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An epidemic of celery mosaic potyvirus in celeriac (Apium graveolens var. rapaceum) in the Netherlands

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Abstract

An annually recurring virus epidemic has caused severe damage and sometimes total yield loss in crops of celeriac in the south-west of the Netherlands for several years since 1969. Celery latent virus, cucumber mosaic cucumovirus and tobacco rattle tobravirus were isolated from diseased plants, but a potyvirus was the most prevalent virus present. It did not cause local lesions in *Chenopodium amaranticolor* or *C. quinoa* and did not infect other non-Umbelliferae. The virus was identified as celery mosaic potyvirus and confirmed to be the causal agent.

Field surveys from 1971 to 1977 showed a rapid increase of disease incidence in consecutive years, soon leading to near-abandonment of the crop in the original centre of cultivation and its northward translocation, gradually followed by the disease. Incidence in fields often increased from zero by the end of July, when symptoms first appeared, to 100% early during September. In 1976 final incidence was 75 to 100% in 41% of the fields inspected. Temporary protection with aphid-proof cages showed that first infection occurred about three weeks before symptoms appear and not before the beginning of July, and that the virus may still spread after late September.

When testing samples from other umbelliferous crops and wild species near infested crops, several viruses were detected but not celery mosaic virus. The virus may be introduced from yet unknown distant sources, as also suggested by the pattern of spread in crops of celeriac.

Celeriac cultivars differ considerably in resistance to the virus, but resistant cultivars generally are of poorer quality for consumption and processing than the original highly sensitive cultivar. In recent years cultivation has recovered considerably with the advent of new cultivars.

Additional keywords: aphid transmission, celery latent virus, cucumber mosaic cucumovirus, resistance, sources of infection, tobacco rattle tobravirus, virus ecology.

Introduction

In the Netherlands, celeriac *Apium graveolens* L. var. *rapaceum* (Mill.) DC. is grown mainly as an arable crop for its turnip-like edible root. Although not of great economic importance, with an annual hectarage of c. 1000-1500, it is locally popular among farmers, especially in the province of Zeeland in the south-west of the country

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(Fig. 3a) because it fits well in crop rotation. Since 1969 a severe recurrent epidemic of virus disease has affected the area around Kruiningen and Waarde on the former island of Zuid-Beveland. Often total loss of affected crops ensued. This led to gradual abandonment of the crop in that region, and production moved northward gradually followed by the disease.

Since 1970 the etiology and epidemiology of the disease were investigated with a view to its possible control. Publication of results has been delayed by the continuing lack of information on the primary sources of infection from which spread occurs. Preliminary reports on celery mosaic potyvirus (CeMV) as the causal virus and on its incidence and distribution were by Bos (1973) and Mandersloot et al. (1973), respectively. The seed-borne celery latent virus (CeLV), also found in celeriac, has been further characterized (Bos et al., 1978), and data on 18 viruses other than CeMV found in umbelliferous crops and wild species in the Netherlands, when seeking infection sources of CeMV, are in press (Van Dijk and Bos, 1989).

The disease

Nursery-grown seedlings of celeriac are usually transplanted to the field by mid-May. Diseased plants do not appear before the end of July or early August. Primarily infected plants occur erratically scattered throughout infested fields. They first attract attention by a golden yellowing of some nearly full-grown leaves (Fig. 1). The leaves formed thereafter remain short, mostly downward-curled or narrowed with a diffuse vein-banding mosaic, giving the hearts of affected plants a stunted, light-green and

Fig. 1. Plant of celeriac 'Roem van Zwijndrecht' with first symptoms of natural infection by celery mosaic virus: golden yellowing of intermediate leaves.

bushy appearance (Fig. 2). Leaves fully developed before infection occurs may remain green, still allowing some further tuber development. Secondary spread of the virus later leads to group-wise occurrence of the disease, with severely affected plants in the centre of the enlarging groups (Fig. 2). New infections may occur up to late during September. Symptoms are especially striking in cv. Roem van Zwijndrecht, which was originally the major variety grown. After early infection, plants of this cultivar remain very stunted and tuber development is minimal. Then, disease incidence mostly is high and yield reduction severe, if not complete.

Materials and methods

Virus identification. Sap from leaf samples was inoculated onto celery (*Apium graveolens* var. *dulce* 'Golden Selfblanching'), *Chenopodium amaranticolor, C. quinoa, Nicotiana glutinosa,* and sometimes *N. tabacum* 'White Burley', cucumber and *Gomphrena globosa.* Leaf samples were often checked with the electron microscope, using crude sap and 2% PTA pH 6.8 as a stain. The most common elongate virus that was isolated (especially isolate Sel6) was tested with antisera to CeMV, isolate 20N, which only infects Umbelliferae, and to isolate 20F, which also infects non-Umbelliferae, both obtained from Dr. E. Luisoni, Torino Italy (Luisoni and Fedi, 1972). Particle length of Sel6 was determined in crude-sap preparations, using TMV particles as an internal standard. Back-inoculations of identified isolates were made onto celery 'Golden Selfblanching' and/or celeriac 'Roem van Zwijndrecht' to fulfill Koch's postulates.

Fig. 2. A group of diseased plants in a field of celeriac 'Roem van Zwijndrecht'. Severely stunted bushy plants infected early during development are in the centre of the group and some late-infected plants with leaf yellowing at its edge.

