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TIMOTHY CARL VARGAS (Name of student) for the MASTER OF SCIENCE (Degree)

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Title: OIL SPRAYS TO PREVENT THE SPREAD OF BEAN YELLOW MOSAIC VIRUS (BYMV) BY APHIDS

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Dr. K. G. Swenson

The influence of oil sprays on the field spread of the aphid-borne bean yellow mosaic virus (BYMV) in bean was studied in 1972 and 1973. Oil sprays failed to significantly reduce virus spread in the 1972 BYMV experiment. However, the data did suggest that the efficiency of the oil sprays could be greatly improved by timing the applications to periods of peak aphid activity. Several factors were identified which could have contributed to the failure of oil sprays to reduce the incidence of BYMV in 1972. An excessive time lag between bean emergence and the first oil application resulted in a considerable amount of virus spread within all of the plots before any oil sprays were applied. In addition, some of the treatments were subject to a disproportionate number of the dispersing aphid population which resulted in non-uniform virus spread throughout the study area. And all treatments were exposed for an excessive length of time to the

gladioli or primary virus reservoir. As a result, the oil sprayed areas were subject to extreme pressure from the secondary and primary virus reservoirs.

Results of the 1973 BYMV field experiment clearly indicate that oil sprays can protect bean plants against spread of BYMV under field conditions. A number of changes in the experimental design were responsible for the overall success of the 1973 experiment. The time lag between bean emergence and the first oil application was eliminated. This change prevented an excessive amount of virus spread early in the season, thus eliminating the high, non-differential virus spread that results when there is a lack of virus protection early in the season. And the exposure period of beans to the gladioli, primary virus source, was substantially reduced so that oil sprays were directed against the secondary virus source and not both primary and secondary virus sources.

Harvest data collected during 1973 indicated that an increase in plant weight may not occur as a result of virus reduction by oil sprays. The data suggest that repeated applications of oil may bring about a physiological response resulting in an increase in plant weight unrelated to virus reduction.

Oil Sprays to Prevent the Spread of Bean Yellow Mosaic Virus (BYMV) by Aphids

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Timothy Carl Vargas

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Professor of Entomology
in charge of major

Redacted for Privacy

Acting Head of Department of Entomology

Redacted for Privacy

Dean of Graduate School

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INTRODUCTION

Aphids spread stylet-borne viruses in spite of modern organophosphate insecticides. Indeed multiple applications of insecticides have failed to control the spread of stylet-borne nonpersistent viruses (Broadbent, 1963).

Investigations by Bradley (1956) on the effect of depth of stylet penetration on virus transmission resulted in the accidental discovery that mineral oils could reduce aphid transmission of plant virus. Interim studies have been concerned with the types of virus and transmission mechanisms that lend themselves to control by oil sprays. Studies have also been conducted on the types of oil that can be used in virus prevention, the response of the host plant to oil sprays, the relationship between oil sprays and virus control on the yields of certain crops, and the mechanisms involved in the inhibitory effects of oils on virus transfer. Very little attention has been given to factors and variables that influence the effectiveness of oil sprays. These factors cannot be overlooked if oil sprays are to be used effectively and economically to control nonpersistent viruses on certain crops. The purpose of this research was to (a) determine the effects of oil sprays on the spread of bean yellow mosaic virus (BYMV) in

the field, (b) clarify some of the factors which influence the effectiveness of the oil sprays, (c) suggest possible methods to improve the efficiency of oil sprays to control field spread of stylet-borne, nonpersistent viruses, (d) determine the effects of oil sprays on crop yield, and (e) determine the type(s) of virus infection best suited for control by oil sprays.

LITERATURE REVIEW

Types of Aphid Transmission of Plant Viruses

The use of oils to control plant viruses is influenced by the way that an aphid transmits a particular virus. Swenson (1968) described three types of plant viruses depending on their method of aphid transmission:

1. Stylet-borne (nonpersistent) viruses are acquired during brief probes, and the number of aphids acquiring virus decreases with acquisition periods longer than 30-60 seconds. These viruses can be transmitted immediately, without a latent period, are retained for about an hour by feeding aphids and no longer than a few hours by nonfeeding or flying aphids.

2. Circulative (persistent) viruses are retained for many days; often for the life of the aphid. The number of aphids that acquire virus increases as the acquisition feeding period increases. A latent period, varying in time depending on the virus, is required before transmission of he virus is possible.

3. Stylet-borne (semipersistent) viruses are acquired with increasing frequency with acquistion periods up to 12 to 24 hours. No latent period is required and these viruses tend to be retained for one to two days. It has been suggested by Day and Venables (1961) that semipersistent viruses are atypical stylet-borne viruses with different physical properties and different distributions in plant tissues so that transmission varies from that of normal stylet-borne viruses.

Laboratory Experiments with Oil Sprays

The concept of using oil sprays to prevent acquisition and transmission of stylet-borne viruses was introduced by Bradley (1956) who

discovered that transmission of potato virus Y (PVY) was greatly reduced when viruliferous aphids probed through a film of low-melting paraffin wax. Subsequent investigations showed that an oil fraction in the wax was responsible for prevention of virus transmission (Bradley, Wade and Wood, 1962). Some mineral and vegetable oils also prevented or reduced transmission. Bradley (1963), using a light white paraffin oil, found that PVY inoculation by Myzus persicae Sulz. could be reduced in any one of several ways: (1) by coating a leaf with oil prior to aphid feeding; (2) by manually touching the labium of an aphid to a leaf coated with oil; or (3) by inserting the exposed stylets of a viruliferous aphid into oil. He also determined that oil-coated plants prevented acquisition and inoculation of PVY without affecting the probing or feeding behavior of the aphids, a fact not substantiated by Russell (1970), who found that oil sprays had an adverse effect on the settling behavior and larviposition of M. persicae.

Hein (1964) showed that transmission of lettuce mosaic virus (LMV) by M. persicae could be reduced by a film of dried whole milk. Milk reduced virus infection to the same extent whether applied to the test plant, the source plant, or both the test and source plants; thus spraying the test plant and the source plant did not increase the efficiency of the milk sprays. She was able to obtain a high degree of efficiency when either the test plant or source plant was sprayed so that spraying of both did not result in a noticeable increase in

efficiency. Additional research showed that dried skim milk did not protect against LMV as did whole milk (Hein, 1965). She concluded that the effective ingredient was the fat within the milk. Hein (1965) also found that the same effect could be obtained with 3-5% emulsions of corn and mineral oil. Celery mosaic virus was reduced by a film of whole milk or an emulsion of 3% corn oil. The effectiveness of milk in preventing inoculation of LMV by M. persicae was also demonstrated in the greenhouse by Jaeger (1966). Cucumber mosaic virus (CMV) inoculation and acquisition by Aphis gossypii Glov. was reduced by a 1% oil emulsion (Loebenstein, Alper and Deutsch, 1964). Their results differed from the results of Hein (1964). Whereas spraying the test plant gave better results than spraying the source plant, spraying both the source and test plants produced the best results. In addition, they discovered that there was a marked reduction in the number of lesions produced by mechanical transmission of tobacco mosaic virus (TMV) when oil was added to the inoculum. Inoculation of beet yellows virus (BYV), a semipersistent virus, by M. persicae was also shown to be inhibited by oil sprays (Vanderveken, Bourge and Semal, 1966). They also reported that oil sprays failed to prevent the inoculation of the nonpersistent bean common mosaic virus, a fact not substantiated by Vanderveken and Vilain (1967). Inoculation of LMV and beet mosaic virus (BMV) by M. persicae was found to be substantially impeded by painting the lower

