.79	12	52.73	16	21.59

*Significant at the .01 level.

plants with B chromosomes showed leaf necrosis on an average of 31.4 days after treatment while those without B chromosomes showed leaf necrosis on an average of 27.0 days after treatment (Fig. 5). This represents a 16.0% difference between the two lines in the time of WSMV symptom expression.

In view of these results, a second experiment similar to the first one was performed with Black Mexican Sweet Corn with (B line) and without (A line) B chromosomes. Cytological analysis indicated that the plants in the B line contained one or more B chromosomes and/or centric B fragments (Table 1).

The data are presented in Table 5 and the results of the two factor analyses of variance of the parameters tested are shown in Table 6. The results confirmed those of the first experiment. No significant differences were found between plants with and without B chromosomes to BMV or MDMV infection but a significant difference was found at the .05 level with respect to the time of WSMV systemic necrosis. Plants in the B line developed leaf necrosis on the average of 26.9 days after inoculation while plants in the A line developed leaf necrosis on the average of 23.4 days after inoculation (Fig. 5). WSMV symptom expression appeared 15.0% later in plants with B chromosomes than in plants without them. Both experiments illustrate that symptom expression of WSMV is significantly delayed in the line with B chromosomes.

A third experiment was conducted with Black Mexican Sweet Corn to determine if B chromosomes could affect the process of cross

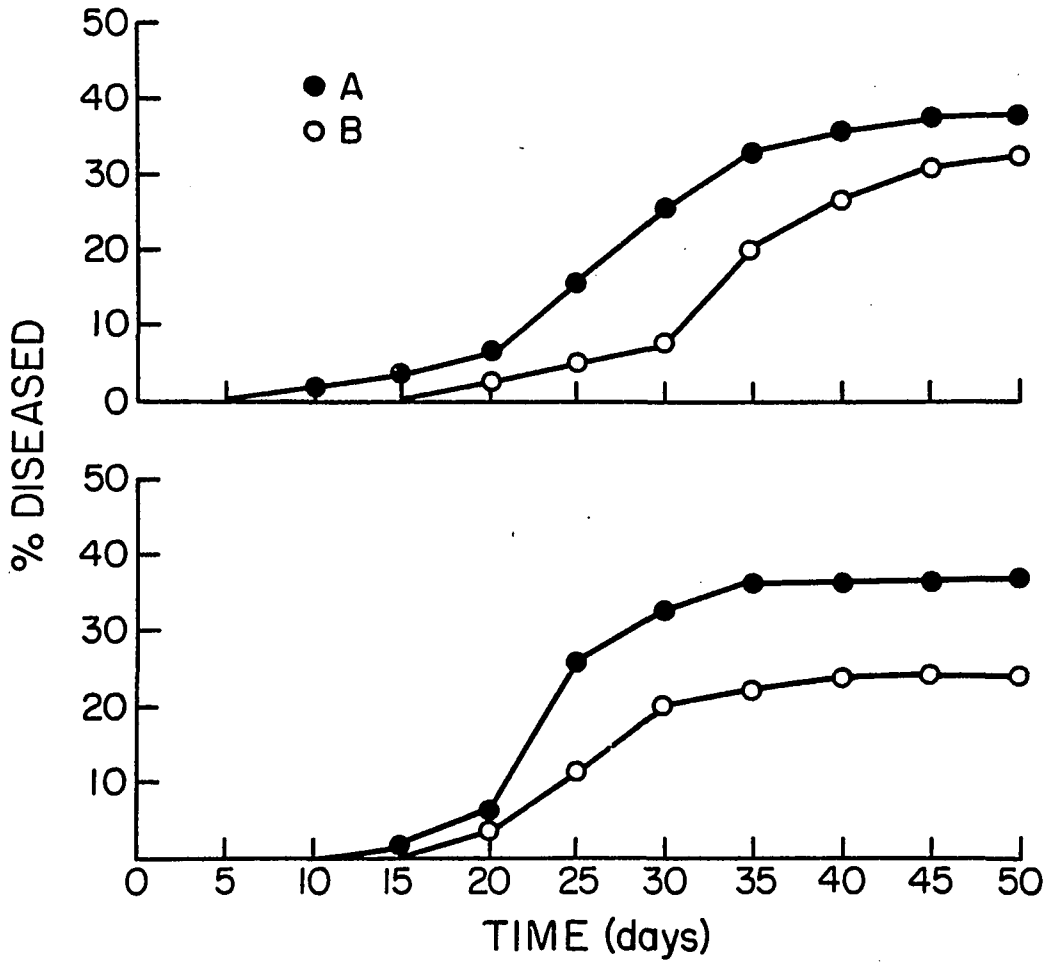

Figure 5. The percent of diseased Black Mexican Sweet Corn plants with (B) and without (A) accessory chromosomes obtained after inoculation with WSMV. -- First experiment above and second experiment below.

Table 5. Viral susceptibility of Black Mexican Sweet Corn with (B line) and without (A line) accessory chromosomes: the figures are based on 75 plants.

Virus Treatment	Onset of Necrosis (days)		Percent with Necrosis		Mean Weight (grams)	
	A line	B line	A line	B line	A line	B line
BMV	5.9	6.9	100	94	52.0	45.0
MDMV	7.8	7.8	86	90	44.7	45.0
WSMV	23.4	26.9*	36	24	54.7	54.0
Control	-	-	0	0	58.7	60.3

*Significant at the .05 level.

Table 6. Two factor analyses of variance of the data from the experiment summarized in Table 5.