Surveying. During 1970 25 growers in Zeeland were questioned. From 1971 to 1977 nearly all celeriac crops grown in the region were inspected in September, and the incidence of infection was rated visually in classes of 0-5, 5-15, 15-20 and 50-100% to study geographic distribution and economic importance. Seasonal development of disease incidence was investigated during August and September 1972 by counting diseased plants weekly in four adjacent rows with 400 plants in total in ten fields located along a more or less straight, c. 8.5 km long transect from the town of Waarde in the centre of the epidemic to Yerseke at its north-western limit. During 1974 similar records were made in three farmers' fields at Oostdijk, Biezelinge and Yerseke.

Other possible sources of infection. Testing of other wild and cultivated Umbelliferae in search of sources of infection was as outlined by Van Dijk and Bos (1989). In 1974 32 leaf samples were collected near severely affected crops of celeriac in Zeeland. In 1988 another 180 samples from wild Umbelliferae from Zeeland and the southern part of the neigbouring province of Zuid Holland, where the disease had also been prevalent, were tested in ELISA with an antiserum prepared to CeMV isolate Sel6 by D.Z. Maat, IPO.

Time of infection. Cageing experiments were made to provide temporary protection for plants of celeriac 'Roem van Zwijndrecht' against virus infection by aphids. Plots with 30 to 36 plants per treatment were covered with nylon gauze over metal frames for different periods of time after insecticide application to prevent aphid infestation during cageing. Disease incidence and symptom development were later compared with those in uncovered plots.

Variety trials. Preliminary observations on 20 cultivars of celeriac were made at Kruiningen near Goes in 1971, and two more detailed experiments followed during 1972 and 1977.

In 1972, 14 cultivars were submitted to natural infection at Oostdijk near Waarde in the centre of the epidemic. Each cultivar was planted with four replicates, 49 plants per replicate. Disease incidence was rated weekly from August 2 onwards.

The more extensive 1977 trial was by the former Experiment Station for Vegetable Growing in the Open at Alkmaar. It was at Colijnsplaat, at the northern margin of the epidemic, and performed in consultation with IPO. Half of the plants of each plot of 47 cultivars and breeding lines, including some cultivars not tested before, in three replicates of 48 plants each, were sap-inoculated in the glasshouse with CeMV isolate Sel6 three weeks before transplanting to the field on June 8. Inoculated plants served as sources of infection for further spread by aphids in the field.

Results

Virus identification. One or two viruses were isolated from all but one of the 16 samples from diseased plants with varying symptoms that were tested during 1971 by sap inoculation onto test plants.

Cucumber mosaic cucumovirus, earlier associated with the epidemic (Scheele, 1970), was isolated from only three samples. It could easily be recognized by host range and symptoms on test plants. The affected leaves had striking yellow ring and line pat-

terns which could readily be reproduced in celery 'Golden Selfblanching'. Such symptoms are rarely observed in celeriac in the field. Inoculated plants usually recovered later from disease.

Six plants contained a virus producing chlorotic local lesions in *C. amaranticolor* and *C. quinoa*, followed by unusual irregular systemic rings or line patterns in this species and especially in *C. quinoa*, together with leaf malformation and some plant stunting in the latter. After back-inoculation the virus did not produce symptoms in celery or in 13 cultivars of celeriac. It was later identified as the ungrouped seed-transmitted celery latent virus and it was found not to enhance the symptoms of celery mosaic virus in celery (Bos et al., 1978).

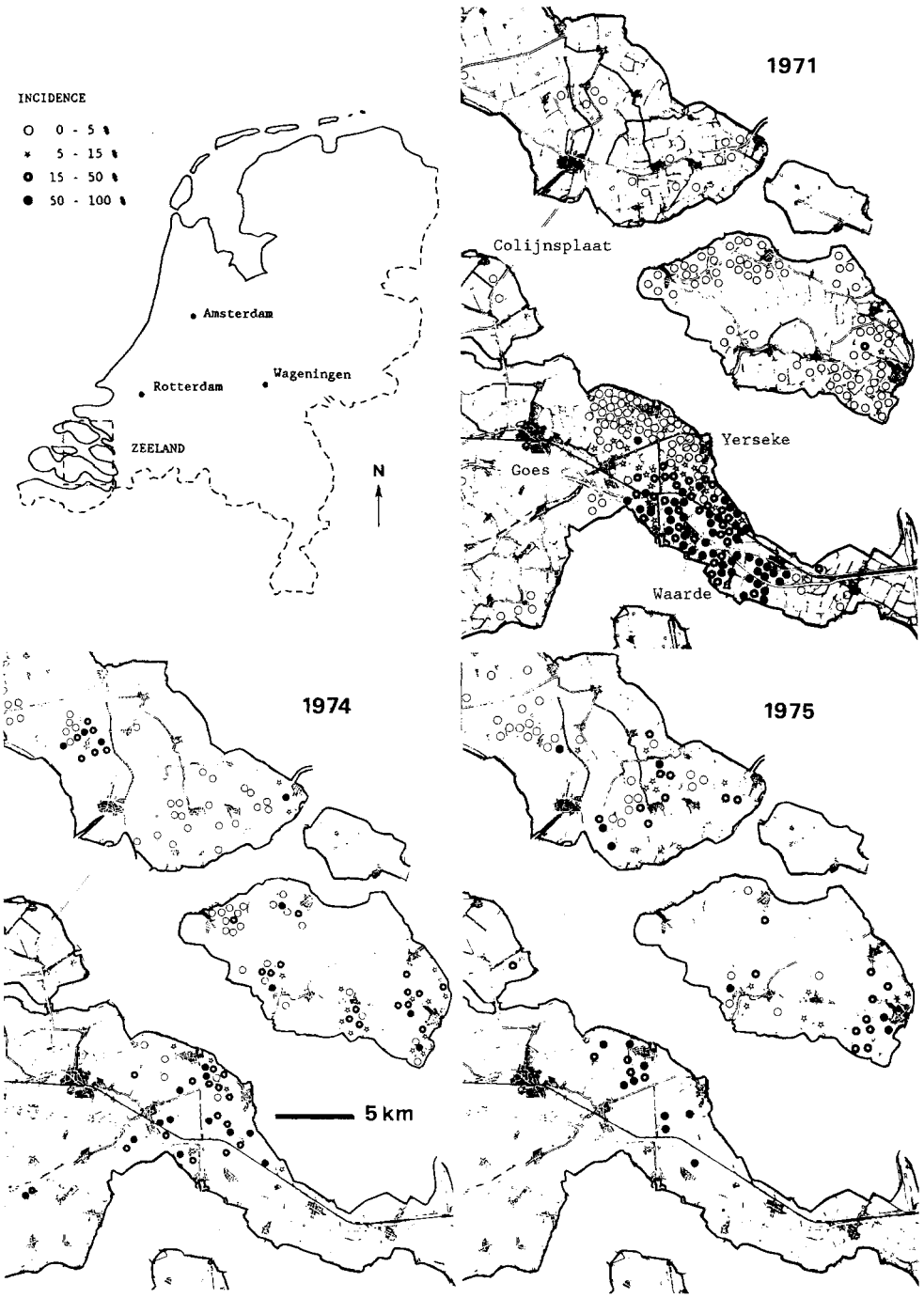
Tobacco rattle tobravirus was isolated from one plant with chlorotic stippling. It could easily be distinguished by its local reaction on both *Chenopodium* species and by its local and systemic reactions on *Nicotiana glutinosa* and *N. tabacum*. After back-inoculation onto celery 'Golden Selfblanching' it produced severe chlorotic stippling and leaf malformation, and its rigid particles of two lengths could readily be recognized by electron microscopy.

