Studies on Epidemiology of Virus Disease of Chickpea in California

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ABSTRACT

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A shift of chickpea culture from summer to winter sowing in California revealed viruses as major production constraints. The incidence of viruses in field trials at Davis, Salinas, and the San Joaquin valley was 60–100%. Six viruses were detected: beet western yellows (BWYV), legume yellows (LYV), subterranean clover red leaf (SCRLV), alfalfa mosaic (AMV), cucumber mosaic (CMV), and lettuce mosaic (LMV). Three aphids were confirmed vectors: *Myzus persicae* (of BWYV and LMV), *Aphis craccivora* (of CMV), and *Acyrthosiphon pisum* (of LYV, SCRLV, and AMV). Aphid monitoring at Davis showed a peak in March–April, followed by high levels of virus incidence. Aphid survival and fecundity in greenhouse trials were markedly less on chickpea varieties with glandular hair exudates than on a hairless variety. Vectors survived long enough, however, to efficiently transmit viruses. Young plants infected with viruses were often killed; infection at later stages had less effect. Virus incidence and yield loss varied among varieties, indicating that selection for low virus incidence (tolremicity) should be a breeding objective.

Chickpeas (*Cicer arietinum* L.) have been grown in coastal areas of California for many decades. In these areas of Mediterranean climate, summer rainfall is essentially absent and chickpeas are grown in heavy black soils with residual soil moisture derived from winter rains, often in yearly rotation with winter wheat or barley. Attempts were made in the winter of 1984–1985 to determine if a shift to winter sowing could result in higher yields and if by winter sowing the crop could also be successful in the central San Joaquin valley where high summer temperatures preclude this cool-season legume as a spring-sown crop. Under dryland culture in the Mediterranean region, the shift from spring to winter sowing of chickpeas results in a 50–100% yield increase if disease is not limiting. The disease of concern in the Mediterranean region is Ascochyta blight (absent in California), which is favored by winter rains (16).

Many plants in the winter-sown experimental plots in California turned yellow in April and either remained stunted or died. Symptoms of dying plants often resembled symptoms attributed earlier to the root-rot complex (22) or to *Thielaviopsis basicola* (Berk. & Broome) Ferraris (3). Isolations for fungal pathogens revealed various fungi in the drying roots but no consistent pathogen. A viral cause was implicated by careful sequential examination of symptom appearance and development, which revealed that phloem discoloration and yellowing preceded wilting. Moreover, several plots were sown in "new" areas where chickpea fungal pathogens would not be expected. Observations in farmers' fields during

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1985–1987 also revealed that isolated chickpea plants undergoing early senescence often had phloem discoloration and that these were virus-infected (*unpublished*).

Approximately 12 viruses affecting chickpea have been reported from various parts of the world, including India, Iran, Australia, and the United States (California and Washington State) (2,5,8,10,12–14,16,21). It appears that viruses have not been considered important in affecting chickpea production except in a few countries, including Iran (12) and northern India (16). It is not clear from the literature whether this apparent lack of importance is real or whether insufficient surveys and research on etiology are responsible. Two brief, unconfirmed reports of three viruses in chickpea in California appeared in the 1950s (10,21). More recently, Duffus (8) listed chickpea as a host of two luteoviruses.

In order to better understand the potential role of viruses affecting winter-sown chickpeas in California, studies were conducted in three locations during 1987–1988. The objectives of our studies were to: 1) identify the most prevalent viruses in chickpea in California and their insect vectors, 2) monitor aphid abundance and species composition on chickpea, 3) monitor incidence of viruses and their impact on yield, 4) study the effect of planting date on virus incidence and yield, and 5) study varietal differences in reaction to virus infection and natural challenge.

MATERIALS AND METHODS

Virus identification. Samples consisting of branch terminal buds and leaves of chickpea plants that showed virus symptoms were tested by means of enzyme-linked immunosorbent assay (ELISA), immunodiffusion tests, host range studies, and electron microscopy. Potential alternate hosts of the viruses were also observed and tested.

Viral antisera utilized in these tests were obtained from the following: beet western yellows (BWYV), legume yellows (LYV), and subterranean clover red leaf (SCRLV) from J. E. Duffus and H. Y. Liu, USDA, Salinas, California; alfalfa mosaic (AMV), cucumber mosaic (CMV), and lettuce mosaic (LMV) from B. Falk, University of California at Davis (UC Davis); and bean yellow mosaic (BYMV) from D. Gonsalves, Cornell University, Geneva, New York. ELISA tests were also done by M. McLaughlin, USDA, Mississippi State University. Confirmatory serological tests for LMV were performed by R. Provvidenti, Geneva, New York, and J. E. Duffus. ELISA tests for luteoviruses, AMV, and CMV were done using the method of Clark and Adams (6). Immunodiffusion tests for LMV, AMV, and CMV were done with agar gels (18).

For host studies of mechanically transmissible viruses (AMV, CMV, and LMV), 1–2 g of tissue from infected plants was ground, using a mortar and pestle, with 15 ml of 0.04 M phosphate buffer (pH 7) and 320-grit Carborundum. The solution was then rubbed onto the leaves of test plants. The plants were kept in the greenhouse and observed regularly for symptoms. Electron microscopy of negatively stained preparations (for AMV, CMV, and LMV) was performed by the facilities for Advanced Instrumentation at UC Davis.