sides of lettuce and sugar beet leaves with a 3% oil emulsion and placing virulent aphids on the tops of the leaves (Külps, 1968). Five different oils, in concentrations varying from 0.5-2%, were effective in reducing papaya mosaic virus inoculation by A. gossypii (Bhargava and Khurana, 1969). Additional tests with groundnut oil showed that there was a complete loss of infectivity if virulent aphids were allowed to feed on papaya plants previously sprayed with a 2% oil emulsion. Oil sprays also prevented acquisition of virus when aphids were allowed to feed on healthy papaya plants following an acquisition feeding period on sprayed infected plants. Furthermore, treatment of both the source and test plants resulted in a 90-100% reduction of virus spread under greenhouse conditions. Cousin and Grison (1969) showed that spraying peas with a 3% oil emulsion reduced pea virus 2 (PV_2) inoculation by Acyrthosiphum pisi. They concluded that the purity of the oil, the type of surfactant, the process of surfactant incorporation into the oil, and the method of agitation all influence the success or failure of the treatments.

A 2% oil emulsion was shown to be effective in preventing BYV inoculation by M. persicae (Russell, 1970). Spraying M. persicae with oil after they fed on BYV-infected plants and before transfer to virus-free beet seedlings also prevented virus transmission. Two laboratory techniques of oil application (spraying and leaf infiltration) were investigated by Vanderveken and Dutrecq (1970) in an attempt to

reduce BMV and BYV inoculation by M. persicae and A. pisum. A 3% oil emulsion applied by leaf infiltration was successful in reducing acquisition and inoculation of BMV but not BYV. Oil sprays were successful in reducing acquisition and transmission of both BMV and BYV, but failed to reduce the incidence of pea enation mosaic virus (PEMV), a persistent virus.

The prospects of utilizing oil sprays to prevent plant virus transmission was reviewed by Vanderveken, Semal and Vanderwalle (1968). The use of mineral oils, lipids, silicone oils (synthetic oils), and terpinol (an essential oil) on the transmission of BMV, a non-persistent virus, and BYV, a semipersistent virus was investigated by Vanderveken (1968). BMV transmission was strongly inhibited by mineral oil, corn oil, and whole milk. Only mineral oil was effective in inhibiting transmission of BYV. Terpinol, silicone oils, and skim milk had little effect on the transmission of either BMV or BYV. The prevention of virus transmission by spraying with oil and whole milk for a number of virus-vector-combinations was examined by Hein (1971). The transmission frequency of all the nonpersistent viruses tested was strongly reduced. However, transmission of the persistent potato leaf roll virus was not affected by either oil or milk. This led Hein (1971) to conclude that transmission of all non-persistent viruses can be influenced by oil sprays or treatments in the laboratory.

Field Experiments with Oil Sprays

Allen (1965) conducted the first field test with oil sprays in an attempt to reduce spread of potato virus A (PVA) in potatoes. Six weekly applications of a 1% oil emulsion effectively reduced the spread of PVA by 50% the first year and 30% the second year. There was little difference between the effectiveness of three and six applications of oil for controlling PVA in plots containing infector plants (Bradley, Moore and Pond, 1966). Sprays varying in concentration from 1-3% of either milk or peanut oil effectively reduced the spread of LMV in young lettuce plants (Jaeger, 1966). Nitzany (1966) showed that 26 weekly applications of a 1.25% oil emulsion were effective in reducing the incidence of PVY in pepper. Oil sprays consistently reduced the spread of cucumber mosaic virus (CMV) in the field and low-volume sprays were more effective than conventional high-volume sprays for reducing virus infection (Loebenstein, Deutsch, Frankel and Sabar, 1966). Twenty weekly applications of a 5-10% oil emulsion significantly reduced the spread of iris yellow mosaic virus (Deutsch and Loebenstein, 1967). Additional research showed that the same results could be obtained with only five weekly applications of a 5% oil emulsion.

The effects of oil sprays were tested against three stylet-borne viruses on three different crops (Ascerno, 1969). In most cases, oil

sprays, varying in concentration from 0.5-4% and applied at various intervals, were effective in reducing CMV spread in gladiolus, BYMV in bush beans, and a lily virus in lilies. Ascerno concluded that the timing of sprays in relation to aphid numbers and the maintenance of a uniform oil residue for long periods of time are essential for maximum protection against stylet-borne viruses. Twelve weekly applications of a 1% oil emulsion greatly reduced the incidence of brown ring formation in lilies (Asjes, 1972). However, one of the field tests failed to give satisfactory results. Asjes (1972) attributed the poor results of this test to a lack of surrounding vegetation during the growing season. This allowed dispersing aphids to attack more of the plant and especially those areas where oil coverage was poor, i.e. the base of the plant. Asjes concluded that the effectiveness of oil sprays could be enhanced by planting a tall-growing plant species throughout the field to act as a physical barrier to migrating aphids.

Oil Sprays and Phytotoxicity

Phytotoxicity, as a result of oil sprays, has not been widely reported. However, some researchers have had problems with oil emulsions causing phytotoxicity. One percent emulsions of castor oil and light paraffin oil applied to papaya, in an attempt to prevent transmission of papaya mosaic virus in the laboratory, caused severe phytotoxicity in the form of reduced leaf-size, swollen veins, upward

rolling of leaf margins, and dwarfing of plants (Bhargava and Khurana, 1969). And one percent emulsions of coconut oil and mustard oil caused milder symptoms of phytotoxicity (Bhargava and Khurana, 1969). Cousin and Grison (1969) found that applications of a 3% oil emulsion caused acute toxicity on peas. The appearance and yield of iris were affected after receiving 20 weekly applications of a 5-10% oil emulsion, during attempts to control the spread of iris yellow mosaic virus (Deutsch and Loebenstein, 1967). Reduction of the number of applications to five and a lower application rate (5%) prevented phytotoxicity and reduced virus spread.

Oil Sprays and Crop Yield

Field tests have shown that oil sprays usually cause a significant reduction in virus spread. However, the reduction in virus spread may not always result in a corresponding increase in crop yields. Bradley, Moore and Pond (1966), in an attempt to reduce PVA spread in potatoes, found that while three applications of oil did not affect tuber yields, six applications reduced yields by 15%. Oil sprays failed to increase or decrease pepper yields even though PVY incidence was reduced in the treated plots (Nitzany, 1966). Oil sprays always reduced CMV spread in cucumbers but increases in yields were observed in only two of six experiments (Loebenstein, Deutsch, Frankel, and Sabar, 1966). They concluded that when

infection occurs late and plants are already near full development, the effect of oil sprays on virus spread may be significant but this reduction in virus spread may have little effect on crop yields. When infection occurs early the plants are stunted and oil applied before that time will prevent stunting, resulting in increased yields. The effects of oil sprays, at rates of 1/2 gal. per acre up to a maximum of 4 gal. per acre, on potato yields varied with the variety (Pond, 1966). However, doubling the 4-gallon rate drastically reduced potato yields. The use of emulsifiable oil on potatoes generally resulted in greater yields than non-emulsifiable oils. This trend was reversed when the number of sprays decreased from six to three. The use of oil sprays reduces the spread of virus. However, the decrease in virus spread may not result in an increase in crop yields. The failure of a decrease in virus spread to increase crop yields may result from poor timing of the oil sprays. Irregularities in the type of oil, oil concentration, and number of oil applications may not reduce virus spread and result in a failure to increase crop yields.