Sap from 10 of the leaf samples and also from some tuber samples tested contained the virus with flexuous particles which was easily sap-transmissible, but not to *Chenopodium* spp. or other non-Umbelliferae. It produced systemic vein-clearing in celery and celeriac about 10 days after inoculation, followed by chlorotic vein-banding, leaf narrowing, leaf curling and plant stunting similar to the field symptoms observed.

Measurement of 106 particles of isolate Sel6 in crude-sap preparations of celeriac gave an average particle length of 789 nm, and 28 particles from celery a length of 787 nm. In serological tests performed by Mr. D.Z. Maat, IPO, the virus reacted up to a dilution of 1024 with a homologous antiserum prepared by Mr. Maat and up to the same dilution with an antiserum to celery mosaic virus isolate 20N infecting Umbelliferae only, but up to dilution 8 with the antiserum to isolate 20F also infecting non-Umbelliferae (Luisoni and Lisa, 1969; Luisoni and Fedi, 1972). Our CeMV isolate Sel6 was tested further for its host range and symptoms and later (Van Dijk and Bos, 1989) compared with parsnip mosaic potyvirus, afterwards found incidentally in celeriac with symptoms resembling those of CeMV, though more severe and malforming and sometimes accompanied by necrosis. It differs from CeMV in producing local lesions in *C. amaranticolor, C. quinoa* and *Vicia faba*. During our later extensive search for sources of infection we again detected CeMV in celery and celeriac but never in other Umbelliferae.

Surveying. The preliminary survey in 1970 revealed a concentration of the disease around Kruiningen and Waarde, the then main centre of celeriac cultivation in Zeeland. The inquiry showed that in 12 of the 25 fields visited (all with 'Roem van Zwijndrecht') over 60% of the plants were diseased. In 6 fields incidence was between 10 and 60%, and in 7 fields it was less than 10%. There was no significant effect of use of insecticides (parathion or systemic insecticides) or of origin of planting material on disease incidence. Early planted and well-developed crops seemed to suffer least.

During the more systematic and detailed surveys conducted after 1970 the following numbers of fields throughout the province were visited with the percentage of fields with disease incidences exceeding 75% indicated in brackets: 1971: 279 (12%); 1972:


INCIDENCE
0 - 5 %
5 - 15 %
15 - 50 %
50 - 100 %
Amsterdam
Rotterdam
Wageningen
ZEELAND
N
1971
Colijnsplaat
Yerseke
Goes
Waarde
1974
1975
5 km

