

# Sugar-beet rhizomania: the spread of a soilborne disease

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Sugar-beet crops can be decimated by rhizomania, a disease caused by a virus, yet transmitted by a fungus in the soil. Although stringent control measures are in place in the UK to prevent its spread; severe economic losses are being experienced in other parts of the world.

Rhizomania disease of sugar-beet – so-called because of its ‘mad root’ symptoms – is caused by a virus (beet necrotic yellow vein virus) transmitted by a soilborne parasitic fungus, *Polymyxa betae*. *Polymyxa* species are members of a small group of zoosporic fungi that do not produce hyphae; indeed the debate continues as to whether they are truly fungi or more closely related to the protozoans. They infect by means of swimming spores which attach themselves to the rootlets and inject their contents (which may contain the virus) into the superficial cells. Here the fungus develops and differentiates to produce a further generation of zoospores which are released to infect neighbouring roots. Several such cycles of multiplication occur during the growing season. At some stage, however, usually in more mature plants, the fungus switches to producing thick-walled resting spores, which are released into the soil when the rootlets decay and can survive almost indefinitely, protecting the virus particles they contain. Virtually undiminished disease intensity has been recorded in fields in Italy where susceptible crop species had not been grown for 30 years. This longevity, unusual in soilborne fungal plant pathogens subject to the degradative activity of the general soil microflora, creates particular problems for disease management since eradication of the disease on a field scale is impossible.

## ● The fungal vector

The fungus itself occurs in almost all fields worldwide where sugar-beet has been grown for any length of time. It is a relatively harmless root parasite. Only where the virus has been introduced does the growth of the plant root become severely damaged, with yield losses of up to 80%. However, the fungal vector plays the key role in the build-up and spread of the disease. Sugar beet is its only major arable crop host and there is effectively no multiplication in the soil in the absence of this crop. Experiments carried out in the Netherlands

have shown an increase in soil inoculum (virus-containing spores per gram soil) under sugar-beet crops of  $10^4$ -fold in a single growing season and  $10^6$ -fold over two growing seasons. This is despite the fact that only about 10–15% of fungal spores contain detectable virus, even in the most severely diseased areas. In normal practice, sugar-beet is grown every third or fourth year in the crop rotation and experience has shown



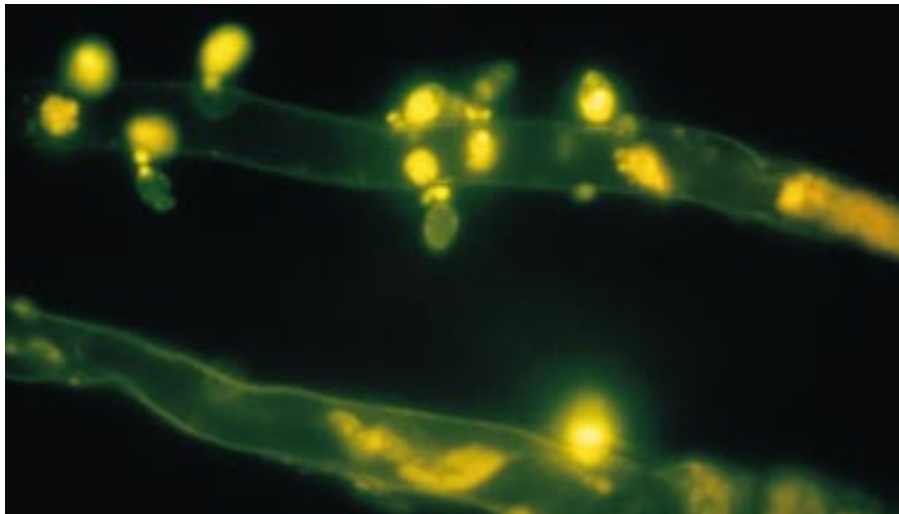
that it takes approximately two to three beet crops from the time a field is first contaminated to multiply up inoculum sufficiently to cause obvious symptoms in the plants depending, of course, on the initial level of contamination. This long ‘latent period’, of at least 5–10 years, during which agricultural activity proceeds unchecked, is a major factor in the spread of rhizomania.

## ● Spread and occurrence of the disease

Historically, the disease was first officially recorded in the Po valley in Northern Italy in 1952, though anecdotal evidence suggests that it had been present in this region at least since the second world war. Subsequently, the disease was recorded in Japan (1965) and, during the 1970s and early 80s, increasingly throughout Europe (Table 1). Though difficult to establish unequivocally so long after the event, much of this long-distance international spread is thought to have been due to seed being contaminated with infested soil. The Po valley was one of the world’s major sugar-beet seed-producing areas during this period and many of these crops were unknowingly grown on rhizomania-infested fields. Unprocessed raw seed carries

**Table 1. First records of rhizomania in different countries**

1950s and 1960s	1970s	1980s and 1990s
Italy (1952)	Yugoslavia (1971)	Hungary (1982)
Japan (1965)	Greece (1972)	USA (1983)
	France (1973)	Switzerland (1983)
	Germany (1974)	Bulgaria (1983)
	Czech. Rep. (1978)	Netherlands (1983)
	China (1978)	Belgium (1984)
	Austria (1979)	UK (1987)
	Romania (1979)	
	USSR (1979)	Sweden (1997)



spores are readily dispersed in this way.