Source	Onset of Necrosis (days)		% with Necrosis (arcsin)		Mean Weight (grams)	
	df	MS	df	MS	df	MS
Block	2	2.10	2	1.84	2	47.55
V (virus)	2	652.67**	2	4,404.50**	3	250.26*
Error V	4	1.01	4	68.08	6	20.26
C (chrom.)	1	10.28	1	118.11	1	12.04
VC	2	4.80	2	94.61	3	22.15
Error C	6	3.69	6	69.11	8	29.00

*Significant at the .05 level
**Significant at the .01 level

protection. The progeny of reciprocal crosses of healthy and viral infected plants were compared with respect to their resistance to subsequent viral infection. These comparisons were made with plants containing B chromosomes and also with plants without them in order to determine what effect these chromosomes might have on the integration of viruses into the maize chromosomes if integration indeed occurs. The viruses used for both the initial infection of the parent plants and the subsequent infection of the progeny of these plants were BMV, MDMV, and WSMV.

The data are summarized in Table 7 and the results of the four way analyses of variance of the experimental variables are shown in Table 8. No differences in the onset of necrosis or the percentage of plants showing necrosis were observed from the secondary infection of the plants. However, the analysis of variance of the plant weights showed a significant difference at the .01 level between the plants with respect to the sex of the treated parents. The plants derived from male infected parents weighed on the average 46.9 grams while those derived from female infected parents weighed on the average 51.7 grams. In addition, the analysis of variance revealed an interaction between the sex of the treated parents and the parental virus treatments at the .01 level of significance. The progeny of BMV infected parents showed no difference in weight between the sexes treated but the progeny of MDMV and WSMV infected parents did show differences in weight between the sexes treated. As was the case in the previous experiments, no differences were found between the A and B lines with

Table 7. Viral susceptibility of Black Mexican Sweet Corn with (B line) and without (A line) B chromosomes derived from reciprocal crosses of healthy and male (M) or female (F) infected parents. -- The figures are based on 50 plants.

Virus Treatment	Infected Parent	Sex of Infected Parent	Onset of Necrosis (days)		Percent with Necrosis		Mean Weight (grams)	
			A line	B line	A line	B line	A line	B line
BMV	BMV	M	5.8	5.1	100	86	61	58
		F	6.3	5.0	96	86	49	49
	MDMV	M	5.7	4.4	98	98	45	44
		F	4.8	5.6	100	100	50	50
	WSMV	M	5.1	6.4	100	100	32	34
		F	6.7	5.0	100	100	34	38
MDMV	BMV	M	4.6	4.8	82	96	57	45
		F	5.0	5.0	74	88	54	44
	MDMV	M	5.2	6.6	98	84	39	45
		F	4.6	4.4	86	98	56	58
	WSMV	M	4.8	5.5	76	68	54	48
		F	5.0	5.3	84	94	57	59
WSMV	BMV	M	12.5	22.5	4	4	42	45
		F	23.8	17.5	23	4	44	42
	MDMV	M	19.6	17.5	16	4	43	54
		F	-	-	0	0	56	60
	WSMV	M	17.5	17.5	4	4	50	49
		F	-	-	0	0	63	67

Table 8. Four factor analyses of variance of the data from the experiment summarized in Table 7.

Source	Onset of Necrosis (days)		% with Necrosis (arcsin)		Mean Weight (grams)	
	df	MS	df	MS	df	MS
C[1]	1	.01	1	26.69	1	8.02
S[2]	1	.07	1	.03	1	156.25*
P[3]	2	.23	2	130.36	2	21.53
V[4]	1	1.08	2	16,564.33*	2	173.69*
CS	1	.57	1	46.69	1	6.25
CP	2	.25	2	23.03	2	37.03
SP	2	.42	2	2.19	2	132.58*
CV	1	1.17	2	87.86	2	10.53
SV	1	.40	2	21.19	2	36.75
PV	2	.38	4	172.61	4	289.99*
Error	9	.49	16	75.05	16	11.68

*Significant at the .01 level.

1 = chromosome type
2 = sex of treated parent
3 = parental virus treatment
4 = progeny virus treatment

respect to secondary infection by BMV or MDMV. In the case of WSMV too few plants inoculated with this virus developed symptom expression to arrive at any conclusion.

K10 Chromosome and Susceptibility

In an experiment the viral susceptibility of sibling groups of maize plants with (K10 k10) and without (k10 k10) the abnormal tenth chromosome were compared. Plants heterozygous (K10 R/k10 r) for the abnormal tenth chromosome carried the linked R allele for purple seed color, whereas plants homozygous (k10 r/k10r) for the standard tenth chromosome carried the linked r allele for colorless seed. The experiment included treatments with BMV, MDMV, WSMV and a control.

A summary of the data is presented in Table 9 and the results of the two factor analyses of variance of the experimental variables are shown in Table 10. No significant differences were found between the K10 k10 and the k10 k10 plant groups with respect to BMV or MDMV infection. However, the WSMV treatment showed a significant difference at the .01 level in the percentage of diseased plants between these two plant groups. Fifty percent of the plants in the K10 k10 plant group showed systemic necrosis while 25% of the plants in the k10 k10 plant group showed necrosis. The group of plants with the abnormal tenth chromosome displayed twice the susceptibility to WSMV infection as the group without the abnormal tenth chromosome.

A cross protection experiment was conducted to determine if the K10 chromosome could affect the integration of viruses into the maize chromosome. The progeny of reciprocal crosses of healthy and viral

Table 9. Viral susceptibility of maize with (K10 k10) and without (K10 k10) the abnormal tenth chromosome. -- The figures are based on 100 plants.

Virus Treatment	Onset of Necrosis (days)		Percent with Necrosis		Mean Weight (grams)	
	k10 k10	K10 k10	k10 k10	K10 k10	k10 k10	K10 k10
BMV	3.0	3.2	89	92	-	-
MDMV	2.8	2.6	99	100	49.5	49.5
WSMV	20.6	22.6	25	50*	49.8	48.8
Control	-	-	0	0	49.8	49.0

*Significant at the .01 level.

Table 10. Two factor analyses of variance of the data from the experiment summarized in Table 9.