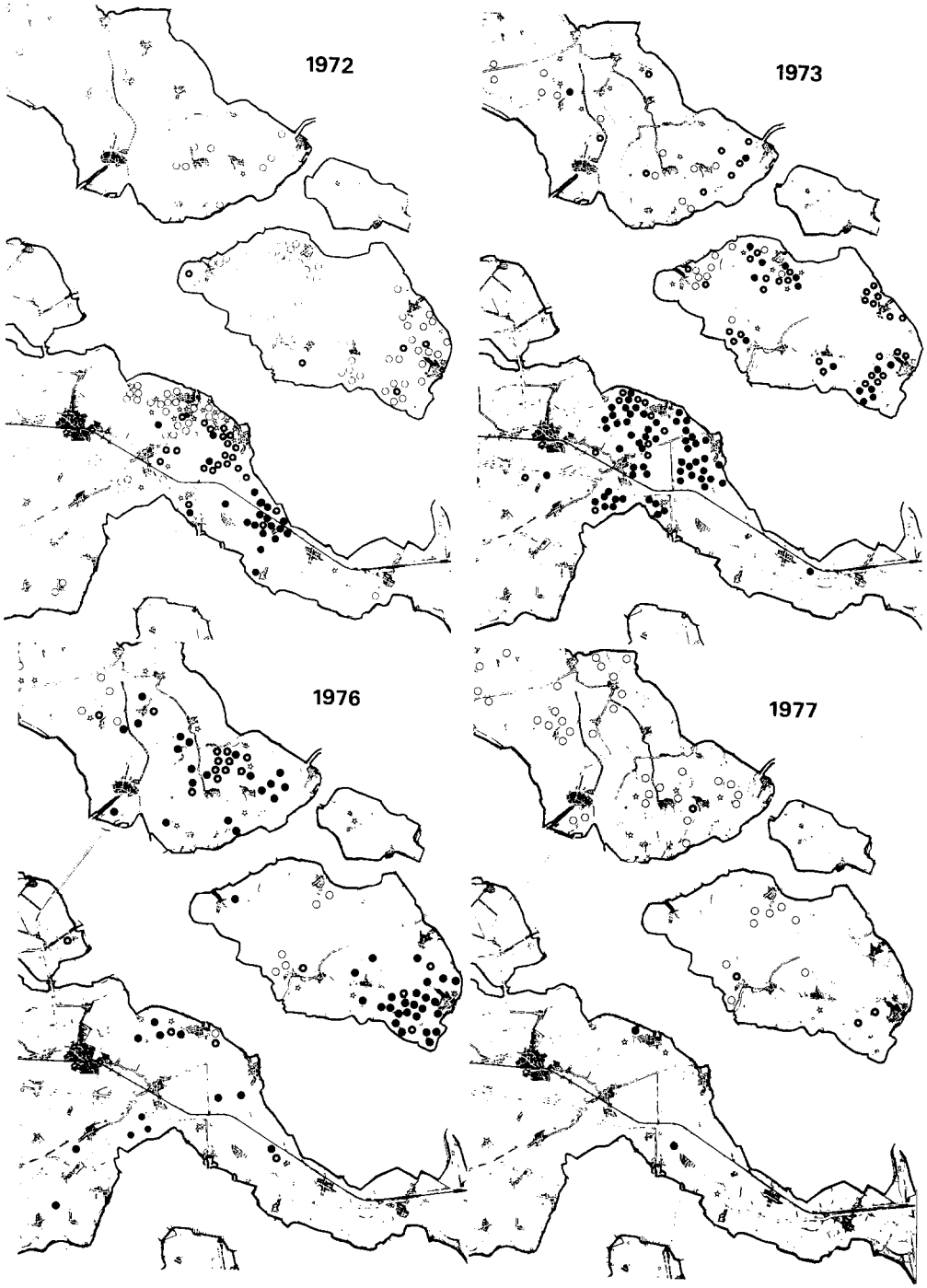


Fig. 3. Distribution and incidence of mosaic disease in celeriac fields in the major celeriac-growing part of the province of Zeeland during 1971 through 1977. Each circle represents one field.

182 (5%); 1973: 162 (30%); 1974: 149 (7%); 1975: 111 (16%); 1976: 117 (41%); 1977: 69 (1.5%). More detailed results with special reference to distribution in the major celeriac-growing part of the province are given in Fig. 3. These data show disease incidences varying according to year with highest percentages of severely affected fields (over 75% of the plants diseased) in 1973 (30%) and 1976 (41%). They also show a rapid increase of disease incidence through the years beginning in the south, a gradual abandonment of celeriac cultivation there, and its translocation northward with a gradually following epidemic.

Fig. 4 records the increase of incidence with time during the growing season of 1972 at 10 locations from the centre to the margin of the epidemic. In the centre (especially at Waarde: Fig. 4, upper line) the rate of infection started linearly and was very rapid, increasing from zero to c. 100% within c. one month. At the margin (Yerseke: Fig. 4, the two lower lines) disease onset was late and spread was slow. In 1974 in three fields (Oostdijk, Biezelinge and Yerseke) the disease started later, but then increased considerably, even at Biezelinge and Yerseke where in 1972 spread was much less.

Other possible sources of infection. None of the 32 samples of other Umbelliferae collected near severely infested fields in 1973 and assayed on test plants, nor any of the 180 samples collected in 1988 and tested in ELISA, were found to contain CeMV. Of the 974 samples of wild Umbelliferae other than celery and celeriac (but including the above 32 from Zeeland) tested biologically, a high proportion contained isolates of

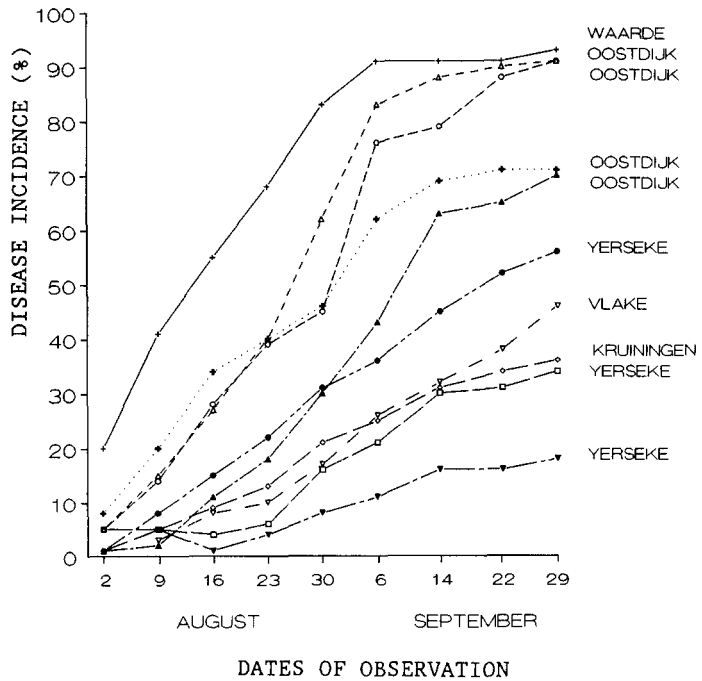


Fig. 4. Increase of mosaic disease in individual celeriac fields at different locations on the former island of Zuid Beveland during 1972. The fields were located along a nearly straight c. 8.5 km long transect between Waarde, in the centre of the epidemic, and Yerseke, at its margin.