The Mechanisms of Oil Action

The mechanism and mode of action of oil have been the subjects of several research papers. The number of local lesions was reduced when TMV was mechanically transmitted in combination with oil (Loebenstein, Alper and Deutsch, 1964). However, when the oil-virus

emulsion was ultracentrifuged highly infectious virus was obtained. They concluded that the oil was not responsible for direct virus inactivation. Hein (1966) also reached this conclusion although she found that oil did not affect mechanical transmission. Oil painted on the lower surface of sugar beet leaves penetrated into the leaves and spread into the subepidermal intercellular cavities (Külps, 1968). He suggested that some stylet-borne viruses may not only be inhibited when aphids insert their stylets through an oil film into the epidermis but also, once into the epidermis, contact with intercellular oil could impede virus transmission. The mechanism by which oil interferes with virus transmission was reviewed by Vanderveken and Vilain (1967). They concluded that the inhibition of virus transmission by oil occurs at the virus-vector or virus-vector host-plant level.

Crane and Calpouzos (1967) found that mineral oil sprays suppressed virus yellows symptoms in sugar beets for 30 to 60 days after symptoms appeared on untreated plants. They concluded that virus transmission was not prevented by a film of oil on the leaf surface. In another paper, Crane and Calpouzos (1968) found that mineral oil sprays suppressed virus yellows symptoms in sugar beets. However, the delay in symptom expression was only 21 to 28 days. They supported their 1967 conclusion that oil sprays may alter the physiology of sugar beet leaves and render them temporarily incapable of expressing symptoms of virus yellows. One percent

emulsions of both castor and mustard oil suppressed papaya mosaic virus symptoms in papaya for 9 and 12 days respectively after symptoms appeared on untreated plants (Bhargava and Khurana, 1969).

The effects of oils on aphid transmission of nonpersistent viruses, semipersistent viruses, and persistent viruses were studied by Vanderveken (1968a). Oil sprays that inhibited the nonpersistent and semipersistent viruses would not inhibit the inoculation of the persistent virus PEMV by Acyrthosiphum onobrychis. The aphid species was not responsible for this failure since oil treatments inhibited BYMV inoculation by A. onobrychis. The lack of inhibition was not due to longer acquisition and inoculation access periods used for the persistent virus, since BYV (a semipersistent virus) transmitted with the same acquisition and inoculation access periods was practically eliminated with oil sprays. Vanderveken (1968b) concluded that the absence of inhibition with aphid-transmitted persistent viruses was due to the virus-vector relationship. In a similar experiment oil sprays that inhibited BMV and BYV failed to inhibit the inoculation of PEMV (Vanderveken and Dutrecq, 1970). They concluded that oil inhibition, in the case of stylet-borne nonpersistent and semipersistent viruses, results from a virus-vector interaction, a conclusion reached earlier by Vanderveken (1968). Furthermore, inhibition occurred whenever contact was established between oil and

virus-bearing stylets.

Oil sprays not only reduced the incidence of BYV inoculation by M. persicae, but oil sprays also had an adverse effect on the settling behavior and larviposition of the vector (Russell, 1970). He concluded that the effect of oil sprays may be two-fold; transmission is inhibited at the stylet-leaf interface and oil sprays may diminish the population of potential vectors present in the sprayed area.

MATERIALS AND METHODS

Aphid Trapping - 1972 and 1973

A Johnson-Taylor Insect Suction Trap (Figure 1) was set up at the Oregon State University Vegetable Research Farm, Corvallis, Oregon. The trap was situated 80 yards south of the BYMV field plot on a strip of fallow ground adjacent to the farm irrigation well during 1972. The trap was located on bare ground 40 feet from a stand of cabbage to the north, 15 feet from an established rhubarb planting to the east, 30 feet from a stand of cabbage to the south, and 200 feet from a bean planting to the west. The trap was set up in the same location in 1973 and except for the corn and tomato planting to the north and the wheat planting to the west, was bordered by the same crops as in 1972.

The trap was operated continually from April 23 through September 21 in 1972 and from May 3 through August 21 in 1973. The trap was emptied each morning between the hours of 9:00 a.m. and 10:00 a.m.

Each time the trap was emptied, the trap collecting cylinder was removed and another cylinder was installed. The cylinder containing the insect catch was placed in an ethyl acetate kill chamber for 5 minutes. The contents of the cylinder was placed in a glass petri dish and examined under a dissecting microscope. There is a

Figure 1. Johnson-Taylor Insect Suction Trap.

lack of vector specificity for BYMV and species that transmit BYMV differ in their ability to do so. In addition, spread occurs by flying aphids as it is doubtful that wingless aphids contribute significantly to virus spread (Swenson, 1968). For these reasons no attempt was made to identify the aphids and only winged aphids were recorded. These records were used to interpret the results of the 1972 and 1973 BYMV field experiment.

Incidence of BYMV in Commercial Gladioli

One hundred gladiolus corms of mixed varieties were planted in the greenhouse at Oregon State University on May 9, 1973. The corms were taken from the same lot used in the 1973 bean yellow mosaic plots. Individual corms were planted in 5 inch clay pots previously filled with a sandy soil. The gladioli were assigned numbers from 1-100 so that an accurate record of plants carrying BYMV could be maintained. Two hundred beans (Phaseolus vulgaris, Var. Oregon 58) were planted in the greenhouse on July 4 at which time the gladioli had reached an average height of 1 1/2 feet. The beans were taken from the same lot used in the 1973 BYMV field experiment, and were planted in sand at the rate of two plants per 4 inch clay pot. Each pot of beans was assigned a number from 1-100 corresponding to the number of the gladiolus plant.

Mechanical inoculation of BYMV from the gladioli to the beans

began on July 17 when the first true leaves of the bean plants had a desired amount of leaf growth (4 sq. inches). However, because of a physiological disorder only 92 beans could be used in the first test. These beans were numbered 1-46 (two beans per pot) and inoculated from gladiolus plants with corresponding numbers. Additional beans were planted at this time to replace those that were discarded. A modified procedure of mechanical virus transmission was used for transferring BYMV from the gladioli to the beans (Yarwood and Fulton, 1957). The inoculum was prepared by grinding 3-4 cm^2 from gladiolus plant tissue in a mortar containing 5 mls. of a 1% K_2HPO_4 solution. The gladiolus tissue used was the youngest or most recently developed plant tissue from either the leaves, buds, or petals. The inoculum was applied to the leaf surface of the bean plants, previously dusted with carborundum, by dipping the index finger into the inoculum and lightly rubbing the inoculum onto the leaf surface. Care was taken not to rub too hard since excessive rubbing could destroy the epidermal cells of the bean plants and render them unsuitable for virus infection. The surface of each treated leaf was lightly rinsed with distilled water and the plants were set aside for observation of virus symptoms. On August 3 the second group of beans, numbered 47-100, were labeled and inoculated from the gladiolus plants with corresponding numbers.

