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Pea Leaf Roll in Northwestern U.S. Pea Seed

Pea leaf roll (PeLR) was first attributed to a virus by Quantz and Volk of West Germany in 1954 (11). The same virus was subsequently termed "topvergelingsvirus vande erwt" (pea top yellows virus) in the Netherlands, and the disease it induced was called "jaunisse apicale du pois" (pea apical yellowing) in France. Bos (2), in 1964, suggested that "to avoid further confusion it might be wise to give preference to the name 'pea leaf roll virus,' especially for the sake of priority."

Before 1980 the pea leaf roll virus (PeLRV) had never been reported in peas in the United States, although Thottappilly et al (14) and Duffus (6) reported PeLRV-like viruses in alfalfa. In 1980 a major pea disease epidemic causing severe crop losses occurred in southern Idaho, where more than 80% of the U.S. pea seed crop is produced annually. The disease, recurring much less destructively in 1981 and 1982, was characterized in many susceptible cultivars by basipetal chlorosis ("yellows" symptoms progressing from the apex of the plant downward) and by a lack of infection gradients across affected fields, unlike diseases frequently produced in that area by pea streak and alfalfa mosaic viruses (7). These disease characteristics closely matched those of pea leaf roll described in the Netherlands (3,8).

Pea leaf roll is not seed-transmissible. Instead, it perennates in overwintering plants, such as forage legumes, and is persistently transmitted by aphids.

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The Southern Idaho Isolate

The southern Idaho isolate of PeLRV has been identified and partially

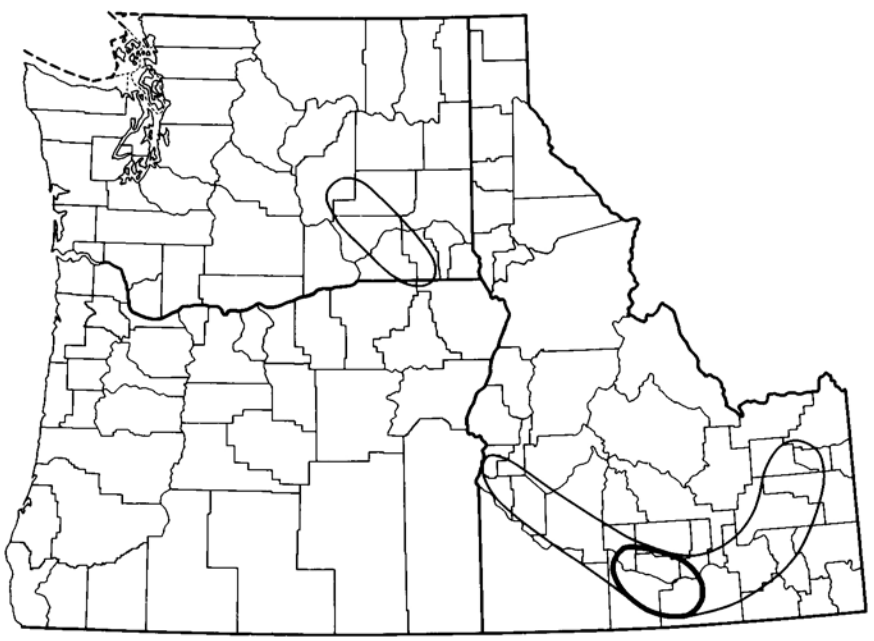

Fig. 1. County-delineated map of Idaho, Washington, and Oregon, showing the area in which PeLRV was epidemic in peas in 1980 (small bold-faced oval) and larger area in which PeLR was generally less destructive to peas during 1981-1982 (thin-lined zone). Most of the U.S. pea seed crop is produced within the larger zone.

Production Areas

characterized (*unpublished*). In essence, the virus was isolated by feeding laboratory-reared pea aphids (*Acyrthosiphon pisum*) on naturally infected pea and alfalfa plants (48-hour acquisition access), transferring them to healthy "intermediate" plants for 17 hours to facilitate discharge of stylet-borne viruses, and finally transferring them to plants of PeLR-sensitive pea cultivars for a 48-hour transmission access. Test plants thus inoculated in successive tests developed yellows symptoms 20–28 days after exposure to aphids. During successive passages of the virus by aphids, the infected plants were determined by enzyme-linked immunosorbent assay (ELISA) to be free from pea streak, alfalfa mosaic, and red clover vein mosaic viruses, and the yellows-inducing virus was mechanically nontransmissible. Subsequent isolates were obtained from symptomless alfalfa plants transplanted into the laboratory from fields adjacent to severely PeLR-affected southern Idaho pea fields. Preparations of spherical viruslike particles, partially purified from laboratory-inoculated pea plants by use of cellulose-digesting enzyme, reacted equally by ELISA to immuno-γ-globulin from antiserum produced by Ashby and Huttinga (1) against PeLRV and from antiserum produced by Duffus (6) against legume yellows virus.