Virus transmission. Three aphid species—*Aphis craccivora* Koch, *Acyrthosiphon pisum* (Harris), and *Myzus persicae* (Sulzer)—were used for transmission studies with the various viruses found in chickpea. Non-viruliferous colonies of *Aphis craccivora* and *A. pisum* were reared on faba bean and those of *M. persicae*, on mustard (cv. Florida Broadleaf). For virus transmission tests, aphids were confined to infected plants kept in cages and allowed a 24-hr acquisition feeding and 48-hr inoculation period for transmission of luteoviruses and a 15-min. acquisition feeding and 24-hr inoculation period for transmission of nonpersistent viruses. After the inoculation periods, aphids were removed and test plants were kept in the greenhouse for observation of symptoms. Virus isolates tested in these studies came originally from chickpea. The luteoviruses were obtained from J. E. Duffus and the others from field-collected plants showing virus symptoms.

Aphid biology. Survival and fecundity of *Aphis craccivora, A. pisum*, and *M. persicae* were studied on three varieties of chickpea: Surutato, Chafa, and Chafa Glabrous. Chafa Glabrous was obtained from the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Hyderabad, India, and is a mutant of Chafa that does not produce glandular hairs. Five 1-day-old adult aphids were placed on each host plant, where they reproduced. Plants were confined inside aphid cages and kept in a greenhouse at 20 ± 4 C. Adult survival was recorded and nymphs were counted and removed every second day. The survival and fecundity test was performed with 10 plants per variety for each aphid species and was terminated after 10 days.

Aphid monitoring. Two water-pan aphid traps were established in trials at Davis. Traps were painted green and consisted of a wooden frame (35 × 35 × 60 cm) with an aluminum foil pie pan (2.5 cm deep × 25 cm diam.) to which a mixture of water and polyethylene glycol was added every other day. Aphids were collected from the traps weekly and stored for later identification.

A suction trap was used at the UC Davis Agronomy Farm to collect aphids at a height of 10 m. This trap was located approximately 1 km northeast of the chickpea plots. Aphids collected from October 1987 to June 1988 were identified by K. Pike, Pullman, Washington. Aphids were also collected from chickpea plants and weeds in experimental plots and identified.

Bait plants. Potted greenhouse-grown Surutato chickpea plants were used to determine incidence of virus at different periods. Successive batches of bait plants (50 2-wk-old plants per batch) were kept in the field from September 1987 until June 1988. Each batch remained in the field for 14 days and was then transferred to the greenhouse for observation of symptoms and for virus assay. The proportion of plants in each batch that became infected was then determined; this is defined as "infection pressure" (19). Viruses were identified as described above.

Field trials. Field experiments were conducted at the UC Davis Agronomy Farm, the UC West Side Field Station (WSFS) in the central San Joaquin valley, and the USDA research farm at Salinas. Field plots of four chickpea varieties—UC15, UC27, Surutato, and 85150—were established on 11 November, 24 November, and 2 December 1987 at Davis, WSFS, and Salinas, respectively. Plots were arranged in a randomized complete block design (RCBD) with four replications, consisting of 10 rows 0.76 × 10 m, sown at a rate of 100 seeds per row. The entire group of plots at each location was bordered by chickpea variety UC5. Plots

Table 1. Percentage of sampled chickpea plants with virus symptoms found to be infected with luteoviruses in three locations in California, 1988

Location	Month			
	February	March	April	May
Davis				
BWYV	83	57	75	55
LYV	0	0	0	0
SCRLV	0	7	0	5
Total[a]	83	57[b]	75	57[b]
WSFS[c]				
BWYV	8	12	19	18
LYV	8	0	11	12
SCRLV	0	0	0	0
Total[a]	16	12	27[b]	22[b]
Salinas				
BWYV	50	8	24	10
LYV	0	0	0	0
SCRLV	0	21	14	12
Total[a]	50	29	29[b]	22

[a]Differences between totals and 100% were various mechanically transmissible viruses (CMV, AMV, LMV).
[b]Totals less than individuals summed are due to double infections.
[c]West Side Field Station in central San Joaquin valley.

were large in order to reduce border effects; stands were adjusted later to 80 plants per row. Before planting, seeds were treated with metalaxyl (Ridomil) to reduce seedling diseases.

A second trial was established at Davis, with monthly sowing from October 1987 to February 1988 (five planting dates). Four varieties—UC15, UC27, ILC144, and ILC3375—were planted, using four replicatons in a RCBD. Plots consisted of four rows 0.76 × 6 m long, with 60 plants per row. The entire group of plots of each planting date was bordered by two rows of chickpea variety UC5 on the north and south sides and by blocks of 16 rows of UC5 across the east and west ends.

Virus incidence was surveyed visually and recorded from the two center rows of each plot—monthly on the first set of trials and bimonthly on the second. All plants showing virus symptoms were marked with different colored flags according to date found. To obtain information on impact of virus infection on plant yield, three virus-infected plants (specific viruses not determined) from each survey date and three healthy plants were harvested from each plot and the seed yields recorded. The remainder of the two center rows was then harvested and the yields determined.