The beans were observed for a total of 4 weeks. Beans showing

BYMV symptoms were removed from the test and the corresponding gladiolus plants were marked as being virus carriers. The remaining gladiolus plants were used to re-inoculate the bean plants which had not developed symptoms of BYMV. This process continued until virus could no longer be transmitted from gladioli. It was then assumed that the remaining gladioli were virus-free.

Field Experiments with Bean Yellow Mosaic Virus in 1972 and 1973

Plot Design

Twenty-five 285-foot east-west rows were lined out and fertilized at the Oregon State University Vegetable Research Farm. A 36-inch row spacing was used and all crops were planted on level ground. Rows 1, 7, 13, 19, and 25 were planted with corn (Zea mays, Var. Golden Jubilee). Rows 4, 10, 16, and 22 were planted with gladioli of mixed varieties. The corn plants were allowed to reach a height 2 1/2 feet and the gladioli a height of one foot before the remaining rows were planted with beans (Phaseolus vulgaris, Var. OR. 58). The corn rows served as a physical barrier to prevent aphid movement from between plots in adjacent columns. A column consisted of two rows of beans planted on each side of a single row of gladioli (Figure 2). The gladioli served as the primary source of BYMV. Each of the four columns were divided into four 60-foot

Figure 2. Two columns (4 rows of beans and 1 row of gladioli) are shown on either side of one of five corn row barriers early in the season.

subdivisions by leaving a ten-foot strip of fallow ground every 60 feet within the bean rows. This formed four replicates that ran north-south through the plots. Four replicates of four treatments were randomized throughout the plots so that each subdivision represented one treatment within a replication to form a 4 X 4 latin square design. In addition to the main plot design (Figure 3), a single 285-foot row of beans was planted 200 yards south of the experimental area and adjacent to a row of gladioli. This single row of beans was used to determine the amount of virus spread outside of the test area. The gladioli were removed after the spray schedule began so that additional virus spread took place from the secondary source of virus infection, i.e., those beans that were infected with BYMV prior to the removal of the gladioli. Gladioli adjacent to the single row of beans outside of the experimental area were not removed during the study. The planting dates for the 1972 field experiment were as follows: corn May 11, gladioli May 18 and beans June 20. The 1973 field experiment was initiated later in the spring with the following planting dates; corn June 11, gladioli June 21, and beans July 5. The single row of beans in the 1973 experiment was planted on June 4. The gladioli were removed from the plots on July 11 in 1972 and on July 16 in 1973.

Figure 3. 1972 and 1973 BYMV field plot design (gladioli already removed).

Treatments

All treated plots were sprayed with a 2% oil water emulsion using VOLCK Supreme Oil. A three-gallon compressed-air hand sprayer was used to apply the treatments in the 1972 field experiment (Table 1). The spray emulsion was agitated manually by shaking the sprayer while in operation. The beans began to emerge on June 30 and the spray schedule began when the beans had reached the two- and three-leaf stage. Treatment A was applied on July 5. The first spray of treatment B was applied on July 6 and the first spray of treatment C was applied on July 7. The first day of application was staggered for all treatments so that the spray concentration could be altered if the beans showed any sign of oil-induced phytotoxicity. The beans and gladioli were sprayed until runoff.

Table 1. Schedule of treatments used in the 1972 BYMV Field Experiment.

Treatment*	First spray	Interval between sprays	Date of application
A	5 days after emergence	None	7/5
B	6 days after emergence	3 days	7/6 & 9
C	7 days after emergence	3 days	7/7, 10 & 13
D	Experimental check	None	

*A = 1 spray
B = 2 sprays
C = 3 sprays
D = check

A three gallon STIHL SG17 power mist blower (Figure 4) was used to apply the treatments in the 1973 field experiment (Table 2). The spray emulsion was agitated by normal body movement during the spraying operation. The beans began to emerge on July 13 and the first spray application for all treatments was made on July 14. An attempt was made to thoroughly cover both the beans and gladioli with a fine mist, but not to the point of runoff.

Virus counts, in both years, were taken when symptoms of BYMV, as described by Zaumyer and Thomas (1957) (Figure 5), began to appear within the plots and continued at weekly intervals for a total of five weeks. Virus counts were made from July 12 through August 8 in 1972 and from July 25 through August 22 in 1973. Counts were made by manually examining each individual plant and recording the presence or absence of BYMV. In order to facilitate the counting procedure, infected plants were marked with 8-inch plant labels to avoid recounting infected plants. An analysis of variance based on the total number of plants infected was conducted on the data collected from each weekly virus count. In addition to the data collected on virus spread, harvest data were collected from the 1973 field experiment. The middle 30 feet of beans from each treatment or subdivision were pulled on August 31 and placed in burlap sacks. The sacks were weighed and the fresh weight of the total plants was calculated for each treatment. These data were then used in an

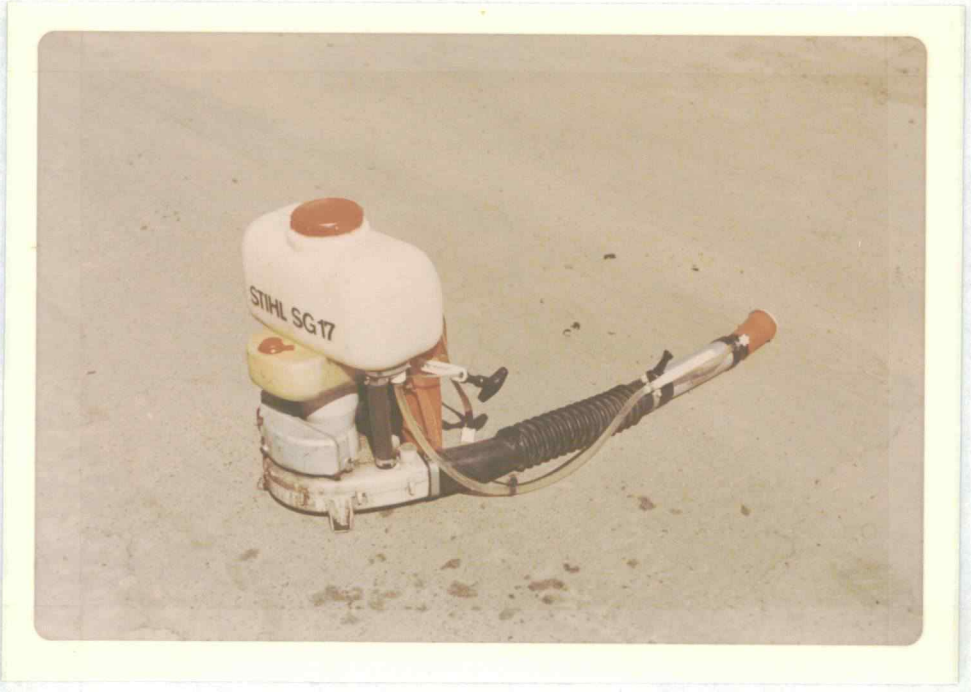

Figure 4. STIHL SG17 Power Mist Blower.