Notwithstanding current flux in luteovirus relationships and terminology (12), recent evidence (9) suggests that the causal agent of the southern Idaho pea disease and those described by Thottappilly et al (14) and by Duffus (6) are isolates or strains of the same virus. All are regarded by me as PeLRV, and I believe that PeLRV may have existed in the U.S. agroecosystem, perhaps principally in alfalfa, for many years. Records in my laboratory, including color photographs of diseased pea plants, indicate that plants with PeLR-like symptoms occurred in the Pacific Northwest several years prior to 1980. Likewise, an extremely high incidence of PeLRV in alfalfa plantings assayed in 1981 suggests an enduring relationship between alfalfa and PeLRV. Alfalfa was also the source of isolates reported by

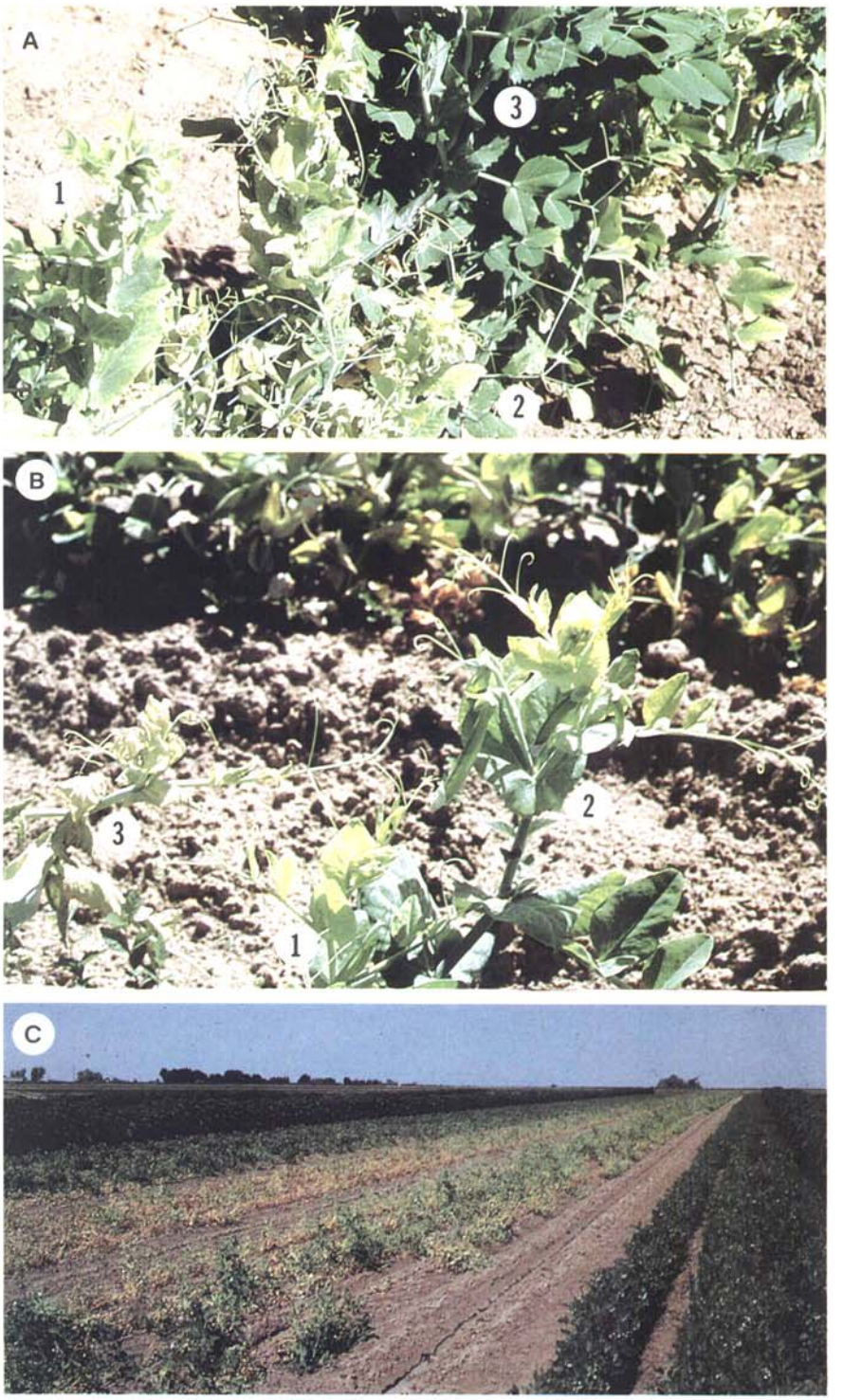

Fig. 2. Symptoms induced by the pea leaf roll virus: (A) Infected pea plants with (1) whole-plant partial chlorosis and (2) severe stunting with rosetting, compared with (3) much larger, healthy plants with normal coloration. (B) Infected plants with "top yellows" symptoms characteristic of some pea cultivars; (1) initial stage of chlorosis, (2) intermediate stage with the beginning of leaf necrosis, and (3) advanced stage with necrotic collapse of leaf and stem tissues. (C) Range of PeLRV and disease response among commercial breeding lines in 1980, showing (left to right) susceptibility with moderate tolerance, susceptibility with extreme sensitivity, susceptibility with trace tolerance, and extreme PeLR tolerance and PeLRV resistance. Numerous breeding lines and cultivars discovered in 1980 to be PeLRV-resistant have since been actively utilized by commercial and institutional breeders. Segregation ratios of F_2 progenies from crosses of resistant and susceptible parents suggest that resistance is conferred by a single dominant gene, tentatively referred to as *Plr*.