Source	Onset of Necrosis (days)		% with Necrosis (arcsin)		Mean Weight (grams)	
	df	MS	df	MS	df	MS
Block	3	.04	3	79.49	3	33.48
V (virus)	2	929.05**	2	5,503.04**	2	2.38
Error V	6	.78	6	39.32	6	16.82
C (chrom.)	1	2.87	1	234.37*	1	7.04
VC	2	2.54	2	117.38	2	3.29
Error C	9	6.87	9	28.26	9	9.21
Corrected Total	23	84.03	23	530.61	23	13.16

*Significant at the .05 level
**Significant at the .01 level

infected plants were compared with respect to their resistance to subsequent viral infection. These comparisons were made with plants containing the K1O chromosome and with plants without it in order to determine what effect these chromosomes might have on the process of cross protection. The viruses used for the initial infection of the parent plants were MDMV and WSMV, and for the subsequent infection of the progeny of these plants the same viruses were used in addition to BMV.

The results are summarized in Table 11 and the analyses of variance of the variables scored are shown in Table 12. The analysis of variance of the percentage of plants with systemic necrosis showed a significant difference at the .01 level between the plants derived from MDMV and WSMV infected parents. No significant variation was found between plants derived from male or female infected parents. No significant differences were found between the K1O k1O and the k1O k1O plant groups with respect to the second infection with BMV and MDMV. There was a significant difference at the .01 level between these two plant groups with respect to the percentage of plants showing WSMV systemic necrosis. When reinfected with WSMV, twenty-two percent of the K1O k1O plants and 10% of the k1O k1O plants derived from MDMV infected parents showed systemic necrosis while 48% of the K1O k1O plants and 28% of the k1O k1O plants derived from WSMV infected parents showed necrosis (Fig. 6). These results confirm those of the previous experiment in which twice as many plants with the abnormal tenth chromosome showed necrosis as those without the abnormal tenth chromosome.

Table 11. Viral susceptibility of maize with (K10 k10) and without (k10 k10) the abnormal tenth chromosome derived from reciprocal crosses of healthy and male (M) or female (F) infected parents. -- The figures are based on 25 plants.

Virus Treatment	Infected Parent	Sex of Infected Parent	Onset of Necrosis (days)		Percent with Necrosis		Mean Weight (grams)	
			k10 k10	K10 k10	k10 k10	K10 k10	k10 k10	K10 k10
BMV	MDMV	M	7.6	9.8	64	80	20	20
		F	6.2	7.6	68	60	20	18
	WSMV	M	4.3	4.9	84	76	19	20
		F	5.9	4.7	76	80	16	18
MDMV	MDMV	M	6.0	7.3	88	80	22	22
		F	7.2	7.5	84	76	21	24
	WSMV	M	4.5	5.4	96	100	26	23
		F	5.1	5.0	100	96	22	21
WSMV	MDMV	M	17.3	26.6	4	28	38	25
		F	22.2	20.1	16	16	29	27
	WSMV	M	24.4	20.1	24	36	28	36
		F	18.3	23.3	32	60	29	28
Control	MDMV	M	-	-	0	0	30	27
		F	-	-	0	0	23	23
	WSMV	M	-	-	0	0	26	26
		F	-	-	0	0	26	28

*Significant at the .01 level when combined.

Table 12. Four factor analyses of variance of the data from the experiment summarized in Table 11.

Source	Onset of Necrosis (days)		% with Necrosis (arcsin)		Mean Weight (grams)	
	df	MS	df	MS	df	MS
C[1]	1	7.37	1	51.04	1	2.67
S[2]	1	1.08	1	1.04	1	28.17
P[3]	1	15.84	1	1,107.04**	1	.00
V[4]	2	628.61*	2	4,017.79**	2	256.29**
CS	1	1.87	1	70.04	1	1.50
CP	1	5.51	1	3.38	1	16.67
SP	1	.26	1	30.38	1	4.17
CV	2	1.14	2	110.29	2	2.79
SV	2	1.19	2	43.79	2	2.79
PV	2	4.20	2	74.04	2	2.38
Error	9	6.56	9	32.91	9	12.19

*Significant at the .05 level
**Significant at the .01 level

1 = chromosome type
2 = sex of treated parent
3 = parental virus treatment
4 = progeny virus treatment

