Chickpea Wilt Incited by Pea Streak Carlavirus

WALTER J. KAISER, Research Plant Pathologist, USDA-ARS, Western Regional Plant Introduction Station, Washington State University, Pullman 99164-6402; ROBERT E. KLEIN, Assistant Plant Pathologist, and RICHARD C. LARSEN, Research Plant Pathologist, USDA-ARS, Irrigated Research Extension Center, Washington State University, Route 2, Box 2953-A, Prosser 99350-9687; and STEPHEN D. WYATT, Associate Professor, Department of Plant Pathology, Washington State University, Pullman 99164-6430

ABSTRACT

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Pea streak carlavirus (PSV) incited a widespread wilting and yellowing disease of chickpea (*Cicer arietinum*) in commercial and experimental plantings in the Palouse region of eastern Washington and northern Idaho. Incidence of PSV usually ranged from 0.5 to 5%. Experimental host ranges of several Palouse PSV isolates were confined to the Fabaceae and one species of Amaranthaceae. Systemic necrosis developed in chickpea, lentil (*Lens culinaris*), pea (*Pisum sativum*), fenugreek (*Trigonella foenum-graecum*), and faba bean (*Vicia faba*), while alfalfa (*Medicago sativa*), white sweet clover (*Melilotus alba*), and hairy vetch (*Vicia villosa* subsp. *villosa*) were symptomless carriers of PSV. The virus produced local lesions without systemic spread in *Gomphrena globosa, Senna obtusifolia*, and *S. occidentalis*. At Central Ferry, Washington, the virus was isolated from naturally infected *Medicago lupulina, M. sativa*, and *Melilotus alba*, but not from 40 other wild species. Pea streak virus was isolated from 93% of alfalfa fields sampled, and virus incidence ranged from 0 to 44%, making alfalfa the primary reservoir and overwintering host of PSV in the Palouse region. All 55 cultivated chickpea germ plasm accessions tested, as well as eight wild annual species of *Cicer*, were susceptible to PSV in inoculation tests. Four wild perennial species of *Cicer* were resistant to the virus. Seed yields of three chickpea lines were reduced 97–99% by inoculation at prebloom and 16–50% by inoculation at full bloom. Seed quality was also adversely affected. No seed transmission of PSV was detected in chickpea, lentil, *M. lupulina*, or *M. sativa*. The pea aphid (*Acyrthosiphon pisum*) transmitted PSV in a nonpersistent manner.

Chickpea (*Cicer arietinum* L.), a relatively new crop in the dryland areas of the Palouse region of eastern Washington and northern Idaho, is grown in rotation with wheat, barley, pea, and lentil. The USDA chickpea germ plasm collection of 3,806 Plant Introduction (PI) accessions is maintained by the Western Regional Plant Introduction Station in Pullman, Washington. Periodically, chickpea PI lines must be propagated when germination decreases or when seed supplies need to be replenished. Propagation is usually done at an isolated research station situated along the Snake River near Central Ferry, Washington.

In 1979, the first year chickpea germ plasm was grown at Central Ferry, several viruses were isolated from plants exhibiting symptoms of stunting, wilting, yellowing, leaf deformation, mosaic, or phloem discoloration. These included alfalfa mosaic virus (AMV), bean leaf roll luteovirus (BLRV), bean yellow mosaic potyvirus (BYMV), pea enation mosaic virus (PEMV), and pea streak carlavirus (PSV) (9).

Pea streak virus (2) is the only carlavirus known to be a chickpea pathogen. Zaumeyer and Patino (13) included chickpea in greenhouse inoculation studies with a pea isolate of PSV from southern Idaho. They observed necrosis of leaflets and wilting of terminal shoots, which are similar to the symptoms exhibited by chickpea infected by PSV under field conditions in the Palouse region.