Within-field spread of virus was estimated by examining spatial distribution and occurrence of symptoms within a late-planted chickpea plot at Davis. In this observational plot, rows were 1.5 m apart. To determine the degree of clustering of virus infection, 100 infected plants in randomly chosen areas were marked with flags and checked for neighbor infection at later dates.

Table 2. Percent female mortality after a 48-hr period and mean number of nymphs produced per female over a 10-day period for three species of aphids reared on chickpea, Davis, California, 1988

	Aphis craccivora		*Acyrthosiphon pisum*		*Myzus persicae*	
Chickpea variety	Percent mortality	Mean no. nymphs/ female[a]	Percent mortality	Mean no. nymphs/ female	Percent mortality	Mean no. nymphs/ female
Surutato	100.0	0.3	72.0	0.1	100.0	0
Chafa	100.0	2.0	100.0	2.3	100.0	0
Chafa Glabrous	12.5	40.9	23.0	31.3	27.5	22.4

[a]Means of 10 plants, five female aphids per plant.

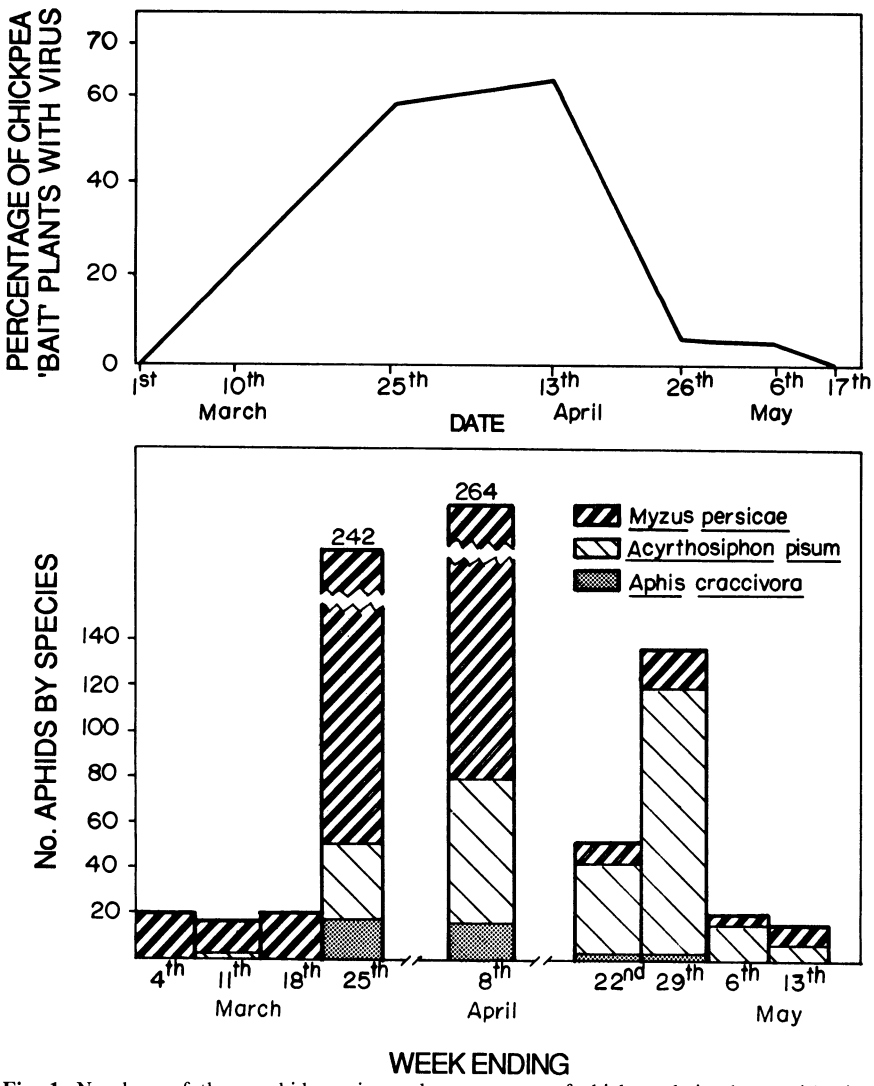

Fig. 1. Numbers of three aphid species and percentages of chickpea bait plants with virus infection during spring 1988 at Davis, California. (Aphids not sampled on 1 and 15 April.)

RESULTS

Virus identification. Six aphid-transmitted viruses were found infecting chickpea in our trials: BWYV, LYV, SCRLV, AMV, CMV, and LMV. This was the first determination of LMV naturally infecting chickpea (1). CMV had never been reported in chickpea in the United States before.

Luteovirus-infected plants became off-green, followed by a gradual yellowing, stunting, and often death. AMV, CMV, and LMV initially caused tip wilting, followed by yellowing, stunting, and sometimes death. In general, field symptoms of plants were very similar regardless of the virus or virus mixture, but severity varied.