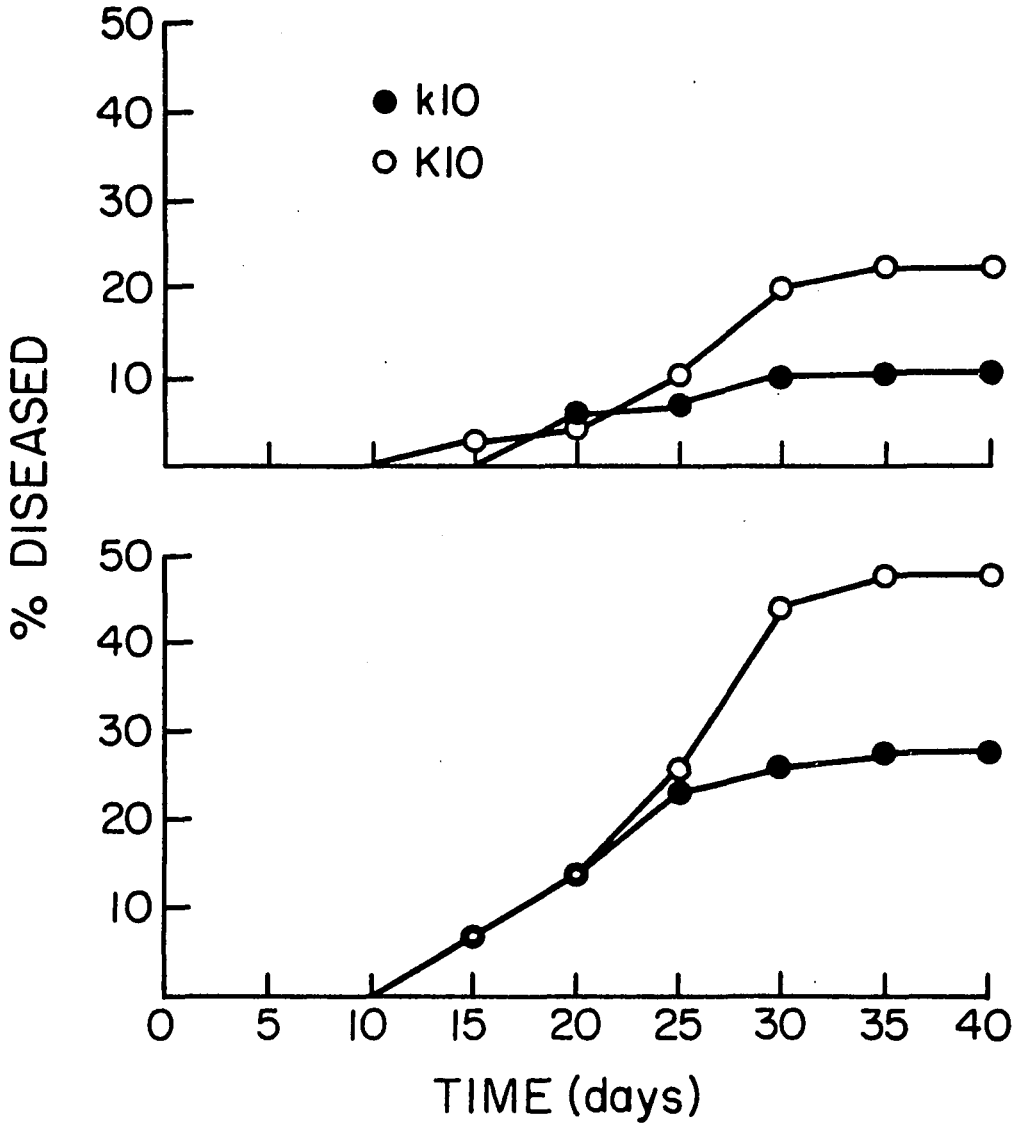

Figure 6. The percent of diseased maize plants with (K10) and without (k10) the abnormal tenth chromosome obtained after inoculation with WSMV. -- The plants were derived from MDMV (above) and WSMV (below) infected parents.

AR Phenomenon and Susceptibility

An experiment was conducted to compare the viral susceptibility of genetically similar maize lines with and without the aberrant ratio (AR) phenomenon. Four lines of maize, two designated NR_1 and NR_2 that exhibited normal gene ratios and two designated AR_1 and AR_2 that exhibited aberrant gene ratios for the alleles A for red aleurone color and a for colorless aleurone were used. In addition to the comparison between the maize lines, one was made within these lines between plants of the Aa and aa genotypes. The experiment included five treatments consisting of the four viruses BMV, MDMV, WSMV and BSMV and a control.

The data are summarized in Table 13 and the results of the three factor analyses of variances of the variables scored are presented in Table 14. The results of the analyses showed no differences between the seed lines with respect to the BMV or BSMV treatments. But with the MDMV treatment, a significant difference at the .05 level was found between the NR and AR lines. Seventy-two percent of the plants in the AR lines showed systemic necrosis while 53% of the plants in the NR lines showed necrosis when treated with MDMV. Another significant difference at the .01 level was found with the WSMV treatment between the AR and the NR lines. In this case, 2% of the plants in the AR lines showed systemic necrosis after being inoculated with WSMV while 24.5% of the plants in the NR lines showed necrosis. No differences in disease susceptibility were found between the Aa and aa genotypic plants within the maize lines.

Table 13. Viral susceptibility of maize with (AR) and without (NR) the aberrant ratio phenomenon. -- The figures are based on 50 plants.

Virus Treatment	Genotype	Onset of Necrosis (days)				Percent with Necrosis				Mean Weight (grams)			
		NR_1	NR_2	AR_1	AR_2	NR_1	NR_2	AR_1	AR_2	NR_1	NR_2	AR_1	AR_2
BMV	aa	7.0	6.5	8.6	10.6	72	76	48	70	-	-	-	-
	Aa	7.9	7.3	5.7	9.8	82	80	93	72	-	-	-	-
MDMV	aa	8.8	5.5	8.2	5.8	48	34	76	74	14	18	14	16
	Aa	7.7	7.5	6.4	6.1	62	68	70	68	13	15	10	11
WSMV	aa	11.2	19.3	12.3	14.7	24	24	4	2	33	32	31	31
	Aa	12.3	18.3	12.3	14.7	20	30	0	2	35	31	33	27
BSMV	aa	22.3	15.0	12.3	12.3	4	6	4	4	13	18	17	16
	Aa	17.2	17.3	18.8	18.3	6	4	6	8	11	16	15	14
Control	aa	-	-	-	-	0	0	0	0	47	49	44	56
	Aa	-	-	-	-	0	0	0	0	45	48	42	55

*Significant at the .05 level when combined
**Significant at the .01 level when combined

Table 14. Three factor analyses of variance of the data from the experiment summarized in Table 13.

Source	Onset of Necrosis (days)		% with Necrosis (arcsin)		Mean Weight (grams)	
	df	MS	df	MS	df	MS
Block	1	.63	1	341.33	1	68.06
V (virus)	2	261.50**	2	8,283.52*	3	4,215.19*
Error 1	2	1.18	2	128.65	3	212.10
G (genotype)	1	2.04	1	225.33	1	42.25
GV	2	.23	2	160.27	3	7.71
Error 2	3	4.40	3	93.29	4	24.66
C (seed class)	3	9.37	3	90.03	3	26.60*
CV	6	22.79**	6	429.30*	9	44.51**
CG	3	3.26	3	50.61	3	2.54
CVG	6	2.58	6	212.88	9	2.78
Error 3	18	4.65	18	155.97	24	6.79

*Significant at the .05 level
**Significant at the .01 level

DISCUSSION

One intent of this study was to determine if accessory or B chromosomes of maize affect the plants resistance to viral infection. It has been found that B chromosomes alter the cellular synthesis in maize of both nucleic acids and proteins (Ayonoadu and Rees 1971). Therefore it was anticipated that these chromosomes could have an effect upon the cellular synthesis of viruses as well and possibly upon the integration of the virus into the maize genome.