The objectives of this study were to determine the symptomatology and the experimental and natural host ranges of PSV isolated from chickpeas and other legumes in the Palouse region, to characterize vector and seed transmission of the virus in chickpea and other hosts, and to measure the effects of virus infection on seed yield and quality of chickpea. A preliminary report has been published (9).

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MATERIALS AND METHODS

Isolation studies. When wilting and yellowing chickpeas were initially collected at Central Ferry in 1979, the symptoms were reminiscent of Fusarium wilt caused by *Fusarium oxysporum* Schlechtend.:Fr. f. sp. *ciceris* (Padwick) Matuo & K. Sato. Stem pieces from wilting plants were surface-disinfested in 0.5% NaOCl for 5 min, and small pieces of tissue were plated on 2% water agar and potato-dextrose agar.

Tissue from the diseased chickpeas also was triturated in 0.06 M K_2HPO_4, pH 7.0, with 0.22-μm (600-mesh) Carborundum added. The triturate was applied to the leaves of *Chenopodium quinoa* Willd., chickpea, and faba bean (*Vicia faba* L.).

Source of virus. Isolates of PSV used in this study were from chickpea from Central Ferry (PSV-CF) and Walla Walla, Washington (PSV-WW); alfalfa (*Medicago sativa* L.) from Pullman, Washington (PSV-A3); black medic (*M. lupulina* L.) from Pullman (PSV-ML), faba bean from Central Ferry (PSV-VF); white sweet clover (*Melilotus alba* Medik.) from Central Ferry (PSV-MA); and pea from Wisconsin (PSV-PV 87) from the American Type Culture Collection (1). Isolates of PSV selected after serial single-lesion transfer on *Senna occidentalis* (L.) H. Irwin & Barneby (= *Cassia occidentalis* L.) were maintained in *V. faba* cv. Herz Freya, *Melilotus alba*, and/or *M. sativa*. In host range studies and resistance tests with different *Cicer* spp., inoculum from PSV-infected plants was triturated in 0.06 M K_2HPO_4, pH 7.0, to which was added a small amount of 0.22-μm Carborundum. The triturate was rubbed onto the leaves of test plants and immediately rinsed off with tap water. After 2–4 wk of incubation, plants were assayed for systemic infection by manual inoculation to healthy Herz Freya faba bean and chickpea PI 458870. At times, plants were assayed by indirect enzyme-linked immunosorbent assay (ELISA) using PSV antiserum supplied by R. O. Hampton, USDA-ARS, Corvallis, Oregon.

Field studies. Beginning in May 1981, different annual and perennial plant species growing near the Snake River at Central Ferry were indexed for PSV on Bountiful bean (*Phaseolus vulgaris* L.), California Blackeye cowpea (*Vigna unguiculata* (L.) Walp. subsp. *unguiculata*), chickpea PI 458870, and *C. quinoa*. Most of the wild annual species had started to flower by the time they were indexed for PSV. In addition to host range studies and symptom expression,

serological tests were used to identify PSV and other viruses isolated from plants in the field trials.

The incidence of PSV infection in *M. lupulina, M. sativa*, and *Melilotus alba* collected at random was determined at different locations in the Palouse region. Tissue samples collected at periodic intervals from 1979 to 1990 were indexed on Herz Freya faba bean, chickpea PI 458870, and, at times, on *S. occidentalis* and/or tested by indirect ELISA.

Seeds of chickpea PI 315787, PI 451432, and PI 458870 were planted in single rows 3.0 m long with 1.5 m between rows in a randomized complete block design with six replicates of 50 seeds per row per replicate. Inoculum was increased in faba bean. Plots were manually inoculated with PSV-CF at the prebloom (31 days after planting) and full bloom (64 days after planting) stages of plant growth. Plants were inoculated by the procedure outlined earlier. In each untreated and treated plot, healthy and infected plants, respectively, were tagged 2–3 wk after inoculation, and mortality counts were made after 80 days. Plots were harvested 98–106 days after planting. Seed yields were determined from 20 tagged plants in each plot.