Table 1. ELISA determinations of viruses in field samples of pea plants (1980) that showed leaf roll symptoms or were symptomless

Symptoms	Viruses detected[a]					
	PeLRV alone	PeLRV + PSV	PeLRV + RCVMV	PeLRV + AMV	PeLRV + PSV + RCVMV	No viruses detected
Pea leaf roll	9/58	31/52[b]	6/52	2/52	4/52[c]	6/58
None[d]	2/18	1/3	0/3	0/3	0/3	15/18

[a] PeLRV = pea leaf roll virus, PSV = pea streak virus, RCVMV = red clover vein mosaic virus, AMV = alfalfa mosaic virus.
[b] Thirty-one of 52 plants (60%) containing PeLRV were coinfected with PSV.
[c] Four of the six plants containing RCVMV and PeLRV also contained PSV.
[d] When possible, symptomless plants were pair-sampled with those showing pea leaf roll symptoms.

Thottappilly et al (14) and Duffus (6). Further, during visits to pea breeding nurseries in the Pacific Northwest in the 1960s, N. Hubbeling (*personal conversation*) observed that occasional plants of some pea lines and cultivars showed symptoms typical of "top yellows" in the Netherlands and concluded that the causal virus (PeLRV) was indigenous to the area.

Known Distribution of PeLR in Peas, 1980–1982

Ecological factors that were only partially defined triggered an unprecedented PeLR epidemic in peas in southern Idaho in 1980. The geographic area within which the causal PeLRV was detected in peas that year and where the disease virtually eliminated plantings of susceptible pea cultivars is shown in Figure 1. Less destructive recurrence of PeLR over a larger area was monitored by ELISA of pea plant samples during 1981 and 1982. A severe epidemic over this larger area before PeLR-tolerant or immune cultivars become predominant could obviously threaten peas as seed and food crops. U.S.-produced pea seeds are generally regarded in the world seed trade as an exceptionally premium-quality product.