At Davis, BWYV always accounted for over 50% of the virus infections, ranging from 55% in May to 83% in February (Table 1). Other viruses found at Davis were LMV, LYV, SCRLV, AMV, and CMV, with several others (based on symptom differences) remaining unidentified. The pattern at WSFS was different, with luteoviruses accounting for only 12–27% of the infections (Table 1). SCRLV was not found at this location, and incidence of BWYV was slightly higher than that of LYV. CMV and AMV were also detected at WSFS, where incidence of CMV was two to three times that of AMV. The relative importance of luteoviruses at Salinas was between that at the other two locations, with 22–50% of infections caused by this group of viruses (Table 1). AMV and CMV were also found at Salinas. We could not confirm the presence of LMV on chickpea at this location, but it likely occurs, since LMV is common in commercial lettuce fields in this area.

We were not able to identify all the mechanically transmissible viruses found in experimental chickpea plots. On the basis of symptomatology, it appeared that BYMV and pea enation mosaic virus were also present. Both viruses have previously been reported infecting chickpea in California (10,21). Limited serological tests by M. McLaughlin suggested the presence of clover yellow

vein virus (CYVV) in chickpea. Under greenhouse conditions, we were able to mechanically transmit CYVV from pea (*Pisum sativum* L.) to chickpea, obtaining severe virus symptoms similar to those observed in the field. Thus, this virus could occur naturally in chickpea; more extensive surveys are needed to ascertain this.

Virus transmission. All the viruses detected were transmitted by aphids in our greenhouse studies. *M. persicae* successfully transmitted BWYV from chickpea to chickpea after 24-hr acquisition access and 48-hr inoculation access periods. LYV and SCRLV were transmitted in a similar fashion by *A. pisum*. AMV was transmitted by *A. pisum* and CMV by *Aphis craccivora* after acquisition access periods of 15 min and inoculation access periods of up to 24 hr. LMV was readily transmitted between chickpeas in a nonpersistent manner by *M. persicae* after acquisition feeding periods of 15 min.

Numerous attempts to transmit LMV and the luteoviruses (including LYV) with *Aphis craccivora* were unsuccessful. This is in contrast to reports from India where *Aphis craccivora* is reported as the most important vector of bean (pea) leafroll luteovirus (15,16), which some consider to be closely related to LYV (J. E. Duffus, *personal communication*; [11]). Our attempts to transmit LMV between chickpeas using *A. pisum* were also unsuccessful. Provvidenti (17), however, reported *A. pisum* as a vector of LMV on peas.

Aphid biology. Chickpea plants were seldom found colonized by aphids in our experimental plots or in farmers' fields. Most of the aphid colonies found were of *Aphis craccivora* and rarely were of *A. pisum*. *M. persicae* was never found colonizing chickpea, although single aphids were found occasionally. Chickpeas exude a fluid containing high concentrations of malic acid, and this compound is known to deter insect attack (20). When colonies of *Aphis craccivora* were found, they were usually on old petioles or peduncles and leaflets where glandular hair exudate production appeared to be low.

Fecundity and mortality of the three aphid species were assessed in the greenhouse on Surutato and Chafa, which have abundant glandular hairs, and on Chafa Glabrous, a mutant form that lacks these hairs. For all three aphid species, mortality was high (72–100%) in 48 hr on the varieties having glandular hairs, in contrast to low mortality on the hairless mutant (Table 2). This suggests that the glandular exudate, rather than other tissue constituents of chickpea, is the lethal agent. Fecundity also was severely inhibited on the varieties having glandular hairs (Table 2).

Aphid monitoring and bait plants. Incidence of virus at Davis was monitored with bait plants from September 1987 until June 1988. During mid-September and early October, an average of 2% of the bait plants became infected with either LMV or LYV. From 10 October until 5 March, no virus infection of the bait plants occurred. Aphid catches during this time showed little aphid activity; thus, the absence of virus could be explained by the absence of vectors. The number of aphids flying began to increase by mid-March and reached a peak in early April (Fig. 1). Incidence of virus followed a similar pattern, increasing from 23% of the bait plants infected in the second week of March to 62% in the first 2 wk of April (Fig. 1). Aphid numbers are given only for *M. persicae*, *A. pisum*, and *Aphis craccivora*, because these were the only