Figure 5. Bean infected with BYMV.

Top: Infected plant.
Bottom: Healthy plant.

analysis of variance to determine the effect of oil treatments on total plant weight.

Table 2. Schedule of treatments used in the 1973 BYMV Field Experiment.

Treatments*	First spray	Interval between sprays	Date of application
A	At emergence	3 days	7/14 & 17
B	At emergence	3 days	7/14, 17, 20 & 23
C	At emergence	3 days	7/14, 17, 20, 23, 26 & 29
D	Experimental check	None	

*A = 2 sprays

B = 4 sprays

C = 6 sprays

D = check

RESULTS AND DISCUSSION

Aphid Trapping - 1972 and 1973

The number of aphids trapped during 1972 and 1973 are shown in Tables 3 and 4, respectively. A discussion of the aphid trapping data and the graphic representation of the relationship between aphid numbers and virus spread is included in the discussion of the 1972 and 1973 field experiments with BYMV.

Researchers in the past have failed to recognize that there are peaks of aphid flight activity. It is during these peak flight periods that the threat of virus spread is greatest. Timing oil sprays to periods of peak flight activity would be much more effective in controlling the spread of virus than random applications made without considering aphid numbers.

Incidence of BYMV in Commercial Gladioli

Table 5 summarizes the results from the first group of gladioli screened for BYMV in the greenhouse. Eleven of the 46 gladiolus plants in group one were carriers of BYMV. Observations from July 17, 7 days after the first inoculation, through July 31 showed that six gladioli were virus carriers since BYMV symptoms developed in the corresponding bean plants. An additional five gladioli were found to be carriers of BYMV at the end of the second observation

Table 3. Numbers of winged aphids collected in a Johnson-Taylor Suction Trap at the Oregon State University Vegetable Research Farm Corvallis, Oregon - 1972.

Date	Months trap was in operation					
	April	May	June	July	Aug.	Sept.
1		0	14	185	8	0
2		0	6	201	7	0
3		0	2	175	0	4
4		0	2	115	0	2
5		0	17	135	1	6
6		4	18	80	0	0
7		2	20	60	2	0
8		1	10	72	0	0
9		2	5	63	0	4
10		8	10	73	0	8
11		5	11	81	0	9
12		3	14	42	1	10
13		3	43	66	1	3
14		2	15	210	9	6
15		3	24	205	6	6
16		1	70	208	18	7
17		0	60	45	7	5
18		3	88	32	5	2
19		7	74	34	20	3
20		1	25	0	23	0
21		0	63	0	18	2
22		4	33	2	9	
23	1	4	101	2	10	
24	0	2	90	0	16	
25	0	5	110	4	12	
26	0	7	106	3	8	
27	0	8	210	5	7	
28	1	5	213	4	3	
29	0	8	224	2	6	
30	0	1	175	8	8	
31		1		10	4	

Table 4. Numbers of winged aphids collected in a Johnson -Taylor Suction Trap at the Oregon State University Vegetable Research Farm Corvallis, Oregon - 1973

Date	Months trap was in operation			
	May	June	July	Aug.
1		27	171	67
2		82	153	24
3	19	93	82	23
4	32	108	124	20
5	41	71	113	17
6	50	74	191	15
7	62	43	187	8
8	30	68	103	56
9	39	80	326	13
10	71	52	33	20
11	103	76	52	9
12	135	22	58	16
13	115	61	193	15
14	144	43	47	66
15	244	13	68	35
16	429	9	-	22
17	98	17	17	41
18	78	165	8	38
19	157	249	34	45
20	86	196	20	24
21	130	135	27	12
22	156	60	33	
23	37	23	45	
24	26	32	72	
25	40	53	49	
26	57	150	53	
27	70	340	63	
28	107	77	41	
29	156	140	23	
30	41	153	10	
31	30		7	

period (August 7-21), following inoculation on July 31. There was no sign of BYMV symptoms in beans at the end of the 3-week period following inoculation on August 21. Two of the gladiolus plants in the first group died from bulb rot.

Table 6 summarizes the results from the second group of gladioli screened for BYMV in the greenhouse. Thirteen of the 54 gladioli in group two were carriers of BYMV. Observations during the 3-week period following inoculation on July 27 showed that 9 gladioli were carriers of BYMV. Four more gladioli inoculated August 17 were identified as virus carriers after a 3-week observation period. There were no symptoms of BYMV in beans after the 2-week period following the September 7 inoculation date. One of the gladioli in group two died from bulb rot.

Results of this experiment showed that of the original 100 gladiolus corms planted in the greenhouse, 3% died from bulb rot and 24-25% were carriers of BYMV, thus one out of four gladioli planted in the 1973 field experiment were carriers of BYMV. It should be kept in mind that these corms were obtained from a good commercial source.

The purpose of this experiment was to determine the amount of virus present in the gladioli (primary virus source) planted in the 1973 BYMV field experiment, and to utilize these data to eliminate some of the variation in primary virus spread due to differences in virus

Table 5. Incidence of BYMV in commercial gladioli (No. 1-46).

	Gladiolus number[1]	Date symptoms first observed	Incubation period[2]
A. Inoculation date - July 10, 1973			
	1	July 25	15 days
	6	July 20	10 days
	31	July 30	20 days
	36	July 20	10 days
	37	July 23	13 days
	38	July 26	16 days
B. Inoculation date - July 31, 1973			
	12	August 13	13 days
	14	August 18	18 days
	20	August 16	16 days
	39	August 17	17 days
	42	August 18	18 days
C. Inoculation date - August 21, 1973			
	No sign of virus transfer from gladioli to beans.		

1 = Includes only those gladioli found to be carriers of BYMV.

2 = Number of days between inoculation and symptom expression.

Table 6. Incidence of BYMV in commercial gladioli (No. 47-100).

	Gladiolus number[1]	Date symptoms first observed	Incubation period[2]
A. Inoculation date - July 27, 1973			
	48	August 9	13 days
	50	August 16	20 days
	52	August 16	20 days
	53	August 14	18 days
	73	August 13	19 days
	80	August 14	18 days
	82	August 10	14 days
	87	August 13	19 days
	93	August 8	12 days
B. Inoculation date - August 17, 1973			
	65	August 20	24 days
	69	September 2	16 days
	76	September 4	18 days
	85	August 30	13 days
C. Inoculation date - September 7, 1973			
	No sign of virus transfer from gladioli to beans.		

1 = Includes only those gladioli found to be carriers of BYMV.