Genetically similar lines of Black Mexican Sweet Corn, one with B chromosomes (B line) and one without them (A line), were compared with respect to susceptibility to BMV, MDMV, and WSMV. In two separate experiments these two lines of sweet corn were found to respond similarily to BMV and MDMV infection but differently to WSMV infection. In the first experiment with WSMV, as illustrated in Figure 5, the plants in the B line on the average showed leaf necrosis 31.4 days after treatment while plants in the A line on the average showed leaf necrosis 27.0 days after treatment. This represents a significant difference of 16.0% between the two lines with respect to the time of WSMV symptom expression. In the second experiment with WSMV, as illustrated in Figure 5, on the average plants in the B line showed leaf necrosis 26.9 days after inoculation while plants in the A line showed signs of necrosis 23.4 days after inoculation. This represents a 15.0% difference, a significant value, in the time of WSMV symptom expression.

Although the illustrations in Figure 5 are suggestive of a difference in the percentage of plants showing necrosis at fifty days, this was not found to be statistically significant.

The experimental results reveal that accessory chromosomes affect the resistance of maize to WSMV but not to BMV or MDMV. Despite differences between these experiments in the number of days until the onset of WSMV necrosis, the relative difference between the lines remained constant. The line of plants with B chromosomes showed a 16.0% and 15.0% delay in the onset of WSMV necrosis relative to the line of plants without B chromosomes in two separate experiments. These results could be obtained if B chromosomes slow the cellular synthesis of either the RNA or the protein components of the WSMV particles.

An experiment was conducted to determine if the abnormal tenth chromosome (K10) of maize has an effect similar to B chromosomes in altering the resistance of maize to viral infections. It has been hypothesized that the K10 chromosome may be the product of an B to A chromosomal translocation (Ting 1958). This idea is based on the similar appearance of portions of these chromosomes at pachytene of meiosis and the neocentric activity of the K10 chromosome which is reminiscent of a B centromere. If this hypothesis is correct then these chromosomes may similarly alter the plants' resistance to viruses.

An experiment was conducted in which sibling groups of maize plants with (K10 k10) and without (k10 k10) the abnormal tenth chromosome were compared with respect to their resistance to BMV, MDMV and WSMV. The results of the experiment showed no difference between these

two plant groups in susceptibility to BMV and MDMV. However, these plant groups did respond differently to infection with WSMV. Fifty percent of the plants in the K1O k1O group showed systemic necrosis while 25% of the plants in the k1O k1O group showed necrosis.

If the K1O chromosome of maize is related to the B chromosome then it might be expected to similarly alter the resistance of maize to viral infections. This was partially found to be the case. The K1O and B chromosomes were similarly found not to alter the resistance of maize to BMV or MDMV but to alter the resistance of maize to WSMV. However, both chromosome types differently affected the plants' resistance to WSMV infection. The B chromosome was found to delay the onset of WSMV systemic necrosis while the K1O chromosome was found to increase the plants susceptibility. Although portions of these chromosomes may be related, the chromosomes as a whole are unique. Genes present in the K1O chromosome but absent from the B chromosomes could account for the different effects they have upon WSMV infection.

In the aberrant ratio (AR) phenomenon observed in maize, unusual progeny ratios of specific marker genes are found. This phenomenon is induced by viral infection and may result from viral integration near the aberrant marker gene (Sprague and McKinney 1966). When plants become infected by one virus they may become resistant to a secondary infection by similar viruses, which is known as cross protection. If lines of maize exhibiting the AR phenomenon do contain cross protective viruses integrated near marker genes, this might be made evident by the alteration of their resistance to subsequent infection by similar viruses.

An experiment was conducted in which four genetically similar lines of maize, two exhibiting the AR phenomenon (AR lines) and two exhibiting normal gene ratios (NR lines) were compared with respect to susceptibility to BMV, MDMV, WSMV and BSMV. The AR stocks used were originally induced by BSMV inoculation of AA plants which were crossed as males to aa plants. A comparison within the maize lines was made between plants with (Aa) and without (aa) the A gene which had been exposed to BSMV.

The results showed no difference between the plant lines with respect to BMV or BSMV infection. However, only 5% of the plants became infected by BSMV which may not be sufficient to reveal any differences between the plant lines. However, when treated with MDMV, 72% of the plants in the AR lines showed systemic necrosis in contrast to 53% of the plants in the NR lines. After being inoculated with WSMV, 2% of the plants in the AR lines showed systemic necrosis while 24.5% of the plants in the NR lines showed necrosis. The genotypic differences of Aa and aa were not found to alter the plants viral susceptibility.

BSMV and WSMV have been shown to induce the AR phenomenon while MDMV has been shown not to (Sprague and McKinney 1971) and BMV has not been tested. If the provirus hypothesis is correct and BSMV was present in the AR lines and is capable of cross protection then these lines would be expected to show resistance to infection by BSMV and possibly WSMV. While the results with the BSMV treatment were not conclusive, the results with the WSMV treatment were consistent with this hypothesis. Although BSMV and WSMV are not structurally similar they

both induce AR and may therefore become integrated at similar sites in the maize chromosome. The presence of the BSMV genome in the AR plant lines may account for their resistance to subsequent WSMV infection.

It has been hypothesized that the AR phenomenon may be induced by the integration of BSMV adjacent to the A allele. In the event that this is true then plants in the AR lines with the A allele might be expected to show more resistance to WSMV than plants without this allele. However, the experimental results did not support this hypothesis since within the AR lines plants with the Aa genotype (i.e., carrying the A allele) and plants with the aa genotype (i.e., not carrying the A allele) were equally susceptible to WSMV infection.