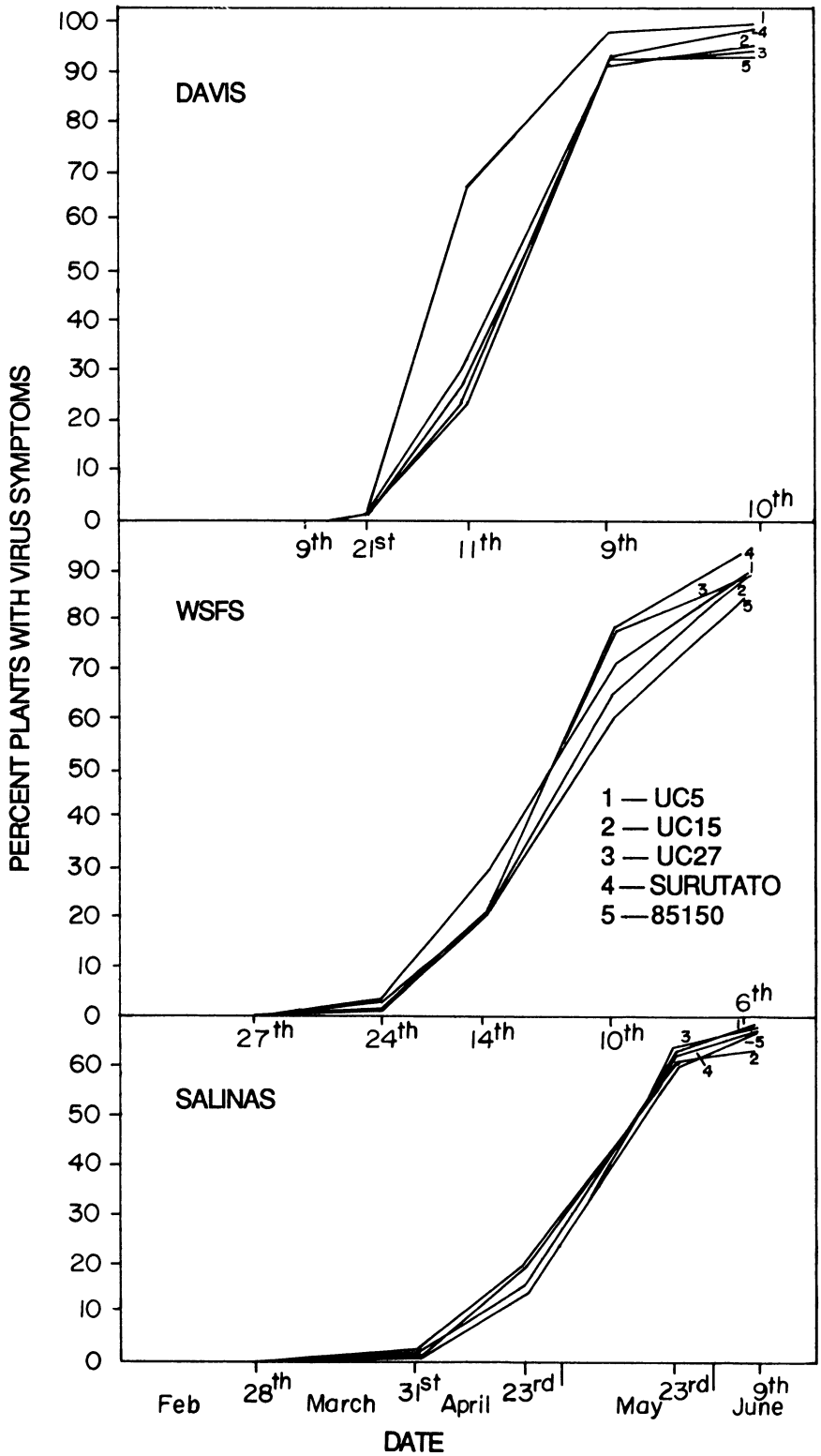

Fig. 2. Percentages of chickpea plants of five varieties with virus symptoms in three locations in California on five survey dates, 1988. WSFS = West Side Field Station, San Joaquin valley.

species confirmed to be vectors in our greenhouse studies. Species composition changed markedly during the season, with *M. persicae* dominant from early March until mid-April, when most of the virus transmission to bait plants occurred. The numbers of *A. pisum* reached a peak in late April, but by then virus incidence declined drastically (Fig. 1). This suggests that *M. persicae* was largely responsible for virus transmission to chickpea at Davis. *M. persicae* was the most efficient vector of BWYV in our greenhouse tests, and BWYV was the most commonly found virus at Davis.

Field trials. Chickpeas sown at the three locations from 11 November to 2 December germinated; the plants remained small and had no symptoms of virus infection during the winter. By the last week of March, virus symptoms were visible on a few plants at each location, and incidence increased rapidly during April, reaching 95% at Davis by early May (Fig. 2). Incidence at Davis was greater and increased faster than at WSFS; incidence at Salinas was only about 60% by the last week of May, compared with 80% at WSFS and 100% at Davis.

Although virus incidence varied slightly among the five chickpea varieties, the only major difference was the earlier higher incidence in UC5 at Davis, probably because of a border effect, since UC5 was planted at the end of the field. The bait plant data indicated that field infection in early season occurred approximately 2 wk before symptoms appeared. Thus, disease incidence (Fig. 2) reflects earlier infection of 2 wk or more.

Significant differences in yield occurred among varieties and locations (Table 3). The Davis and WSFS locations have similar good soils and similar winter climates. In the absence of virus, chickpeas yield similarly in both locations (*data not shown*); the higher yields of chickpeas at WSFS than at Davis (Table 3) probably reflect the lower incidence of BWYV (Table 1), the lower overall virus incidence, and the lesser effect of late virus infection. The varietal yield differences at Davis reflected an earlier high incidence of virus on UC5 and a greater depression of yield with later infection on the later maturing variety 85150. Surutato was less depressed in yield with late-season infection than the other varieties, explained by its being an earlier variety. The yield of UC5 at WSFS was also depressed owing to higher incidence of early virus infection. At Salinas, a more coastal location, plot yields were low despite lower virus incidence, because of poor plant growth on a granitic sandy soil prone to drought. Surutato, with a smaller root system, suffered considerable yield depression in Salinas. The other varieties have similar yield potential if viruses are absent (*data not shown*).

Virus infection (appearance of symptoms) any time before flowering usually either killed the plants or prevented any seed yield. Infection at 5 mo reduced yield by 45–80% at WSFS, by 70–82% at Salinas, and by 90–99% at Davis (Fig. 3). Infection at about 6 mo resulted in a yield reduction of only 0–30% at WSFS, of 27–58% at Salinas, and of 63–79% at Davis (Fig. 3).