2 = Number of days between inoculation and symptom expression.

incidence in gladioli corm lots and in aphid populations. If one can determine the amount of virus contained in the primary virus source prior to field experimentation, it would be possible to standardize the amount of primary virus spread in any given year or location. It may be possible to do this by using the percent virus found in the primary virus source and daily aphid trapping data. For example, assume that 20% of the gladioli in 1973 were infected with BYMV and the exposure period of beans to gladioli was 4 days. During these 4 days 500 aphids were trapped and virus counts from check plots after 3 weeks showed that the amount of primary virus spread was 40%. Now assume, in the following year, that mechanical transmission studies showed that 10% of the gladioli to be used as a primary virus source were infected with BYMV. Since the gladioli contained half as much virus as in the previous year, exposure to twice as many aphids (1000) should result in approximately the same amount of primary virus spread. Therefore, the time of removal of the gladioli could be determined by virus incidence in the gladioli and the number of aphids trapped. However, more than one factor may influence the amount of primary virus spread in any given year. One factor influencing the amount of primary virus spread is the composition of the aphid population during the period of bean exposure to gladiolus. If the aphid population, in 1973, was composed primarily of species particularly efficient as vectors of BYMV and if the

population the following year was composed of inefficient vectors of BYMV, then the amount of primary virus spread would differ even though efforts were made to minimize this difference.

Field Experiments with Bean Yellow Mosaic Virus in 1972 and 1973

Table 7 summarizes the weekly virus counts for 1972 and gives the levels of significance from an analysis of variance based on a latin square.

The third virus count (July 24) showed that 67% of the control plants were infected. Treatment A had 59% virus incidence, treatment B had 48% virus incidence, and treatment C had a virus incidence of 54%. However, the last virus count (August 8), showed that 98% of the control plants were infected, 93% of the treatment A plants were infected, 99% of the treatment B plants were infected, and 95% of the treatment C plants were infected. It should also be noted that row and column differences were highly significant for most of the count dates. In addition, 99% of the plants in the single row of beans outside of the experimental area were infected on August 8.

The treatment effects appear negligible when examining the data (Table 7). The general lack of statistical significance was probably due to a number of key factors any of which could have had an influence on the success or failure of the oil sprays. The first and most

Table 7. Weekly counts of bean plants showing BYMV symptoms in 1972.

Date	Treatments A	B	C	D	Total	Signif. level	Rows 1	2	3	4	Signif. level	Columns 1	2	3	4	Signif. level
July 12	80*	82	88	69	319	$P \leq .20$	57	82	70	110	$P \leq .01$	74	72	80	93	$P \leq .20$
July 18	368	300	387	357	1412	$P \leq .20$	265	324	352	471	$P \leq .20$	313	342	351	406	$P \leq .20$
July 24	608	487	525	702	2322	$P \geq .01$	490	613	549	670	$P \leq .02$	501	546	563	712	$P \geq .01$
July 30	896	871	849	984	3600	$P \leq .20$	834	905	865	996	$P \leq .20$	760	928	872	1040	$P \geq .05$
August 8	961	978	931	1027	3897	$P \leq .20$	936	982	910	1069	$P \leq .20$	876	993	927	1101	$P \geq .10$
Total	2913	2718	2780	3139			2582	2906	2746	3316		2582	2906	2746	3316	

* = total number of plants showing BYMV symptoms.

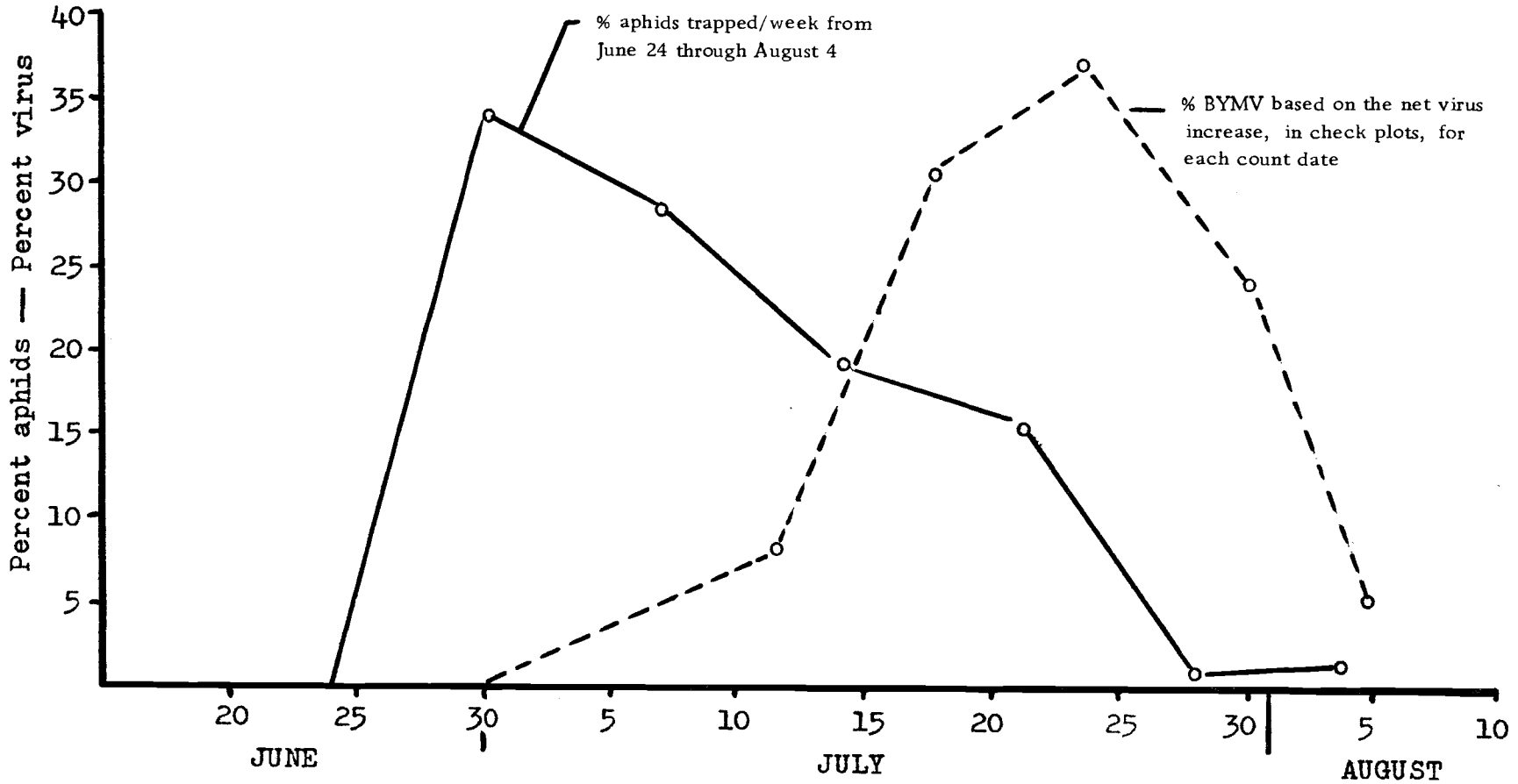

Figure 6. Relationship between aphid numbers and incidence of BYMV in 1972

apparent was the excessive time lag between bean emergence and the first oil application. This time lag coupled with the fact that a large number of aphids were trapped during this period (Table 3) was very important, since the beans had virtually no protection early in the season when bean susceptibility was probably the greatest (Swenson, 1968). As a result, a considerable amount of virus spread occurred within all of the plots before any oil sprays were applied. Significant differences among treatments may have occurred throughout the season had the oil sprays been applied when aphid numbers were high, if one considers the positive relationship between dispersing aphid numbers and virus spread. The relationship between aphid numbers and virus spread is illustrated in Figure 6. It is evident from the graph that a positive relationship exists. The fact that there was a sudden increase in the number of aphids trapped on July 14 (Table 3), immediately following the oil sprays probably accounts for the significant differences among treatments for the July 24 count date (Table 3). If one figured back 10-12 days (the number of days between inoculation and symptom expression for BYMV, Tables 3 and 4) from the July 24 count date and used this date as the last day that aphid flights could have influenced the July 24 BYMV count date, one can see that oil sprays apparently reduced the incidence of BYMV (Table 7) resulting in a significant difference for treatments. However, in the absence of additional oil sprays virus

infection would tend to equalize. For example, aphids landing in a plot with 20% of the plants already infected with virus would have a greater chance of infecting a healthy plant than would aphids landing in a plot in which 50% of the plants were already infected. After several weeks, assuming that the rate of virus infection was the same for both plots, there would be a significant decrease in the magnitude of the difference between the two plots. This probably accounts for the small difference among treatments on the date of the last virus count (Table 7), and had there been more oil sprays, significant differences among treatments may have been apparent from the third count date through the final virus count date.