Seed transmission. Seeds of PSV-infected chickpea, alfalfa, *M. lupulina*, and lentil (*Lens culinaris* Medik.) were sown in moist vermiculite, and one to five germinating seeds were transferred to pasteurized potting medium (55% peat moss, 35% pumice, 10% sand) in 15- or 25-cm-diameter plastic pots. Then, 3–6 wk after transplanting, plants were indexed for PSV on Herz Freya faba bean, chickpea PI 458870, and *S. occidentalis* in groups of one to five.

Vector transmission. Insect transmission studies were carried out with the pea aphid (*Acyrthosiphon pisum* (Harris)). Nonviruliferous pea aphid colonies were maintained on healthy Herz Freya faba bean. Aphids were starved about 2 hr on moist filter paper in petri dishes and then given acquisition feeding periods of 1–15 min on faba bean infected with isolate PSV-CF. After feeding, aphids were transferred in groups of five to healthy chickpea and faba bean (test plant) seedlings and kept in aphid-proof cages for up to 4 hr. The test plants were then sprayed with an aphicide and held for 3–4 wk in the greenhouse for symptom development.

Serology. ELISA was used to detect PSV in some plants. Plant samples were triturated at approximately 1:10 (w/v) in phosphate-buffered saline (0.07 M phosphate, pH 7.4, 0.15 M NaCl) to which had been added 0.5% Tween 20, 2% polyvinylpyrrolidone, and 0.2% ovalbumin. Extracts were pipetted into microtiter plates and incubated 1 hr at room temperature. Incubation of anti-PSV antiserum and antirabbit IgG:alkaline phosphatase conjugate, both diluted 1:3,000 in the sample buffer described above, was 1 hr at room temperature and 37 C, respectively. The substrate used was *p*-nitrophenyl phosphate, and the plates were read on an ELISA reader at 410 nm when color development in the positive control was approximately one absorbance unit. Healthy and virus-infected chickpea also were tested against antisera to AMV, BLRV, BYMV, and PEMV, which also infect chickpea naturally in the Palouse region.

Electron microscopy. Extracts of faba bean and alfalfa infected with PSV isolates CF, A3, and PV 87 and purified virus suspensions of isolates A3 and PV 87 were diluted in distilled water. A carbon-backed, Formvar-coated grid was placed on a drop of the crude extract or purified viral suspension for 5–10 min, then rinsed with distilled water and negatively stained with a 2% aqueous solution of uranyl acetate. The virus preparations were examined under a Zeiss EM9-S2 (crude extracts) or an Hitachi H-600 (purified virus) transmission electron microscope.

RESULTS

Incidence and symptoms of disease. Symptoms on chickpea infected by PSV-CF include yellowing of the foliage, wilting of the terminal shoots (Fig. 1), and intense discoloration of phloem tissues. Many chickpea plants infected in the seedling stage wilted and died before setting pods. Seed harvested from surviving plants were small, discolored, and misshapen and germinated at low rates.

Pea streak virus was isolated from commercial chickpea plantings and germ plasm trials in different areas of the Palouse region. Infection varied greatly from year to year and location to location and usually ranged from 0.5 to 5%. However, the incidence of PSV-infected chickpea appeared to be higher when fields were planted near alfalfa.

Isolations on agar media from stems of yellowing and wilting chickpea did not yield any pathogenic fungi or bacteria, and PSV was the only virus consistently isolated from these diseased plants. In isolations and back-inoculations from naturally infected and manually inoculated chickpea, respectively, PSV was the only pathogen isolated.