18 different viruses but not CeMV (Van Dijk and Bos, 1989). Such tested species included *Anthriscus sylvestris*, *Heracleum sphondylium*, *Pastinaca sativa* and wild carrot (*Daucus carota*) from near diseased celeriac crops.

Time of infection. Symptoms never appeared before late July, and seldom before early August.

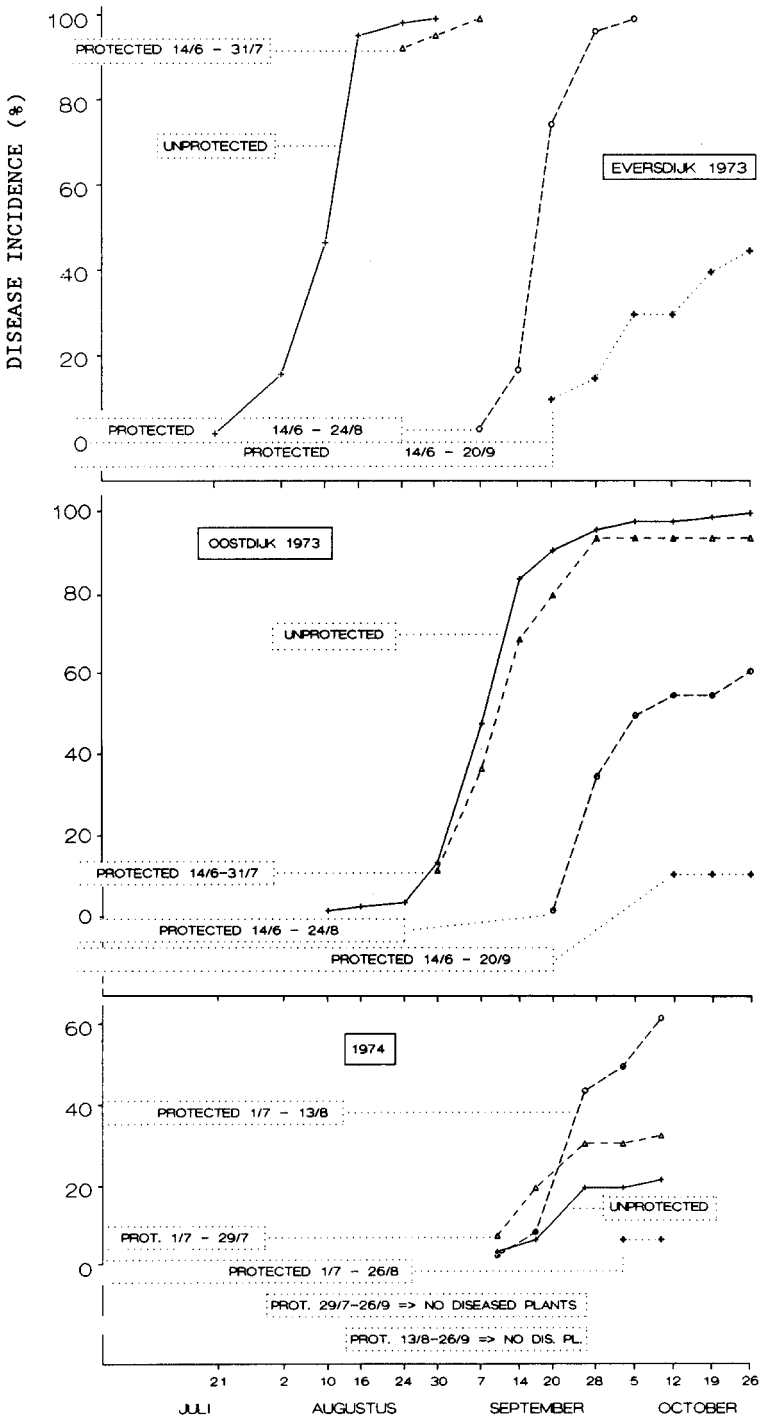
The 1973 cageing experiment was at Eversdijk near Biezelinge, where in previous years disease incidence was low, and at Oostdijk, where it was high. Few plants developed symptoms while or shortly after having been under cover. They occurred exclusively in external rows where they may have contracted infection via leaves in contact with the gauze, probably probed by aphids from outside. None of the plants in inner rows ever showed symptoms before cage removal. The results represented in Fig. 5a, b were unexpected, as epidemic build-up was earlier and more rapid at Eversdijk than at Oostdijk. First symptoms developed 2 to 4 (mostly 3) weeks after cage removal. At Eversdijk 3 weeks after cage removal on July 31 disease incidence already approached 100%. It also increased rapidly after cage removal on August 24, demonstrating massive infection pressure around late July and towards the end of August. Considerable infection occurred after September 20, as indicated by incidences in plots covered till then. Early symptom development at Eversdijk soon after cage removal on September 20 can be explained by accidental entry of aphids.