Some of the variation among treatments had nothing to do with treatment effects. The presence of high virus incidence in replicate four and column four suggests that aphids entered the plots from the east or northeast (Table 7). However, it is doubtful that aphids entered the plots from these directions when one considers the prevailing coast winds. It is also doubtful that a windbreak effect as described by Lewis (1966) influenced aphid density in the plots. An explanation as to why treatments located at the eastern end of the plots were subject to a disproportionate number of the virulent aphid population remains difficult at best.

The lengthy exposure period of all treatments to the gladioli or primary virus source probably affected the results. This lengthy

exposure to the primary BYMV source and the excessive lag period between bean emergence and oil applications contributed to the substantial amount of virus spread early in season. In addition, the presence of a large primary source of virus throughout the period of oil application meant the oil sprays were subject to extreme pressure in the form of a tremendous secondary (those beans infected with BYMV) and primary (infected gladioli) virus reservoir. The gladioli were removed prior to the second period of high virus potential which contributed to the significant difference among treatments from the July 24 BYMV count date.

In view of the above factors, the results of the 1972 BYMV field experiment (Table 7) may have led to a better understanding of the mechanisms involved in limiting virus spread with oil sprays. Above all, this experiment pointed out the need for precise timing of oil sprays in relation to plant maturation and aphid numbers.

Table 8 summarizes the weekly virus counts for 1973 and gives the levels of significance from an analysis of variance based on a latin square. Figure 7 illustrates the relationship between aphid numbers and virus spread for 1973.

The third virus count (August 1) showed that 78 % of the control plants were infected. Treatment A had 55% virus incidence, treatment B 58%, and treatment C 57%. Unlike the last count date of 1972 (August 8), in which there was little difference among treatments,

Table 8. Weekly counts of bean plants showing BYMV symptoms in 1973.

Date	Treatments				Total	Signif. level	Rows				Signif. level	Columns				Signif. level
	A	B	C	D			1	2	3	4		1	2	3	4	
July 25	123	161	154	255	693	$P \geq .05$	185	154	180	174		126	218	177	172	$P \leq .20$
August 1	405	417	470	595	1887	$P \geq .05$	485	416	468	518		381	508	475	523	$P \geq .20$
August 8	539	556	555	768	2418	$P \geq .10$	619	547	602	650		481	631	631	675	$P \geq .20$
August 15	639	634	612	821	2706	$P \geq .05$	691	626	664	725		569	699	707	731	$P \geq .20$
August 22	717	709	66	883	2975	$P \geq .05$	757	708	730	780		658	770	755	792	$P \leq .20$
Total	2423	2477	2457	3322			2737	2451	2644	2847		2215	2826	2745	2893	

* = total number of plants showing BYMV symptoms.

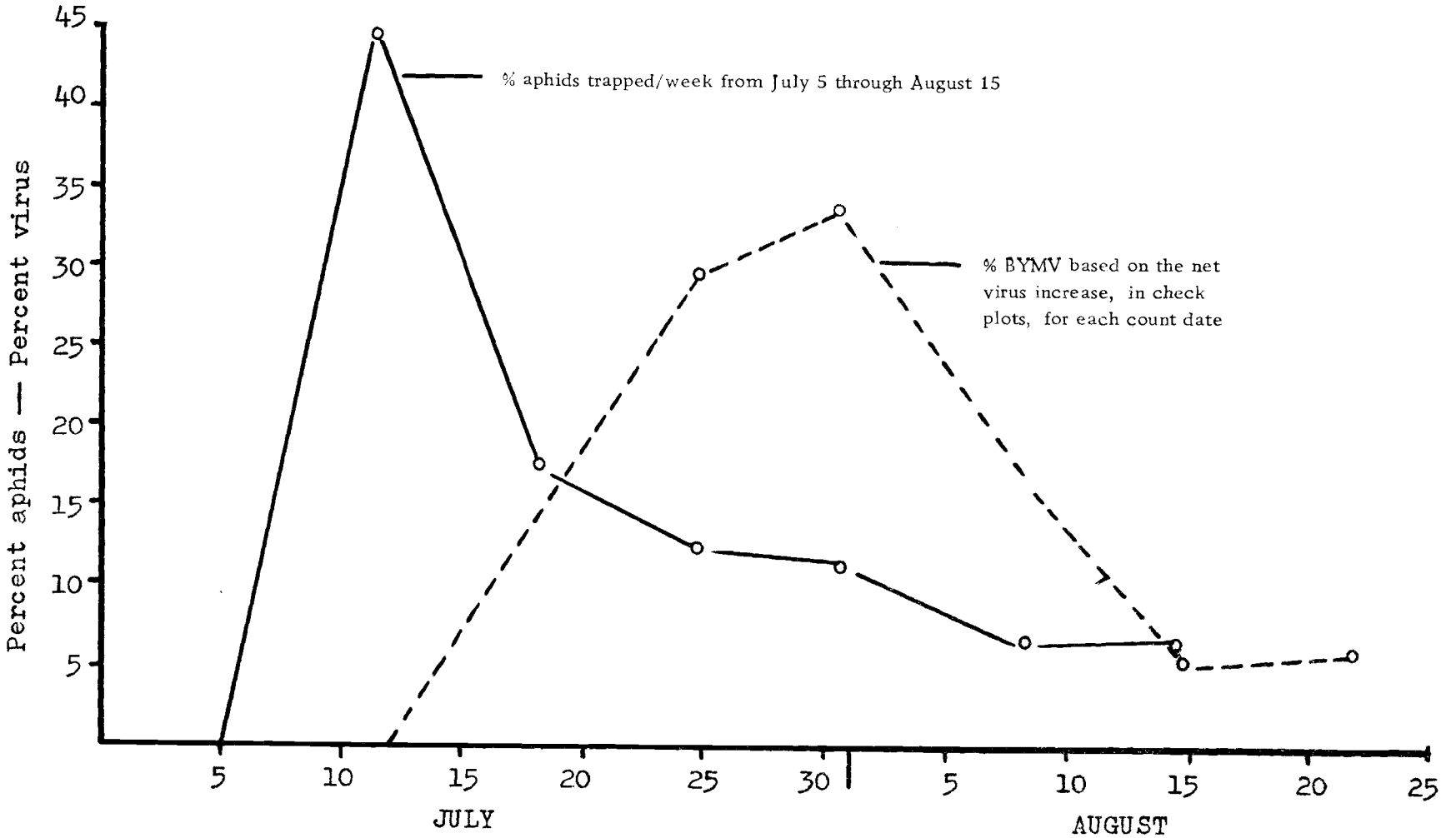

Figure 7. Relationship between aphid numbers and incidence of BYMV in 1973.

the final count date of 1973 (August 22), showed that 90% of the control plants were infected, 73% of the plants in treatment A were infected, 74.5% of the plants in treatment B were infected, and 68% of the plants in treatment C were infected. There was no significant difference among replications (rows) and only a moderate degree of significance occurred among columns. In addition, 100% of the beans outside of the experimental area were infected on August 22.