Host range studies. All PSV isolates from the Palouse produced similar symptoms on the different plant species used in the host range study (Table 1). The virus (PSV-CF) induced systemic symptoms in several legumes, including chickpea, faba bean, fenugreek (*Trigonella foenum-graecum* L.), lentil, *M. lupulina*, and pea (*Pisum sativum* L.), while *M. sativa* and *Vicia villosa* Roth subsp. *villosa* were symptomless carriers of PSV. *Melilotus alba* was a symptomless host for all PSV isolates except PSV-WW, which induced symptoms of stunting, mosaic, and leaf deformation. The virus produced local lesions without systemic spread in *Gomphrena globosa* L., *Senna obtusifolia* (L.) H. Irwin & Barneby, and *S. occidentalis* (Fig. 2). No symptoms developed on, and no virus was isolated from, *Chenopodium amaranticolor* Coste & Reyn, *C. guinoa, Cucumis sativus* L., *Nicotiana glutinosa* L., *N. tabacum* L., *P. vulgaris*, and *V. unguiculata*.

Isolate PSV-PV 87 produced symptoms indistinguishable from those of PSV-CF on all plant species inoculated except *C. amaranticolor* and *C. quinoa*; on these two hosts, PSV-PV 87 induced chlorotic local lesions. Isolate PSV-WW was more virulent on all systemically infected hosts than the other Palouse isolates.

More than 50 cultivated chickpea PI accessions tested were susceptible to PSV

Fig. 1. (Left) Wilting and yellowing of chickpea PI 458870 inoculated with pea streak virus isolate CF from chickpea; (right) healthy plant.

isolates CF and A3. Additionally, systemic symptoms were incited in the following wild annual *Cicer* spp.: *C. bijugum* K.H. Rech. (PI 458550 and PI 458551), *C. chorassanicum* (Bge) M. Popov (PI 458553), *C. cuneatum* Hochst. ex Rich. (PI 458554), *C. echinospermum* P.H. Davis (PI 489776 and PI 527930), *C. judaicum* Boiss. (PI 458558, PI 458559, and PI 510659), *C. pinnatifidum* Jaub. & Spach (PI 458555 and PI 458556), *C. reticulatum* Ladiz. (PI 489777 and PI 510655), and *C. yamashitae* Kitamura.

Several wild perennial *Cicer* spp. were resistant or immune to PSV. These included *C. anatolicum* Alef. (PI 383626), *C. canariense* Santos Guerra & G.P. Lewis (PI 557453), *C. microphyllum* Benth. (PI 532928), and *C. oxyodon* Boiss & Hoh. (PI 561103).

Field studies. Forty-one different annual and perennial plant species were indexed for virus infection at Central Ferry in 1981, but PSV was isolated from only two of 33 *Melilotus alba* plants and one of 14 *M. lupulina* plants. The virus was not isolated from the following species (number after each species indicates number of plants indexed): *Achillea millefolium* L., 7; *Alnus* sp., 3; *Amaranthus albus* L., 23; *A. retroflexus* L., 23; *Amsinckia* sp., 2; *Artemisia absinthium* L., 4; *Chenopodium album* L., 21; *C. chenopodioides* (L.) Aellen, 3; *Cichorium intybus* L., 7; *Cirsium arvense* (L.) Scop., 7; *C. vulgare* (Savi) Ten., 6; *Clematis ligusticifolia* Nutt., 3; *Dipsacus sylvestris* Huds., 5; *Erigeron* sp., 2; *Erodium cicutarium* (L.) L'Hér. ex Aiton, 5; *Euphorbia* sp., 9; *E. maculata* L., 6; *Helianthus* sp., 8; *Hypericum perforatum* L., 5; *Kochia scoparia* (L.) Schrader, 6; *Lactuca serriola* L., 4; *Lepidium perfoliatum* L., 9; *Ligustrum vulgare* L., 1; *Melilotus officinalis* (L.) Lam., 6; *Mentha arvensis* L., 5; *Monarda fistulosa* L., 6; *Nepeta cataria* L., 4; *Oenothera biennis* L., 2; *Polygonum* sp., 5; *Rosa* sp., 6; *Rumex crispus* L., 6; *Salix* sp., 6; *Salsola kali* L., 2; *Sisymbrium altissimum* L., 2; *Solanum nigrum* L., 10; *S. sarrachoides* Sendtner, 6; *Tetradymia* sp., 1; *Tragopogon pratensis* L., 7; and *Tribulus terrestris* L., 12.