Results on individual plant yield are based on means of three plants per plot,

Table 3. Mean[a] yield (kg/ha) of five chickpea varieties in three locations in California, 1988

Chickpea variety	Location		
	Davis	Salinas	WSFS[b]
UC15	1,043	1,147	2,369
UC27	1,056	871	2,441
Surutato	562	314	1,717
85150	787	845	2,054
UC5[c]	384	1,072	1,782
CV		22.7	
F probability			
Location		<0.01	
Variety		<0.01	
Variety × location		NS	
LSD (5% level)		395.8	

[a] Means of four replicates, two 10-m rows each.
[b] West Side Field Station in central San Joaquin valley.
[c] Not included in statistical analysis.

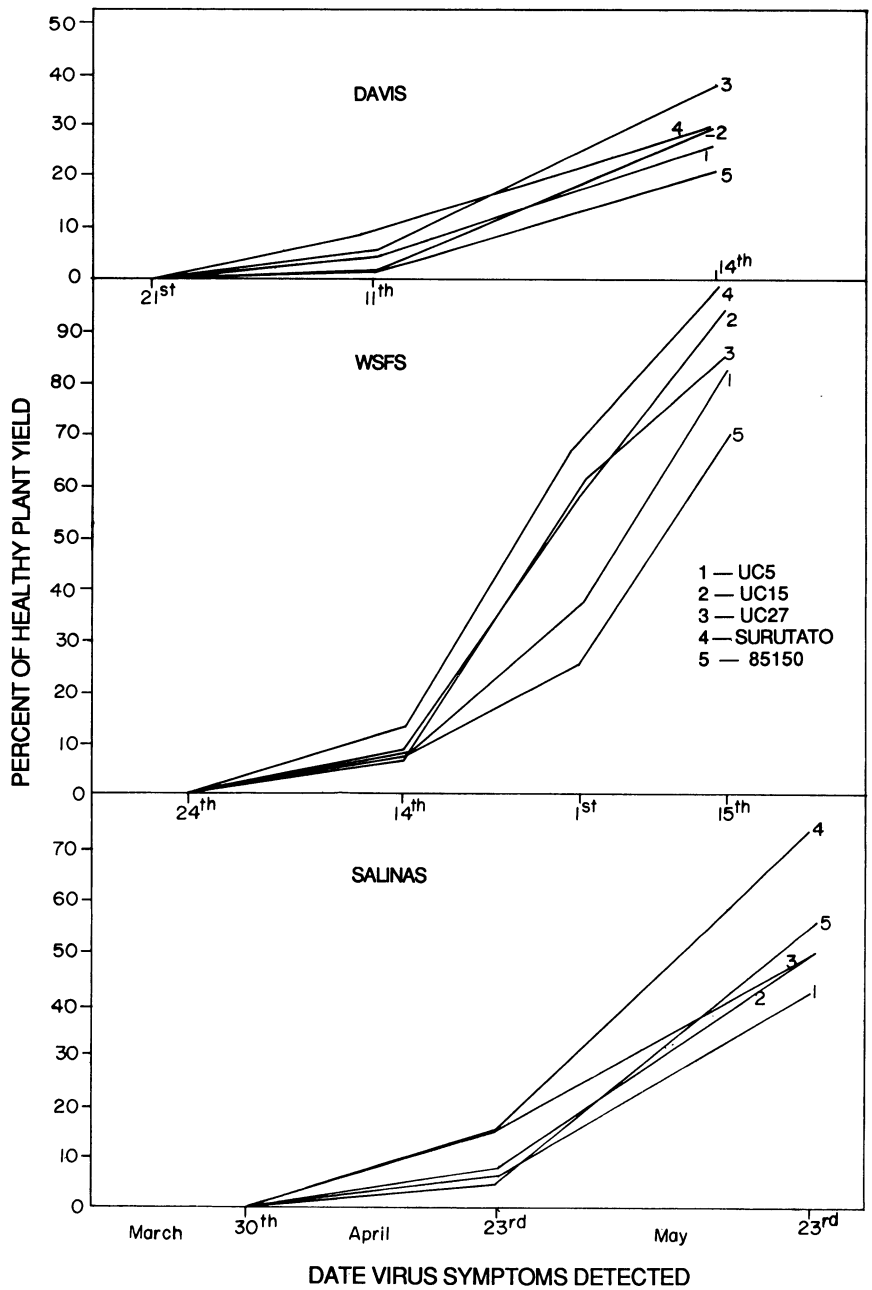

Fig. 3. Yield of chickpea plants of five varieties naturally infected with viruses at different phenological stages, expressed as percentages of healthy controls, in three locations in California, 1988. Figures represent means of three plants per replicate, four replicates per variety. WSFS = West Side Field Station, San Joaquin valley.

comparing infected with healthy plants in the same rows. The comparisons thus reflect within-row competition between virus-stunted and neighboring healthy plants. Because of compensation, the yield depressions reflect more than virus effect per se. Yields were significantly reduced by virus infection in all varieties, with less depression the later in the season that infection occurred (Fig. 3). The later maturing variety 85150 had a greater yield depression with late infection. Later infection at Davis resulted in much greater yield reduction than at the other two locations, with late infection at WSFS being especially mild in effect. The more severe effect at Davis may be due to the higher incidence of BWYV at this location (Table 1). Despite almost total infection, plot yields were substantial for some varieties. This reflects the small effect of virus infection on plants close to maturity. Thus, the interaction of timing and incidence of infection and growth stage critically influences virus effect on yield (Table 3, Figs. 2 and 3).