Results of this experiment clearly indicate that oil sprays protected beans from BYMV infection. A number of changes in experimental technique, resulting from the findings of the 1972 BYMV field experiment, contributed to the general success of this experiment. The excessive time lag between bean emergence and the first oil application was eliminated by beginning the spray treatments when the beans were in the late cotyledon stage. This eliminated the high non-differential virus spread that results when there is a lack of virus protection early in the season. It should be noted that the initial virus count (July 25) occurred 3 days after the first virus symptoms were noticed. This would account for the seemingly high virus level of that count date (Table 8).

The exposure period of all treatments to the gladioli or primary source of virus was drastically reduced. Thus, oil sprays were directed against secondary virus spread and not both secondary and primary virus spread. It should be kept in mind that the purpose

of these field experiments was not to reduce primary virus spread, but to reduce secondary virus spread resulting from primary infection, with oil sprays. The gladioli were included to ensure enough initial virus spread to make the experiment worthwhile.

As was previously stated, the 1973 BYMV field experiment showed that oil sprays effectively reduced BYMV spread in bean. However, the effects of the oil sprays were not proportionate since most of the reduction in virus spread can be attributed to the first and second spray applications. This accounts for the small differences among treatments A, B, and C on the majority of the count dates (Table 8). Two factors may have contributed to the disproportionate effects of the oil sprays. The first and most plausible explanation is that the first two oil applications were made early in the season when the bean seedlings were most susceptible to virus infection (Swenson, 1968). Therefore, oil applications made during this period would have a greater potential for reducing virus spread than applications made later in the season when the bean plants were more mature and less susceptible to virus transmission. It is also possible that the composition of the aphid population changed, during the period in which the first two oil sprays were applied, from one composed of members particularly efficient as vectors of BYMV to one composed of inefficient vectors of BYMV for the remainder of the experiment. In view of the rather constant daily number of aphids trapped (Table 4)

during the spray schedule one would have to assume the first explanation to be the most probable of the two.

An analysis of variance for the harvest data collected from the 1973 BYMV field plots is shown in Table 9. Yields based on the total fresh weight of the bean plants from the middle 30 feet of each treatment were as follows: treatment A = 194.5 lbs., treatment B = 218.5 lbs., treatment C = 219.0 lbs., and treatment D = 195.75 lbs.

Table 9. Analysis of Variance for 1973 harvest data based on a 4 X 4 latin square design.

Source of variation	SS	df	MS	F
Rows	11.28	3	3.76	0.3275
Columns	206.59	3	68.86	5.9982[1]
Treatments	138.22	3	46.07	4.0130[2]
Error	68.91	6	11.48	
Total	425.00	15		

1 = Significance at the P 0.10 level.

2 = Significance at the P 0.20 level.

Plots in columns one and two were harvested and weighed in the morning and the plots in columns three and four in the afternoon which probably accounts for the relatively high significance level for columns. Plots harvested in the morning contained a higher moisture content than plots harvested in the afternoon.

Initially, it appears that an increase in plant weight resulted from the use of oil sprays to reduce virus spread among plants in treated plots; however, this may not have been the case. In examining the data in Table 8, it is apparent that there was very little difference in virus incidence among treatments A, B, and C on each of the five count dates; however, plants in treatment A weighed considerably less than plants from treatments B and C. One possible explanation for these results may be that oil sprays act as an anti-transpirant. Repeated applications of oil slows down the transpiration rate of the plant causing an increase in plant weight. In this case two sprays may not have been sufficient, but repeated applications of four to six sprays may have been sufficient to reduce the transpiration rate of the plant causing an increase in plant weight. The fact that treatment C received two more oil applications than treatment B without a corresponding increase in plant weight raises a second possible explanation. It may be that the effects of oil on plant weight resulted from applications during the periods of water stress.

In summary, the data collected from the 1973 BYMV field experiment provide evidence that oil sprays can reduce the spread of stylet-borne viruses; however, the data also indicate that a reduction in virus spread may not result in a corresponding increase in plant weight.

SUMMARY AND CONCLUSIONS

The influence of oil sprays on the field spread of the aphid-borne bean yellow mosaic virus in bean was studied in 1972 and 1973. Oil sprays failed to significantly reduce virus spread in the 1972 BYMV experiment. Several factors were identified which could have contributed to the failure of oil sprays to reduce the incidence of BYMV in 1972. Firstly, an excessive time lag between bean emergence and the first oil application resulted in a considerable amount of virus spread within all of the plots before any oil sprays were applied. Secondly, some of the treatments were subject to a disproportionate number of the dispersing aphid population which resulted in non-uniform virus spread throughout the study area. Thirdly, all treatments were exposed for an excessive length of time to the gladioli or primary virus source. As a result, the oil sprays were subject to extreme pressure from the secondary and primary virus reservoirs.

The 1972 data indicate that oil sprays failed to reduce virus spread; however, the data does suggest that the efficiency of the oil sprays could be greatly improved by timing the applications to periods of peak aphid activity.

Results of the 1973 BYMV field experiment clearly indicate that oil sprays can protect bean plants against spread of BYMV

under field conditions. A number of changes in the experimental design were responsible for the overall success of the 1973 field experiment. The time lag between bean emergence and the first oil application was eliminated. This change prevented an excessive amount of virus spread early in the season thus eliminating the high non-differential virus spread that results when there is a lack of virus protection early in the season. Secondly, the exposure period of beans to the gladioli, primary virus source, was substantially reduced so that oil sprays were directed against the secondary virus source and not primary and secondary virus sources.

Harvest data collected during 1973 indicated that an increase in plant weight may not occur as a result of virus reduction by oil sprays. The data suggest that repeated applications of oil may bring about a physiological response resulting in an increase in plant weight unrelated to virus reduction.

Research has shown oil sprays to be effective in reducing a number of stylet-borne viruses on several different crops. However, oil sprays have sometimes failed to increase crop yields even though there may have been marked reductions in the incidence of virus. For this reason, future research should be designed to study oil sprays on such crops as lilies and gladioli where more benefit can be derived from reducing the primary source of virus rather than attempting to increase yields in susceptible plants.

In conclusion, research directed at reducing virus incidence on crops in which the value of the commodity is based on the presence or absence of virus as well as timing oil applications to coincide with peak migrations of known virus vectors could determine the practicality of using oil sprays to produce virus free plant material.

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