From 1979 to 1990, surveys were made of *M. lupulina, M. sativa*, and *Melilotus alba* in different areas of the Palouse region for infection by PSV (Table 2). The virus was widely distributed in eastern Washington and northern Idaho, where it was isolated from all three hosts in most years. Isolates of PSV were obtained from *M. sativa* from 15 of 16 sites, and incidence ranged from 0 to 44% in plants tested. *Melilotus alba* was also frequently infected with PSV; at some locations, incidence exceeded 30% of plants tested. Infection of *M. lupulina* by PSV ranged from 0 to 33% of plants tested.

Field inoculations of the three chickpea PI accessions at prebloom and full bloom adversely affected yields and increased mortality (Table 3). Yields were reduced 97–99% in plants inoculated at prebloom and 16–50% in plants inoculated at full bloom. Mortality ranged from 90–98% and 10–45% following inoculations at prebloom and full bloom, respectively. Seed harvested from plants inoculated at prebloom and full bloom were usually smaller and often discolored and misshapen.

Seed transmission. No transmission of PSV was detected in 46, 62, 149, and 270 seedlings of chickpea, *M. lupulina, M. sativa* (local cultivar), and Benewah lentil (PI 564719), respectively. The percentage of small, misshapen, and discolored seeds of chickpea harvested from plants infected with PSV before pod set was 75–100%, and 15–40% of these seeds germinated.

Vector transmission. *A. pisum* transmitted PSV from infected faba bean to chickpea and faba bean in a nonpersistent manner. Transmission efficiencies (percentage of inoculated plants that became infected) of groups of five aphids were 5–10% to chickpea but 50–70% to faba bean. Symptoms in chickpea and faba bean inoculated by aphid transmission were identical to those on plants inoculated manually.

Serology. Indirect ELISA detected all isolates of PSV used in this study and detected PSV in different naturally infected and mechanically inoculated plant species. None of the PSV isolates reacted with antisera to AMV, BLRV, BYMV, or PEMV.

Electron microscopy. Long, semiflexuous rods typical of the carlavirus group (11) were observed in crude extracts of faba bean and alfalfa infected with three isolates of PSV and in purified viral suspensions of two PSV isolates. The virus particles in crude extracts and purified viral suspensions had modal lengths of 630–640 nm.

DISCUSSION

Pea streak carlavirus is one of 16 viruses that infect chickpea worldwide (8), and it is the only carlavirus presently known to be a chickpea pathogen. The virus is found only in North America, where it infects several plant species in the Fabaceae (7,11), but it has been reported affecting chickpea only in the Pacific Northwest (9), although the crop also has been grown commercially in

Table 1. Host range and symptoms of pea streak virus isolate CF from chickpea

Test species	PI line or cultivar	Host reaction[z]
Chenopodium amaranticolor	...	NS
C. quinoa	...	NS
Cicer arietinum	PI 273879	Y, W, PD
	PI 458870	Y, W, PD
Cucumis sativus	Ohio MR-17	NS
Glycine max	Bragg	NS
Gomphrena globosa	...	NLL
Lens culinaris	PI 477920	St, Y, W
Medicago lupulina	...	St, LD, M
M. sativa	Hairy Peruvian	SI
Melilotus alba	...	SI
Nicotiana glutinosa	...	NS
N. tabacum	White Burley	NS
Phaseolus vulgaris	Bountiful	NS
Pisum sativum	Alaska	M, SN, St
	Dark Skin Perfection	M, SN, St
Senna occidentalis	...	NLL
S. obtusifolia	...	NLL
Trigonella foenum-graecum	PI 515953	St, LD, M
Vicia faba	Herz Freya	NLL, SN, St
V. villosa subsp. *villosa*	...	SI
Vigna unguiculata subsp. *unguiculata*	California Blackeye	NS

[z] Test plants were back-inoculated to healthy *Vicia faba* cv. Herz Freya. LD = leaf deformation, NLL = necrotic local lesions, NS = not susceptible, SI = symptomless systemic infection, SN = systemic necrosis, St = stunting, M = mosaic, W = wilting, Y = yellowing, PD = phloem discoloration.