PeLR Symptomatology in Peas

Disease and symptom development during May–June 1980–1982 in fields and nurseries of diverse pea cultivars and lines were expressed in a variety of ways. The plant-response terminology of Cooper and Jones (4) is herein endorsed and applied. The PeLR-sensitivity of these cultivars and lines interacted noticeably with the time of PeLRV infection. Cultivars now known from greenhouse studies to be moderately PeLR-sensitive were killed outright when infected by PeLRV in the five- to eight-node stage but survived to produce seed when infected during the 10- to 14-node stage. Extremely PeLR-sensitive selections or cultivars were killed in 1980 regardless of the time infected. Certain pea lines were severely stunted when infected early but were stunted less and developed whole-plant partial chlorosis when infected at later stages (Fig. 2A), either surviving poorly or collapsing 6–10 days after symptom onset. Many plants with typical yellows symptoms (Fig. 2B) contained PeLRV as well as pea streak virus, possibly both transmitted by the same aphid. Coincidence of PeLRV with other viruses in plants tested by multiple ELISA is shown in Table 1. The high frequency of coinfection by PeLRV and pea streak virus necessitated initial separation of the two viruses and separate cultivar screening to establish that PeLRV had been the principal pathogen in 1980 and had incited the observed field symptoms in 36 selected pea cultivars.

Every major U.S. pea seed company, all holding genetically diverse *Pisum* materials, possessed cultivars or advanced breeding lines that were PeLR-tolerant and/or PeLRV-resistant (Fig. 2C).

PeLRV in Alfalfa

Coincident with the 1980 PeLR epidemic in peas was apical chlorosis of alfalfa plants in nearby fields. Examination revealed exact counterparts of PeLR symptoms in peas: chlorotic terminals on otherwise normally green plants, interveinal and marginal leaf chlorosis, and chlorotic necrosis of terminal leaves resulting in "white flags" on severely affected plants (Fig. 3). Comparable symptoms have been observed in alfalfa for many years, particularly in fields irrigated after the first cutting for hay. Since this usually occurs in June, the condition has been colloquially termed June yellows. Apical chlorosis is usually enhanced when the first irrigation after cutting coincides with cool, ie, less than 15 C (60 F), soil temperatures.

In preliminary tests by aphid transmission and subsequently by ELISA, PeLRV was detected in 30 of 31 alfalfa plants transplanted from the 1980 epidemic area into greenhouses. Conversely, tissue samples from 19 alfalfa plants from western Oregon or western Montana, outside the PeLR epidemic area, contained no ELISA-detectable PeLRV. An Idaho alfalfa planting where about half the plants were infected with PeLRV was sampled for ELISA in 1982 under conditions favoring development of apical chlorosis. ELISA results showed that the proportion of normally green plants containing PeLRV equaled that of plants with apical chlorosis. Likewise, numerous plants with various degrees of apical chlorosis contained no detectable PeLRV. Thus, PeLRV infection was not correlated with June yellows.

The perfect simulation of PeLR-like symptoms (in this case the condition is theorized by some soil scientists to result from oxygen tension in a cold, water-soaked root zone) cautions against diagnosing "yellows diseases" by symptomatology alone. At the same time, induction of almost identical yellows symptoms by such contrasting stress-inducing factors invites inquiry into the nature of luteovirus effects on host plants.

PeLR Disease Cycle

Several ecological factors interact to produce PeLR epidemics in peas. Some have yielded to experimental inquiry and others are not yet understood. Factors integrated by current concepts are presented as a PeLR disease cycle in Figure 4. Alfalfa, as the perennial PeLRV inoculum reservoir, plays the central role in the cycle. The pea aphid (*A. pisum*) and other aphid species commonly overwinter on alfalfa. Winter severity determines the form in which *A. pisum* survives and influences the earliness of population increase. At temperatures promoting terminal growth of alfalfa plants, the ELISA-measurable concentration of PeLRV gradually increases to a maximum level in the spring and decreases with the onset of daytime temperatures above 30 C (86 F). PeLRV is sometimes not ELISA-detectable in infected alfalfa plants during July through September. Rising spring temperatures also increase the reproductive rate of *A. pisum*.

Development of winged *A. pisum* may depend on several factors, including temperature and colony crowding (10). The number of winged aphids before the first alfalfa cutting for hay may be the most critical factor determining PeLRV spread to peas. Aphid numbers before the second or subsequent alfalfa cuttings may be less significant, since normally high temperatures would have depressed the PeLRV concentration in alfalfa plants by that time and PeLRV infections in pea plants approaching maturity would cause relatively less crop loss. Short-cycled spread from infected peas back to young alfalfa plantings could increase the PeLRV inoculum reservoir and intensify the disease cycle. Field spread of PeLRV in 1980 appeared to have resulted from

aphid migration flights over a period of 3–4 weeks in May and June during, and perhaps slightly before, alfalfa cutting. Migratory aphid flights triggered by population pressures or the first alfalfa cutting presumably would have accounted for most of the PeLRV spread during that season.