Studies involving the inheritance of the AR condition have shown the aa genotypic plants in the AR line are not equivalent to the aa genotypic plants in the NR line. A cross involving a male Aa genotypic plant from the NR line to a female aa genotypic plant from the NR line will result in a normal progeny ratio. However, crosses involving the same male parent to a female aa genotypic plant from the AR line will often result in an aberrant progeny ratio (Sprague and McKinney 1971). The results of the cross protection study, in accordance with the inheritance studies, suggest that proviruses may exist in both the Aa and the aa genotypic classes of the AR lines.

A study was made to determine if the B or the K10 chromosomes could affect the integration of viruses into the maize chromosome. If the viruses were capable of cross protection then these chromosomes would in effect indirectly alter the resistance of maize to subsequent

viral infections. The B and K10 chromosomes have similar properties which could affect viral integration. Both the B chromosome (Hanson 1969) and K10 chromosome (Rhoades and Dempsey 1957) increase recombination in maize. Since viral integration probably comes about through a recombinational event (Campbell 1962), these chromosomes could enhance this process. In addition, each B chromosome increases the amount of cellular DNA by 5% (Ayonoadu and Rees 1968), and the K10 chromosomes similarly increases the amount of DNA per cell. These chromosomes could offer additional sites for the random or specific integration of viruses.

In two cross protection experiments comparisons were made between the progeny of the reciprocal crosses of healthy and viral infected plants with respect to subsequent viral infection. In the first experiment, the comparison was made with plants containing B chromosomes and with plants without them in order to determine what effect these chromosomes might have on cross protection. The viruses used for both the initial infection of the parent plants and subsequent infection of the progeny of these plants were BMV, MDMV and WSMV. No differences were found in the resistance of the progeny of the plants which had been previously infected by the viruses. In the second experiment a similar comparison was made, but with plants containing the K10 chromosome and with plants without it, in order to determine what effect this chromosome might have on cross protection. MDMV and WSMV were used for the initial infection of the parent plants and BMV, MDMV, and WSMV were used for the subsequent infection of the

progeny. No differences were found in the resistance of the progeny of plants which had been derived from previously infected parents that would be suggestive of cross protection.

The results of the experiments with B and K10 chromosomes did not reveal any evidence of cross protection. It was therefore not possible to ascertain what effect the B or K10 chromosome might have on viral integration. The induction of the AR phenomenon, which may represent the integration of viruses into the maize genome, occurs at a frequency of one in two hundred systemically infected plants (Sprague and McKinney 1966). In view of this, it is possible that the frequency of viral incorporation into the chromosomes of maize could be too low in the B and K10 chromosome experiments to have detected it.

The cross protection study involving the abnormal tenth chromosome confirmed the results of the earlier study by indicating that while the K10 chromosome does not affect the plants resistance to BMV or MDMV it does alter the plants resistance to WSMV. When infected with WSMV, 22% of the K10 k10 plants and 10% of the k10 k10 plants derived from MDMV infected parents showed systemic necrosis (Fig. 6). Forty-eight percent of the K10 k10 plants and 28% of the k10 k10 plants derived from WSMV infected parents showed necrosis when infected with WSMV (Fig. 6). Thus, despite differences in the susceptibility of the plants derived from MDMV or WSMV infected parents, twice as many plants with the K10 chromosome became necrotic than without this chromosome.

The relative susceptibility of plants with the K10 chromosome to those without it is suggestive of a 2:1 genetic ratio. In actuality

this 2:1 ratio may represent a 3:1 ratio of WSMV susceptibility governed by a two gene model of duplicate dominant epistasis. According to this hypothesis, the results obtained suggest that one gene (Ws_1) could be linked closely to the R gene on the K10 chromosome and the other gene (Ws_2) would assort independently of the first gene. Plants with the $\underline{Ws}_1$ and/or $\underline{Ws}_2$ genes would be susceptible to viral infection. The plants used in the experiments would represent the progeny of a $\underline{Ws}_1ws_1$ $\underline{Ws}_2ws_2$ x ws_1ws_1 ws_2ws_2 cross. Thus, the purple (K10 R/k10 r) aleuroned seeds would represent the $\underline{Ws}_1ws_1$ $\underline{Ws}_2ws_2$ and $\underline{Ws}_1ws_1$ ws_2ws_2 genotypic classes and all would be susceptible to WSMV infection. The seeds with colorless (k10 r/k10 r) aleurone would represent the genotypes ws_1ws_1 $\underline{Ws}_2ws_2$ and ws_1ws_1 ws_2ws_2 and only the former class would be susceptible to WSMV infection.

This model may be applied to the results obtained with accessory chromosomes. This model relies on the $\underline{Ws}_1$ gene being tightly linked to the R gene. Thus, the $\underline{Ws}_1$ gene would be present on the K10 chromosome and absent from the B chromosome (Fig. 1). If the $\underline{Ws}_1$ and/or $\underline{Ws}_2$ genes were present in both the A and B lines they would be expected to have an equal percentage of plants showing necrosis which was found to be the case. If the B and K10 chromosomes were related then the K10 chromosome would be expected to delay the onset of WSMV necrosis as the B chromosome does. Although this was not found to be the case in these experiments subsequent studies may reveal this to be true.

It has been proposed that heterochromatin might function to protect the more vital euchromatin of cells from mutagens and viruses

(Hsu 1975). Since plants with the B and K10 chromosomes contain more heterochromatin than those without them it was possible for this study to provide information on the validity of this hypothesis. The results obtained showed that heterochromatin in the form of B and K10 chromosomes does not alter the susceptibility of maize to BMV or MDMV infection but it may delay the time of WSMV disease onset. Thus while heterochromatin does not appear to protect euchromatin from exposure to viruses in general it may do so with certain viruses by delaying thier ability to penetrate the nuclear membranes of cells.