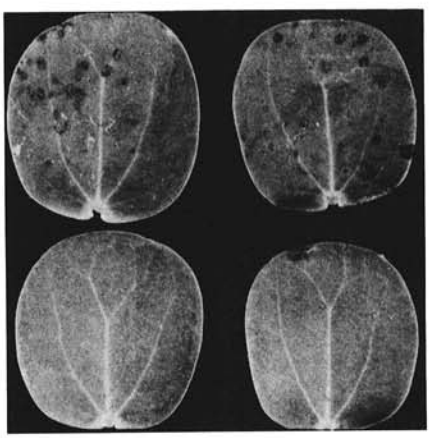

Fig. 2. (Top row) Necrotic local lesions on *Senna occidentalis* (= *Cassia occidentalis*) inoculated with pea streak virus isolates from (left) chickpea and (right) ATCC PV-87. (Bottom row) Leaves from healthy plants.

California for many years. In addition to chickpea, PSV is a pathogen of two other commercially important food legumes in the Palouse region, lentil (6) and pea (3). The incidence of PSV and other viruses infectious to chickpea tends to increase following a mild winter, which apparently favors aphid survival, an early buildup of pea aphid populations, and subsequent spring flights from virus-infected alfalfa to different food legumes (3). These conditions occurred in 1983 (10) and 1990 (12), when several viruses, including PSV, devastated chickpea, lentil, and pea plantings in the region.

In chickpea, PSV induces wilting and yellowing symptoms that may be confused with symptoms of Fusarium wilt. Therefore, it is advisable to use various criteria in addition to symptomatology to identify PSV and other viruses, such as some strains of AMV and BYMV (6,7), that may cause chickpea plants to wilt and die. In this study, we used host range, serology, and electron microscopy to identify PSV as the cause of the wilting and yellowing disease of chickpea in the Palouse region. In young chickpea plantings, PSV can drastically reduce seed yields and cause death of a high percentage of infected plants. Because of the effects of PSV infection on seed quality, germination and vigor of chickpea seedlings may also be adversely affected.

Since 1979, more than 25 isolates of PSV from different hosts in the Palouse region have been studied. All isolates caused wilting and yellowing in all commercial chickpea cultivars presently grown in the Pacific Northwest. All the isolates were similarly virulent except PSV-WW, which was more virulent on several systemically infected hosts, including chickpea, faba bean, lentil, pea, and *Melilotus alba*. No resistance to local isolates of PSV has been observed in 55 chickpea PI accessions from the Pullman germ plasm collection. Additionally, no resistance was observed in PI accessions of eight wild annual *Cicer* spp. However, resistance was observed in PI accessions of several wild perennial *Cicer* spp., including *C. anatolicum, C. canariense, C. microphyllum*, and *C. oxyodon*. Several of these perennial *Cicer* spp. also have resistance to Ascochyta blight caused by *Ascochyta rabiei* (Pass.) Labrousse and to several abiotic stresses (W. J. Kaiser, *unpublished*). If PSV becomes a constraint to chickpea cultivation in the Pacific Northwest, additional greenhouse and field screening trials will be required to locate suitable sources and levels of resistance. Additional breeding and genetic engineering research may also be required to transfer PSV resistance from wild perennial *Cicer* spp. to commercially desirable chickpea cultivars.