In the 1974 cageing experiment (Fig. 5c) the low disease incidence even in the unprotected plot demonstrated low natural infection pressure at the location. This agrees with the low overall incidence of the disease in the province that year. It is also consistent with the observations in three farmers' fields recorded at the end of the previous section demonstrating a late influx of virus vectors that year. The results of the cageing experiment demonstrate that the main virus introduction that year was between August 13 and 26. The 1974 summer was cool with only one spell of sunny and warm weather from August 14 to 23. Higher disease incidence in the temporarily protected plots than in the unprotected ones may be ascribed to the poorer and more open stand of the protected plots.

Variety trials. In the preliminary trial in 1971 20 celeriac cultivars differed considerably in vulnerability to natural infection. Six cultivars showed disease incidences of 75-100% towards the end of the growing season, which also demonstrated high natural infection pressure. In the other cultivars incidence was 50-75 in 4, 25-50 in 3, 10-25 in 5, and less than 10 in 2. 'Roem van Zwijndrecht', the then prevailing cultivar, 'Hild's Neckarland' and 'Zwindra' were among those most vulnerable. 'Ceva' and 'Bronskogel' were least vulnerable but unattractive for processing.

During 1972, 14 cultivars were field tested in greater detail and results of weekly disease ratings are recorded in Fig. 6. 'Prager Reuzen' and 'Iram' emerged as new resistant cultivars, though also unacceptable to the processing industry. The more vulnerable cultivars showed considerable differences. Some of the partially resistant cultivars not only showed less plants infected at harvest and less conspicuous symptoms than the less resistant cultivars, but spread in them was delayed (Fig. 6, Table 1, first two columns). Results of disease ratings were similar in years with obviously different infection pressures (Table 1, last two columns).

Results of the third and more extensive experiment have already been reported in


DISEASE INCIDENCE (%)
100
80
60
40
20
0
PROTECTED 14/6 - 31/7
UNPROTECTED
EVERSDIJK 1973
PROTECTED 14/6 - 24/8
PROTECTED 14/6 - 20/9
OOSTDIJK 1973
UNPROTECTED
PROTECTED 14/6-31/7
PROTECTED 14/6 - 24/8
PROTECTED 14/6 - 20/9
1974
PROTECTED 1/7 - 13/8
UNPROTECTED
PROT. 1/7 - 29/7
PROTECTED 1/7 - 26/8
PROT. 29/7-26/9 => NO DISEASED PLANTS
PROT. 13/8-26/9 => NO DIS. PL.
21 2 10 16 24 30 7 14 20 28 5 12 19 26
JULI
AUGUSTUS
SEPTEMBER
OCTOBER
DATES OF OBSERVATION

◀ Fig. 5. Diagrams representing the increase in disease incidence after varying periods of protection from aphids by covering of field plots with nylon cages at two locations in 1973 (top and middle) and one location in 1974 (bottom). Transplantation, end of May.

Table 1. Difference between vulnerable cultivars of celeriac in time to 50 and 80% disease incidence in the field in 1972, and final incidences on 22 September 1972 and 27 September 1971.

Cultivars arranged according to decreasing vulnerability	Time in days from August 1 to incidence of		Final incidence (%)	
	50%	80%	1972	1971
Zwindra	14	26	100	83
Roem van Zwijndrecht RZ	21	33	94	74
Hild's Neckarland	24	32	97	80
José	27	35	94	67
Roem van Zwijndrecht SG	28	46	87	66
Lustra	30	49	84	51
Marmerkogel	32	53	81	
Limburgse	37	—	65	25
Pomona	41	—	70	32
Albaster	50		55	17

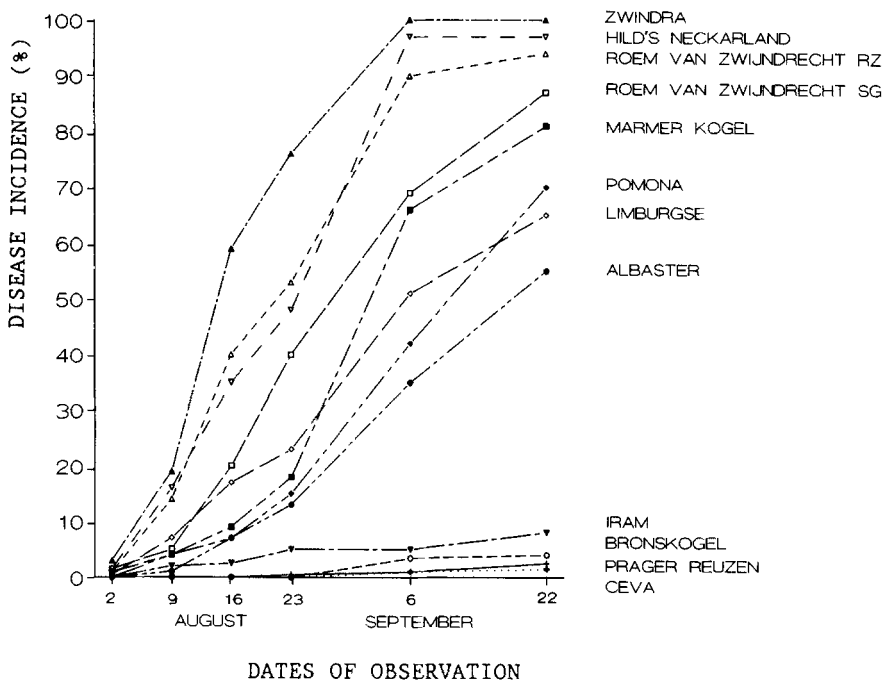


Fig. 6. Differences between celeriac cultivars in increase of disease incidence in trial field during 1972.