Relatively high concentrations of PeLRV in peas, 10- to 100-fold higher than in alfalfa, could greatly facilitate secondary aphid spread of PeLRV from peas under conditions favoring vector activity. PeLRV-infected plants were detected in limited plantings of lentil and chickpea grown near infected alfalfa fields. Infected lentil plants were sometimes symptomless but typically were stunted and chlorotic, in some cases showing apical reddening. Infected chickpea plants were stunted and severely chlorotic or dead. Broadbean (*Vicia faba*) (14) and at least two common clover species (*Trifolium incarnatum* and *T. subterraneum*) (6) are hosts of PeLRV.

The harvest of annual crops and drying of weed species colonized by *A. pisum* are followed by fall regrowth of alfalfa after the final cutting. Fall migratory flights of *A. pisum* to alfalfa complete the cycle.

Temperature-Year Patterns Relative to PeLR Epidemiology

Ambient temperature is among the most significant exogenous factors affecting aphid reproduction rates and production of winged forms. Air temperatures in alfalfa fields therefore could be expected to significantly influence the size and status of natural pea aphid populations. The ambient temperature patterns during 1971–1982 that could have influenced aphid survival, population size, and, particularly, proportion of winged forms to disseminate PeLRV at the first cutting of alfalfa are shown in Table 2. No marked yearly deviations from mean temperature values were obviously associated with the onset of the PeLR epidemic in peas. Although the number of days with temperatures above 70 F (21 C) during October–December 1979 (favoring fall increases in aphid populations and thus potentially large overwintered populations) exceeded the mean by 100%, the following January–March temperature minima (limiting winter survival) were lower than normal. A similar excess in days with temperatures above 70 F (21 C) occurred in the fall of 1980; yet the incidence of PeLR in 1981 was dramatically less than in 1980.

Customary climatological data highly useful to agriculture and other enterprises may therefore be inadequate for understanding aphid behavior associated with pea leaf roll outbreaks. The lack of obvious interconnections between anticipated temperature effects on aphid biology and pea aphid transmission of PeLRV suggests complex, perhaps subtle interactions. For instance, all environmental factors affecting alfalfa plant physiology would certainly influence both PeLRV synthesis and aphid biology and population development. Determination of definitive effects on PeLRV dissemination therefore may require complex analyses of all conceivable climatological and edaphic variables.

The Outlook for PeLR

The pathological and ecological mechanisms involved in the establishment of PeLRV in alfalfa stands, particularly in southern Idaho, are unknown. Having been established, however, PeLRV can be expected to persist and expand into new and surrounding alfalfa production areas. Although factors triggering PeLR epidemics are not yet understood, those favoring large aphid populations in alfalfa can be assumed to promote the likelihood of PeLR epidemics. The active participation of research personnel of the major pea seed companies in developing PeLR-tolerant and/or PeLRV-resistant pea cultivars assures progress toward the ultimate control of PeLR in peas. Several other major pea disease problems of this century have been effectively resolved through cooperative efforts of U.S. Department of Agriculture and state agricultural experiment station scientists

Table 2. Temperature (F) patterns that could have influenced aphid winter survival and seasonal population levels[a]

Year	Jan.–Mar. min (survival)[d]	Feb.–May mean max[b] (increase)[d]	May–June max (decrease)[d]	Oct.–Dec. mean max[c] (increase)[d]	No. days >70 F (>21 C) Oct.–Dec. (increase)[d]	Oct.–Dec. min (survival)[d]
1971	0	42–67	83–97	58–34	8	−1
1972	−9	40–69	87–91	60–32	6	−19
1973	1	41–71	87–93	64–41	10	12
1974	−11	42–66	82–100	63–38	9	6
1975	−4	37–62	85–84	61–42	9	11
1976	7	38–71	85–96	63–44	6	2
1977	−7	48–61	77–94	66–42	8	13
1978	8	42–64	82–88	67–34	9	−9
1979	−13	39–69	84–93	67–42	16	−2
1980	−9	45–64	88–92	64–43	13	8
1981	17	45–64	82–95	58–42	2	8
1982	−14	35–66	80–87	...	...	...
Mean	−2.8	41.2–66.2	83.5–92.5	62.8–39.4	8.4	2.6

[a] Data from Twin Falls Weather Station provided by Myron Molnau, Department of Agricultural Engineering, University of Idaho, Moscow 83843.
[b] Average of daily high temperature. Warm temperatures favor early population increase, promoting large populations before first alfalfa cutting.
[c] Moderate fall temperatures favor population increase on postsummer alfalfa growth, promoting potential of large overwintering population.
[d] Expected aphid population response to indicated temperature factor.