It has been proposed that organisms containing B chromosomes may have a selective advantage over those without them (Darlington and Upcott 1941). The results of this study indicate that plants with B chromosomes do not have a selective advantage over those without them to BMV, MDMV and probably WSMV infection. Although B chromosomes did delay the onset of WSMV necrosis this delay would probably not be sufficient to alter the plants fitness.

SUMMARY

A study was made to determine if accessory (B) chromosomes, the abnormal tenth chromosome (K10) and the aberrant ratio (AR) phenomenon affect the resistance of maize to viral infection. Stocks of maize with and without these elements of otherwise similar genetic background were compared.

Accessory chromosomes were not found to alter the resistance of maize to BMV or MDMV infection. However, these chromosomes did affect the resistance of the plants to WSMV infection. The group of plants with B chromosomes became diseased 15% and 16% later than the group without them in two separate experiments (Fig. 5).

The abnormal tenth chromosome of maize was not found to affect the plants resistance to BMV or MDMV infection but it did affect the resistance of the plants to WSMV infection. In two experiments twice as many plants showed WSMV symptoms in the group that contained the K10 chromosome than in the group that did not. This ratio was found to remain constant despite differences in the percentage of diseased plants obtained (Fig. 6).

The aberrant ratio phenomenon observed in maize is induced by viruses and may be the result of viral integration. The AR stocks used in this study were induced by BSMV inoculation. The results obtained indicate that the AR phenomenon had no effect on BMV or BSMV infection in maize. The infection rate with BSMV was only 5% which may not be

sufficient to reveal any differences between the lines of maize tested. Plants with the AR phenomenon showed more susceptibility to MDMV infection and much less susceptibility to WSMV infection.

Cross protection experiments were conducted to determine if viruses become integrated into the genome of maize and if so whether the B or K10 chromosomes would affect this integration. The presence of proviruses would be detected if they alter the susceptibility of the plant to subsequent infections. The progeny of reciprocal crosses of healthy and infected plants with and without B or K10 chromosomes were compared with respect to viral susceptibility. No differences were found that would be indicative of proviruses. Therefore it was not possible to ascertain what effect the B or the K10 chromosomes might have on viral integration.

LITERATURE CITED

Ayonoadu, U. and H. Rees. 1968. The influence of B-chromosomes on chiasma frequencies in Black Mexican Sweet Corn. Genetica 39:75-81.

Ayonoadu, U. and H. Rees. 1971. The effects of B chromosomes on the nuclear phenotype in root meristems of maize. Heredity 27: 365-383.

Bancroft, J. B. 1970. Brome Mosaic Virus. No. 3 C.M.I./A.A.B. Description of plant viruses.

Blackwood, M. 1956. The inheritance of B chromosomes in Zea mays. Heredity 10:353-366.

Brakke, M. K. 1971. Wheat Streak Mosaic Virus. No. 48 C.M.I./A.A.B. Description of plant viruses.

Campbell, A. 1962. Episomes. Adv. Genet. 11:101-145.

Carlson, W. R. 1969. A test of homology between the B chromosome of maize and the abnormal chromosome 10, involving the control of non disjunction in B's. Molec. Gen. Genet. 104:59-65.

Chang, C. C. and G. Y. Kikudome. 1974. The interaction of knobs and B chromosomes of maize in determining the level of recombination. Genetics 77:45-54.

Chiko, A. W. 1973. Failure to transmit Barley Stripe Mosaic Virus by aphids, leafhoppers and grasshoppers. Plant Dis. Rep. 57:639-641.

Chilton, M. D. and B. J. McCarthy. 1973. DNA from maize with and without B chromosomes: a comparative study. Genetics 74: 605-614.

Darlington, C. D. and M. B. Upcott. 1941. The activity of inert chromosomes in Zea mays. J. Genet. 41:275-296.

Findley, W. R., E. J. Dollinger, R. Louie and J. K. Knoke. 1973. Locating genes for Maize Dwarf Mosaic resistance by means of chromosomal translocations in corn (Zea mays L.). Crop Sci. 13:608-611.

Ford, R. E. 1974. Physical and serological properties of Maize Dwarf Mosaic and Sugarcane Mosaic Viruses. Phytopathology 64: 312-317.

Genter, C. F. 1973. Effects of Maize Dwarf Virus on mechanically inoculated maize. Crop. Sci. 13:531-535.

Hanson, G. 1969. B chromosome stimulated crossing over in maize. Genetics 63:601-609.

Harrison, B. D., H. L. Nixon and R. O. Woods. 1965. Lengths and structures of particles of Barley Stripe Mosaic Virus. Virology 26:284-289.

Hill, J. H., C. A. Martinson and W. A. Russell. 1974. Seed transmission of Maize Dwarf and Wheat Streak Mosaic Viruses in maize and response of inbred lines. Crop Sci. 14:232-235.

Hsu, T. C. 1975. Symposium No. 14: Chromosome structure a possible function of constitutive heterochromatin: the bodyguard hypothesis. Genetics 79:137-150.

Huebner, R. J. and G. J. Todaro. 1969. Oncogenes or RNA tumor viruses as determinants of cancer. Proc. Natl. Acad. Sci. U.S. 64: 1087-1094.

Jones, R. K. and S. A. Tolin. 1972. Concentration of Maize Dwarf Mosaic Virus in susceptible and resistant corn hybrids. Phytopathology 62:640-644.

Kikudome, G. Y. 1957. Studies on the phenomenon of preferential segregation in maize. Genetics 44:815-831.

Kozhuchow, Z. A. 1933. Uber die Natur der Extrachromosomen bei Zea mays L. Planta 19:91-116.

Kuwado, Y. 1915. Ueber die chromosomenzahl von Zea mays L. Bot. Mag. Tokyo 29:83-89.