Dutch (De Kraker, 1979). The main results will be summarized here for completeness. At transplanting, the sap-inoculated lots of all genotypes, including 'Bronskogel' and 'Monarch' but excluding 'Brandel', had over 75% of the plants with symptoms. In most of them incidence was over 90%. Symptoms were least conspicuous in 'Brandel', and occurred in only 33% of the plants. After transplanting, diseased plants of 'Bronskogel', 'Iram' and 'Monarch' recovered considerably. Others, such as 'Roem van Zwijndrecht' and 'Hild's Neckarland' declined almost completely. Virus spread to the non-inoculated plants in other plot halves was rapid, and percentages of plants remaining healthy varied from 0 ('Albaster', 'Balder', 'Hild's Neckarland', 'José' and 'Zwindra'), 21-50 (two selections of 'Roem van Zwijndrecht'), to 100 ('Brandel'). Unfortunately, the latter also is poor in processing quality. 'Monarch' scored highest in capacity to recover from infection and combines resistance with reasonable quality for consumption and processing. It is followed by 'Iram' with slightly lower virus resistance (De Kraker and Riepma, 1979).

General discussion

Although earlier ascribed to cucumber mosaic virus (Scheele, 1970), the cause of the epidemic is now known to be celery mosaic virus, an aphid-transmitted potyvirus (Shepard and Grogan, 1971). Identification was by host range and symptoms, particle morphology and size, and close serological relationship with the Italian 20N isolate that does not infect non-Umbelliferae. The original antiserum to CeMV (Shepard and Grogan, 1976) is no longer available (Dr. J.F. Shepard, pers. comm. 1976). The other viruses isolated did not produce symptoms characteristic of the epidemic disease or did not contribute to the symptoms caused by CeMV (celery latent virus), or were found incidentally only (cucumber mosaic virus, tobacco rattle virus). CeMV may be confused with parsnip mosaic potyvirus, later found incidentally in some diseased celeriac plants with symptoms resembling those of CeMV, though more severe. It was recently also isolated from naturally infected CeMV-resistant celeriac 'Monarch' with severe symptoms (Van Dijk and Bos, 1989). Another virus encountered incidentally in celeriac, with small chlorotic or yellow spots or concentric rings but difficult to transmit mechanically, is celery yellow spot virus (Van Dijk and Bos, 1989).

CeMV is known from several countries (Shepard and Grogan, 1971) including those neighbouring the Netherlands: Germany (Götte, 1957; Brandes and Luisoni, 1966; Wolf, 1968), Belgium (Roland, 1951; De Vos and Liekens, 1970; Matthieu and Verhoyen, 1979), France (Marchoux et al., 1969, 1970) and Britain (Walkey et al., 1970; Pemberton and Frost, 1974, 1986) where incidence often also is high. In France cucumber mosaic virus was more prevalent in celery and celeriac than CeMV (Marchoux et al., 1970).

In the Netherlands the disease may have occurred long before 1969 without reaching epidemic proportions. This publication documents its annually recurrent rapid epidemic development in celeriac, and the gradual further geographic spread up to and including 1977, the last year of quantitative observation.

Epidemic development was usually sigmoid as for many other infectious diseases. With vector-borne viruses, the rate of increase usually depends on number and proximity of sources of infection, number and efficiency of vectors, wind direction, and on host susceptibility (e.g. Bos, 1983). The curves of Fig. 4 strikingly resemble those

of watermelon mosaic potyvirus 2 in cantaloups in Arizona (Nelson and Tuttle, 1969). There, the sources of infection (wild growing mallow) were near the crops. Non-persistently transmitted viruses are generally assumed to spread distances of less than 100 m and to do so into crops from its border with the source crop or wild vegetation in gradients depending on wind direction, as with bean yellow mosaic potyvirus from adjacent clover fields into crops of *Phaseolus* bean (Hampton, 1967). Although Fig. 4 and the gradual translocation of the epidemic suggest the occurrence of nearby sources of CeMV, such gradients of infection have never been observed in celeriac fields in the Netherlands. First infections usually appear scattered throughout fields, suggesting virus introduction by aphids from long distances. Moreover, no other nearby sources of infection have so far been found. Large-scale testing of Umbelliferae, wild or cultivated, other than celery and celeriac revealed the presence of 18 different viruses but no CeMV (Van Dijk and Bos, 1989). *Anthriscus sylvestris*, *Heracleum sphondylium* and *Pastinaca sativa* also tested are known to occasionally harbour CeMV in Britain (Walkey and Cooper, 1971). Poison hemlock (*Conium maculatum*), mentioned as a major wild host of CeMV near celery fields in Britain (Walkey and Cooper, 1971; Pemberton and Frost, 1974) and of the virus in Washington State (Howell and Mink, 1981), is rare in the province of Zeeland. Cultivated carrot, susceptible to Dutch isolates of CeMV, cannot play a role in the ecology of the virus since it does not overwinter in the field. Wild carrot, prevalent in Zeeland, could act as a source of infection of CeMV, as in Washington State USA (Howell and Mink, 1981), but we could not isolate the virus from it when sampled near severely diseased crops of celeriac. Overwintering celery and celery grown for seed were assumed to be major overwintering sources of the virus in Belgium (Bosschaert et al., 1974). In the Netherlands celery or celeriac are not replanted a second year for seed production, and harvested celeriac tubers are rarely stored locally in Zeeland and they are not present there at the time of planting (F. Vader, pers. comm. 1989).