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Fig. 3. Alfalfa plants in a field adjacent to PeLR-devastated pea fields, in 1980. The symptomatological counterparts of PeLR in peas were not correlated with infection of alfalfa plants by PeLRV.

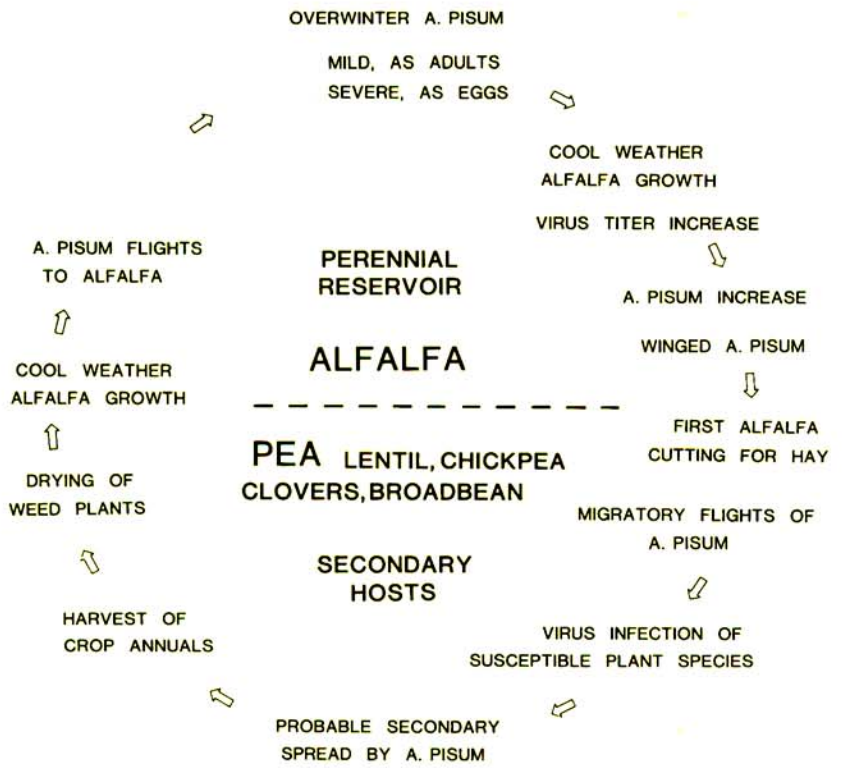

Fig. 4. Pea leaf roll disease cycle, depicting current concepts of the relationships among the pea aphid (*Acyrthosiphon pisum*), alfalfa as the principal PeLRV inoculum reservoir, other susceptible leguminous plants that are potential secondary PeLRV hosts, and annual season (winter above and summer below dotted line).

with the excellent personnel of seed companies, and I am confident that PeLR will also yield to comparable efforts.

As new, improved PeLRV-resistant pea cultivars are being developed, possible use of persistent, systemic aphicides to discourage prolonged feeding and colonization of aphids migrating from alfalfa to peas is being evaluated (13). Simultaneously, a valuable "aphid watch," monitoring spring aphid populations in alfalfa, is being published and distributed to seed company and research personnel by University of Idaho extension entomologist R. L. Stoltz.

Control of PeLR in peas by means of a regulated period during which no alfalfa is grown within a control area, as was accomplished with beet western yellows of sugar beets in the Salinas Valley of California (5), may not be practicable. Cooperative work between plant virologists and alfalfa breeders, however, could produce finished alfalfa synthetics resistant or immune to PeLRV and to other viruses for which alfalfa is a natural inoculum reservoir. In the meantime, PeLRV-resistant pea cultivars and management of and protection from aphid populations in alfalfa offer the greatest promise for PeLR control.

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