In the date-of-planting trial at Davis, virus incidence increased with time in all plots but varied by variety and planting date (Fig. 4). The October planting was severely affected by crown blight caused by *Sclerotinia sclerotiorum* (Lib.) de Bary, and it was abandoned. The chickpeas in the plots planted later were little affected by crown blight. Virus incidence was highest on variety ILC3375 on all survey dates in plots of each planting date. In the February planting on the 6 April survey date, virus incidence was 23% on ILC3375 and only 3–7% on the other varieties (Fig. 4). ILC3375 had been selected to represent the highly susceptible class, and it had lower plot yields at all planting dates than the other varieties (Table 4). Individual healthy plants of ILC3375, however, yielded similarly to those of other varieties, and virus-infected plants were equally depressed in yield. Thus, it was the high virus incidence that lowered plot yields.

The incidence of virus in April survey dates was lowest on ILC144 for all planting dates (Fig. 4). In the December planting, incidence was very low in early April (17% compared with up to 37% for other varieties). There were highly significant differences in yields (kg/ha) among the four varieties tested, dates of planting, and the interaction between varieties and dates (Table 4). Yield of ILC144 in the December planting was almost double that of UC15 and UC27 (Table 4), both of which had higher incidence of virus (Fig. 4). However, yield differences among these three varieties for other planting dates were not significant. The yield of individual ILC144 plants infected with virus was not significantly different ($P = 0.05$) from those of infected plants of other varieties. ILC144 plants infected with virus had severe yield depressions, ranging from 69 to 100%, when compared with healthy checks. This variety, thus, appears not to be tolerant of virus infection, but it is tolremic, i.e., it has a lesser tendency to become infected (4).

Observations on within-field spread revealed that, initially, plants with symptoms appeared at random. Of the flagged plants, 71% remained bordered by healthy plants to maturity. In 9% of sites, a neighboring plant became visibly diseased 2–4 wk after the originally diseased plant; a proportion of this 9% could have been new random infections other than spread from a neighbor infected earlier. However, 20% of virus-infected plants flagged appeared to have been infected as pairs in a row. This degree of clustering was assumed to be from an initial viruliferous invader and not due to within-field spread.

Weed hosts. There were abundant winter weeds near the plot at Davis, and alfalfa, sugar beets, safflower, and various fruit crops were also planted nearby. Winter and early spring weeds included many species of Cruciferae, including wild radish and shepherd's purse (*Capsella bursa-pastoris* (L.) Medik.), known hosts of BWYV (9). *Malva* spp., *Lactuca* spp., common groundsel (*Senecio vulgaris* L.), and *Chenopodium* spp. were the most common early spring weeds. Many of these are hosts of both *M. persicae* and BWYV (7,9).

At WSFS, winter mustards, London rocket (*Sisymbrium irio* L.), and *Malva* spp. were the major winter and early