We conclude that alfalfa was the most important and widely distributed reservoir of PSV in the Palouse region from 1979 to 1990. Of the alfalfa plantings sampled, 94% contained PSV, and virus incidence reached 44% in some fields. We conclude that black medic, an annual, and white sweet clover, a biennial, were of lesser importance as reservoirs of PSV because of their restricted and scattered distribution. Our conclusions agree with those of Hampton and Weber (4,5) that alfalfa is the primary reservoir of PSV in the Pacific Northwest. Alfalfa plantings vary greatly in size and are widely distributed in the Palouse region. Although we did not collect data on the proximity of chickpea and alfalfa fields, we observed that the incidence of PSV in chickpea appeared to be higher when alfalfa fields were nearby. Presently, chickpea is grown on approximately 3,000 ha in eastern Washington and northern Idaho, but production is expected to expand substantially in 1994 when growers will be able to plant newly released cultivars resistant to Ascochyta blight. It is likely that the incidence of

Table 2. Incidence of pea streak virus in three legumes in the Palouse region of eastern Washington and northern Idaho[y]

Plant species	Year	Location	Plants tested (no.)	Plants infected	
				No.	%
Medicago lupulina	1979	Pullman, WA	3	1	33.3
	1980	Pullman	11	0	0
	1981	Central Ferry, WA	14	1	7.1
	1984	Pullman	6	2	33.3
Medicago sativa	1979	Pullman	9	4	44.4
	1980	Pullman	11	2	18.2
	1984	Pullman	32	12	37.5
	1985	Central Ferry	14	2	14.3
	1988	Colfax, WA	40	2	5
	1988	Central Ferry I[z]	79	18	22.8
	1988	Central Ferry II	40	13	32.5
	1988	Central Ferry III	40	16	40
	1988	Central Ferry IV	40	5	12.5
	1988	Central Ferry V	40	15	37.5
	1988	Central Ferry VI	40	11	27.5
	1988	Dusty, WA	40	4	10
	1988	Genesee, ID	40	2	5
	1988	Pullman I	40	9	22.5
	1988	Pullman II	25	10	40
	1988	Spangle, WA	40	0	0
Melilotus alba	1979	Central Ferry	15	2	13.3
	1981	Central Ferry	33	2	6.1
	1982	Central Ferry	18	2	11.1
	1982	Wawawai, WA	9	1	11.1
	1984	Central Ferry	41	3	7.3
	1984	Lewiston, ID	6	2	33.3
	1984	Pullman	6	0	0
	1987	Central Ferry	18	3	16.7
	1988	Central Ferry	19	0	0
	1990	Central Ferry	40	0	0

[y] Virus was detected by indirect ELISA and/or by indexing on *Vicia faba*, *Cicer arietinum*, and, at times, *Senna occidentalis*.
[z] Central Ferry sites I–VI were located within 0.5–10 km of the Central Ferry Research Station.

Table 3. Effect of infection by pea streak virus isolate CF from chickpea on seed yield and mortality of three chickpea Plant Introduction lines inoculated at two stages of growth at Central Ferry, Washington

Line, origin	Growth stage at inoculation[x]	Mean seed yield per plot[y] (g)	Percent mortality
PI 315787, India	Untreated	738.2 a[z]	0
	Full bloom	429.3 b	35
	Prebloom	0.3 c	98
PI 451432, Iran	Untreated	668.3 a	0
	Full bloom	331.7 b	45
	Prebloom	0.2 c	99
PI 458870, United States	Untreated	716.2 a	0
	Full bloom	600.8 b	10
	Prebloom	21.8 c	90

[x] Inoculations at prebloom and full bloom were done 31 and 64 days after planting, respectively, and plots were harvested 98–106 days after planting.
[y] Based on seed harvested from 20 chickpea plants in each of six 3-m single-row plots.
[z] Numbers for each PI accession followed by the same letter do not differ significantly ($P = 0.05$) according to Duncan's new multiple range test.

PSV in chickpea will increase as chickpea cultivation expands and chickpea fields are located closer to PSV-infected alfalfa.

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