Lane, L. C. 1974. The components of Barley Stripe Mosaic and related viruses. Virology 58:323-333.

Longley, A. E. 1927. Supernumerary chromosomes in Zea mays. J. Agr. Research 35:769-784.

Longley, A. E. 1937. Morphological characters of teosinte chromosomes. J. Agr. Research 54:835-862.

Longley, A. E. 1945. Abnormal segregation during megasporogenesis in maize. Genetics 30:100-113.

Longley, A. E. 1956. The origin of diminutive B-type chromosomes in maize. Am. J. Botany 43:18-22.

Longley, A. E. and T. A. Kato Y. 1965. Chromosome morphology of certain races of maize in Latin America. Intern. Center for the Improvement of Maize and Wheat, Res. Bull. No. 1. Chapingo, Mexico.

McKinney, H. H. 1949. Tests of varieties of wheat, barley, oats and corn for reaction to Wheat Streak Mosaic Virus. Plant Dis. Rep. 33:367-368.

Messieha, M. 1967. Aphid transmission of Maize Dwarf Mosaic Virus. Phytopathology 57:956-959.

Nel, P. 1969. An effect of B chromosomes on crossing over in chromosome 5. Maize Genetics Cooperation News Letter 43:54-55.

Nel, P. 1973. The modification of crossing over in maize by extraneous chromosomal elements. Theo. Appl. Genetics 43: 196-202.

Ostergren, G. 1945. Parasitic nature of extra fragment chromosomes. Botan. Notiser 2:157-163.

Pitout, M. J. and N. Van Schaik. 1968. Deoxyribonucleic acid from Black Mexican Sweet Corn. South African Journal of Science 64:25-29.

Pring, D. R. 1974. Barley Stripe Mosaic Virus infection of corn and the "Aberrant Ratio" genetic effect. Phytopathology 64:64-70.

Pring, D. R. and W. G. Langenberg. 1972. Preparation and properties of Maize Dwarf Mosaic Virus Ribonucleic Acid. Phytopathology 62:253-255.

Randolph, L. R. 1928. Types of supernumerary chromosomes in maize. Anat. Rec. 41:102.

Randolph, L. R. 1941. Genetic characteristics of the B chromosomes in maize. Genetics 26:608-631.

Rees, H. 1974. B chromosomes. Science Progress, Oxford 61:535-554.

Rhoades, M. M. 1942. Preferential segregation in maize. Genetics 27:395-407.

Rhoades, M. M. 1952. Preferential segregation in maize. In: Heterosis, edited by J. W. Gowen. Iowa State College Press, Ames.

Rhoades, M. M. and E. Dempsey. 1957. Further notes on preferential segregation. Maize Genetics Cooperation News Letter 31:77-80.

Rhoades, M. M. and E. Dempsey. 1966. The effect of Abnormal Chromosome 10 on preferential segregation and crossing over in maize. Genetics 53:989-1020.

Rhoades, M. M. and E. Dempsey. 1972. On the mechanism of chromatin loss induced by the B chromosomes of maize. Genetics 71: 73-96.

Roman, H. 1948. Directed fertilization in maize. Proc. Natl. Acad. Sci. U.S. 34:36-42.

Roman, H. 1950. Factors affecting mitotic nondisjunction in maize. (Abstr.) Genetics 35:132.

Rosario, M. S. and E. Del. 1959. Studies of Aceriatulipae Deifer (Eriophyidae) and other eriophyid mites in Kansas in relation to the transmission of Wheat Streak Mosaic Virus. Dis. Abstr. 20:863-864.

Sandfaer, J. 1973. Barley Stripe Mosaic Virus and the frequency of triploid and aneuploids in barley. Genetics 73:597-603.

Scott, G. E. and L. R. Nelson. 1971. Locating genes for resistance to Maize Dwarf Mosaic in maize seedlings by using chromosomal translocations. Crop. Sci. 11:801-803.

Sill, W. H. and P. C. Agusito. 1955. Host Range Studies of Wheat Streak Mosaic Virus. Plant Dis. Rep. 39:635-639.

Snazelle, T. E., J. B. Bancroft and A. J. Ullstrup. 1971. Purification and serology of Maize Dwarf Mosaic Virus and Sugarcane Mosaic Viruses. Phytopathology 61:1059-1063.

Sprague, G. F. and H. H. McKinney. 1966. Aberrant ratio: An anomaly in maize associated with virus infection. Genetics 51:1287-1296.

Sprague, G. F. and H. H. McKinney. 1971. Further evidence on the genetic behavior of AR in maize. Genetics 67:533-542.

Timian, R. G. 1975. Barley Stripe Mosaic Virus and the world collection of barleys. Plant Dis. Rep. 59:984-988.

Ting, Y. C. 1958. On the origin of Abnormal Chromosome 10 in maize. Chromosoma 9:286-291.

Tu, J. C. and R. E. Ford. 1970. Maize Dwarf Mosaic Virus infection in susceptible and resistant corn: Virus multiplication, free amino acid concentrations and symptom severity. Phytopathology 60:1605-1608.

Van Schaik, N. and M. J. Pitout. 1966. Base composition of deoxyribonucleic acid from maize with heterochromatic B-chromosomes. South African Journal of Science 62:53-56.

Ward, E. J. 1973a. Further studies on the effects of accessory chromatin in maize. Can. J. Genet. Cytol. 17:124-126.

Ward, E. J. 1973b. Nondisjunction: Localization of the controlling site in the maize B chromosome. Genetics 73:387-391.

Ward, E. J. 1973c. The heterochromatic B chromosome of maize: The segments affecting recombination. Chromosoma 43:177-186.

Zuber, M. S., E. S. Hilderbrand, P. J. Loesch and A. J. Keaster. 1973. Prediction of reactions to Maize Dwarf Mosaic Virus in double-cross hybrids based upon single-cross reaction. Crop Sci. 13:172-175.