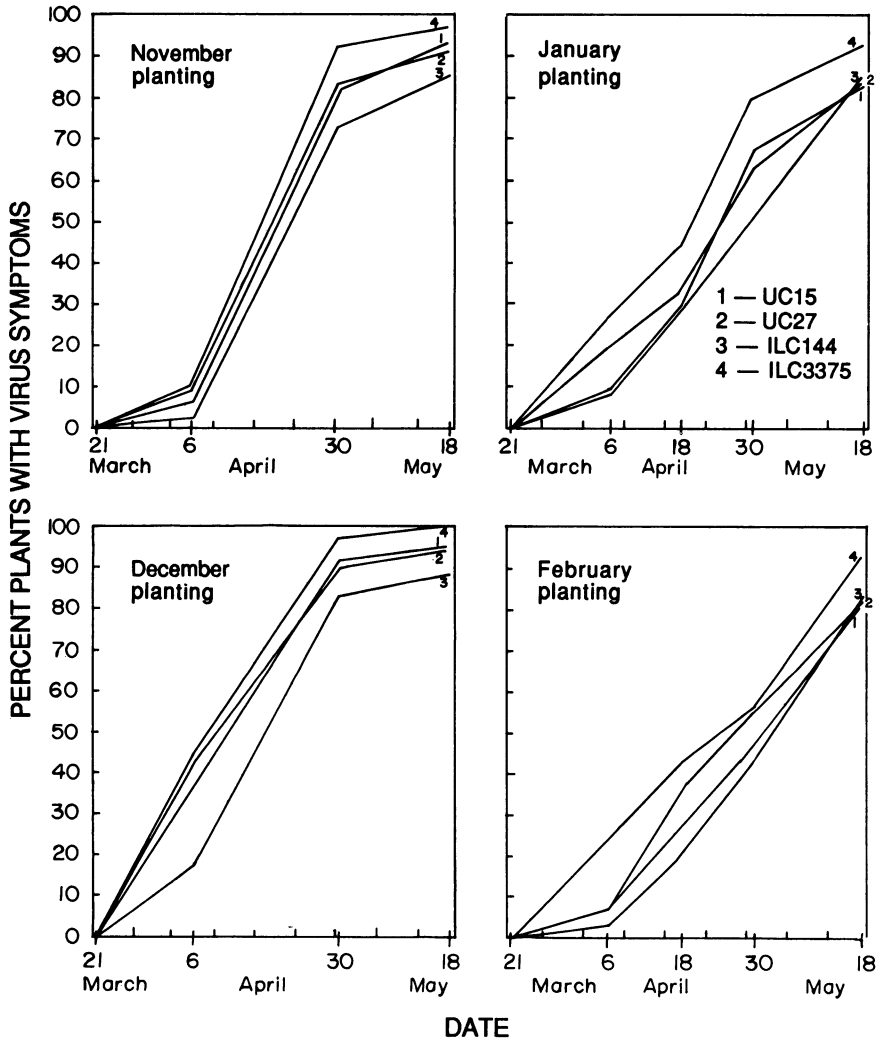

Fig. 4. Percentages of chickpea plants with virus symptoms as affected by planting date, variety, and survey date, Davis, California, 1988.

Table 4. Mean[a] yield (kg/ha) of four chickpea varieties on four different planting dates, Davis, California 1988

Chickpea variety	Planting date			
	Nov. 1987	Dec. 1987	Jan. 1988	Feb. 1988
UC15	825	446	1,565	1,098
UC27	660	423	1,825	1,012
ILC144	937	817	1,798	1,188
ILC3375	138	49	501	375
CV	30.8			
F probability				
Variety	<0.01			
Date	<0.01			
Variety × date	<0.01			
LSD (5% level)	311.8			

[a] Means of four replicates, two 6-m rows each.

spring weeds. Nearby fields contained almond orchards, lettuce, broccoli, and, more distantly, alfalfa, sugar beets, and safflower. Sampling and testing with ELISA revealed BWYV in safflower, sugar beet, alfalfa, shepherd's purse, and *Malva* spp. and LYV in alfalfa, from several locations.

DISCUSSION

In summary, chickpeas are vulnerable to many viruses that occur naturally in California, but incidence is strongly related to location. Moreover, all viruses and vectors are invading from weeds and other crops, and therefore the nature of the agroecosystem near any given chickpea field will determine timing and amount of virus infection.

Coastal summer-grown chickpeas generally escape much viral infection, and the shift to winter sowing creates virus vulnerability and considerable yield loss at some sites. In the central San Joaquin valley, large fields of winter-planted chickpeas may or may not be subject to high virus incidence and yield loss, depending on location and year. The plant size and growth stage of the crop at the time of vector flights in the spring apparently influence incidence and definitely influence the effect of infection on yield and seed quality.

Our data confirm the importance of the glandular exudate (largely malic acid) as a major defense mechanism against aphid colonization of chickpea. The presence of the exudate is not enough, however, to prevent virus transmission to chickpea. It is not known if the exudate increases incidence of the nonpersistently transmitted viruses by increasing mobility of invading aphid migrants before they are killed. In our tests, all aphid species were able to survive 24–36 hr on chickpea varieties with abundant exudate. This suffices for virus transmission to occur, as shown by our transmission tests, which explains why a crop that is seldom colonized by aphids can still incur such high incidence of virus infection. This also points out the need to breed chickpeas for resistance to viruses, because the relatively high level of insect resistance in this crop (20) is clearly not enough to prevent epidemics of aphid-transmitted viruses.

The spatial distribution and timing of symptom appearance, combined with the paucity of aphid colonization in chickpea, indicate that most virus transmission resulted from viruliferous invaders, initially infecting one or two plants at a site, and that there was little within-field spread. How many chickpea plants an invading vector would probe before dying on chickpea in less than 48 hr is unknown. More extensive research is required to firmly determine the details of the nature of spread. More extensive experiments are also required to accurately determine the role of *Aphis craccivora* as a vector of chickpea viruses in California and elsewhere. Our evidence indicates that it is not an important vector, even though it is the most commonly found aphid on chickpea in California.

Our data plus observations in farmers' fields reveal that individual plant response to virus infection is only very indirectly related to plot or field yield loss. If varietal susceptibility is high and infection occurs only at early stages and at random, even a moderate incidence may affect yield very little. This is because infected plants are killed while small, and healthy neighbors grow into their space, compensating for their loss. Infection occurring later in crop phenology may result in greater yield loss, if infected plants are not killed and neighboring healthy plants are unable to compensate because of competition for resources. Finally, a point is reached where very late infection is hardly damaging because seed set and seed fill are largely complete or can be completed from plant reserves. In farmers' fields, infection frequency is usually higher near field edges, and very large fields may have little visible incidence when plants are in full pod set, even when the fields had readily visible incidence of up to 30% 1 mo earlier. Plants infected shortly after flowering may produce seed that is small, stained brown, and shriveled. This reduces quality and results in an excessive winnowing loss. Thus, depending on complex variables of the epidemic, losses can be of both seed quality and quantity, and these losses will vary greatly because of many interactions.

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