Seed transmission of CeMV has never been reported (Shepard and Grogan, 1971) and is unlikely to initiate crop infestation. We have never observed diseased plants in nurseries and never in farmers' fields before the end of July. Crops grown from planting material distributed to different parts of the country only contracted disease when planted in the region where the epidemic occurred. The only virus isolated from seeds and glasshouse-grown seedlings of celeriac was celery latent virus (Bos et al., 1978).

Insect transmission is by many aphid species in the non-persistent manner (Shepard and Grogan, 1971), but with considerable differences in efficiency (Karl and Wolf, 1974). *Cavariella aegopodii* reaches peak population densities in the Netherlands during the first half of July but occurs in high densities throughout June or even earlier, with low densities during autumn according to observations with the 12.2 m aerial suction trap at Colijnsplaat, Zeeland (documentation IPO). It may not be the major vector since these flight data do not correspond with results of our cageing experiments. These indicate large-scale plant infection near the end of July in 1973 and preponderantly between August 13 and 26 in 1974, and show considerable further virus spread late during the 1973 season. *Myzus persicae* might play an important role because suction trap data in the region show that it reaches high flight densities late during July, and in 1973 and 1974 peak densities were observed by mid or late August (documentation IPO).

Non-persistent transmission is generally assumed to be over short distances only. However, retention of CeMV (necrotic strain) was found to be up to 6 h in *Myzus persicae* during post-acquisition starvation (Matthieu and Verhoyen, 1981), and aphids can disperse far during this period. This long retention may explain early-season spread of beet mosaic potyvirus by this aphid in California from large 'overwintering' sugar-beet crops to newly planted spring crops over distances up to 12 to 15 miles (Shepherd and Mills, 1970). Recent studies have shown that aphid vectors of maize dwarf mosaic potyvirus may retain the virus for more than 18 h (Berger et al., 1987). This led Zeyen et al. (1987) to ascribe a dramatic 1977 epidemic of the virus in sweet corn in Minnesota in the absence of local sources of infection to a weather-conditioned abnormal influx of viruliferous aphids from the southern Great Plains of North America. A similar spread from distant, yet unidentified sources might hold for CeMV in Zeeland although the epidemic build-up during a series of consecutive years and the gradual translocation of the epidemic suggest the occurrence of sources of overwintering in the region itself.

Since sources of infection of CeMV cannot (yet) be avoided and systemic or other conventional insecticides are not likely to be of much help in decreasing non-persistent virus spread, frequent spraying with mineral oil has been tested in Belgium. Although successful in experiments (Matthieu and Verhoyen, 1979), later field tests with a number of mineral oils showed unacceptable reduction in tuber development and other limitations. They have therefore not been recommended for general use (Matthieu et al., 1982).

Cultivar resistance now plays a major role in solving the problem. New cultivars like 'Monarch' and 'Iram' have a reasonable degree of resistance. They not only suffer less from infection, but their wide application has led to lower disease incidence and delayed epidemic crop attack. The new cultivars also better meet present demands for anthocyanin-free celeriac. With their advent, celeriac cultivation has recovered considerably in Zeeland.

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Samenvatting

Een epidemie van selderijmozaïekvirus in knolselderij (Apium graveolens var. rapaceum) in Nederland

Sinds 1969 trad gedurende een aantal opeenvolgende jaren in de provincie Zeeland in knolselderij een ernstige virusziekte epidemisch op. Vaak mislukten gewassen geheel. Uit viruszieke planten konden het latente selderijvirus, komkommermozaïekvirus, tabaksratelvirus en een potyvirus worden geïsoleerd. Het laatste kwam echter het meest voor en was in zijn optreden gecorreleerd met de voor de epidemie karakteristieke symptomen. Het kon op grond van waardplanten en symptomen, deeltjesvorm en grootte (ca 780 nm) en serologie worden geïdentificeerd als het selderijmozaïekvirus. In de kas konden in selderij de voor de ziekte kenmerkende symptomen ermee

worden gereproduceerd.

Reeds in 1970 waren in de omgeving van Kruiningen en Waarde in 12 van de 25 geïnspecteerde velden meer dan 60% van de planten ziek. Een systematische inventarisatie gedurende 1971 tot en met 1977 toonde aan dat de ziekte in die periode snel verder om zich heen greep en de teelt in het oorspronkelijke teeltgebied niet meer lonend maakte. De ziekte volgde echter geleidelijk de noordwaarts uitwijkende teelt. In 1976 was 41% van de 117 geïnspecteerde velden voor meer dan 75% aangetast. In zulke velden kon het percentage zieke planten vanaf eind juli tot begin september toenemen van 0 tot nagenoeg 100.

De eerste symptomen treden jaarlijks rond eind juli of begin augustus op. Proeven, waarbij veldjes met planten gedurende bepaalde perioden werden afgedekt met bladluisdichte gaaskooien, toonden aan dat de eerste infectie optreedt ca 3 weken (2-4) voor het verschijnen van de eerste symptomen en op zijn vroegst begin juli, en dat verdere verspreiding nog tot na 20 september plaatsvindt. De besmettingsbronnen zijn nog steeds niet gevonden. De wijze van optreden van de ziekte in het veld doet vermoeden dat het virus niet afkomstig is van dichtbij voorkomende wilde planten.

Er blijken grote rasverschillen in resistentie te bestaan. De voor consumptie en conservenverwerking gewilde rassen, waaronder vooral 'Roem van Zwijndrecht', zijn alle zeer kwetsbaar. Met het geleidelijk naar voren komen van meer resistente, en voor de export vooral gevraagde anthocyanine-vrije rassen, zoals 'Monarch' en 'Iram', heeft de knolselderijteelt zich in Zeeland grotendeels hersteld.

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