In most countries where sugar-beet is grown, the disease has been established too long or has already spread too extensively for any measures aimed at limiting further spread to be worthwhile. Instead, reliance is being placed on growing recently introduced sugar-beet varieties that have partial genetic resistance to the virus. As a result the disease has spread very rapidly in many countries

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Fig. 1. The 'mad-root' symptoms of rhizomania disease.  
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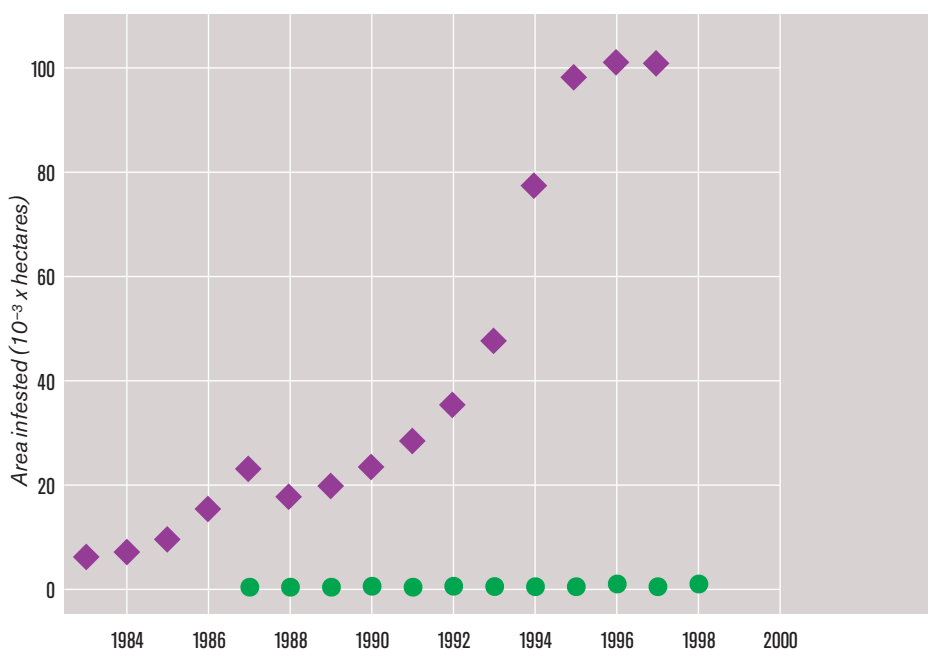
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Fig. 2. *Polymyxa betae* zoospores infecting sugar-beet rootlets.  
IACR-BROOM'S BARN

much dust and soil and, although seed destined for the production of commercial root crops is cleaned, processed and treated with fungicides prior to use, the seed used by plant-breeding companies for performance and breeding trials often was not so treated. In the USA, rhizomania was first discovered in 1983 on a small breeder's trial plot in California and within 5 years had spread to 30,000 hectares! The virus has never been detected within clean seed and repeated efforts to transmit the virus by means of vectors other than *P. betae* (e.g. insects and nematodes) have failed.

What has been demonstrated, however, is that movement of even small amounts of contaminated soil by whatever means has the potential to spread the disease. Again, studies in the Netherlands have shown that as little as 5 kg of rhizomania-infested soil spread over a 1 hectare area of a field is sufficient to generate uniformly severe symptoms in a sugar-beet crop within two growing seasons. Clearly, therefore, even a few grams would be sufficient to initiate the small patches of disease that normally appear when rhizomania first shows up in a field. Throughout the world sugar-beet tends to be grown in intensive arable areas and large-scale machinery is often used in the cultivation and harvesting of the crop. Furthermore, the harvested roots have to be transported to a central factory for processing and sugar extraction. All of this results in considerable movement of soil from field to field and from farm to farm. Also, waste soil and water accumulated at factories, from washing the roots prior to processing, has to be disposed of. In most countries this not inconsiderable amount of waste (1 million tonnes of soil per annum in the UK alone) finds its way back on to agricultural land. In Japan, this practice is known to have played a major role in contaminating a large part of the sugar-beet-growing area in 1975. Irrigation also contributes to spreading the disease in many areas of the world by re-cycling contaminated drainage water, since the fungal resting

in recent years. The total infested area in Western Europe, for example, increased from 245,000 hectares in 1993 to 530,000 hectares in 1997. In France, where surveys have recorded the diseased area each year, rhizomania increased from 6000 hectares in 1983 to 101,000 hectares in 1996, where it has subsequently stabilized (see Fig. 3). By contrast, countries with a cooler, maritime climate, such as the UK and Sweden, exhibit a much slower rate of spread; indeed some of these, such as Denmark and Ireland, have yet to record the disease. This is because both fungus and virus are favoured by warm soil temperatures, the optimum being 25°C.

Fig. 3. Annual area affected by rhizomania in France (◆) and the UK (●)



Data: IACR-Broom's Barn

### ● Control measures

In the UK, since the first recorded outbreak in 1987, strenuous efforts have been made to slow disease spread. Rhizomania is a notifiable plant disease under statutory control. As part of the policy of containment operated by MAF, for example, imports of plant material that might carry soil (e.g. seed potatoes for planting) must come from certified rhizomania-free areas, and used agricultural machinery is required to be steam-cleaned at the port of entry. Soil limits are imposed on all vegetables, including potatoes, imported for consumption and controls placed on any waste material arising from their subsequent processing in this country. Import controls

### ● The future

Rhizomania has become a major economic problem for the sugar-beet industries of most countries where the crop is grown in only the last 20 years. Increased intensity of cropping and the scaling-up of production methods have encouraged its multiplication and spread. The ease with which the agents are transmitted in soil, their almost indefinite persistence and the long incubation period before crops show symptoms of the disease, all contribute to its continued, inexorable spread. Ultimately, it is likely that almost all the world's sugar-beet-growing land will be contaminated with the virus, as it is already with the fungal vector. However, virus-resistant varieties have become very widely grown over the last few years, and are likely to provide the long-term solution to the problem. Indeed, rhizomania resistance will eventually become an essential prerequisite to successful sugar-beet production. Once again, plant breeders have risen to the challenge of a major disease threat with an economically and environmentally acceptable solution.

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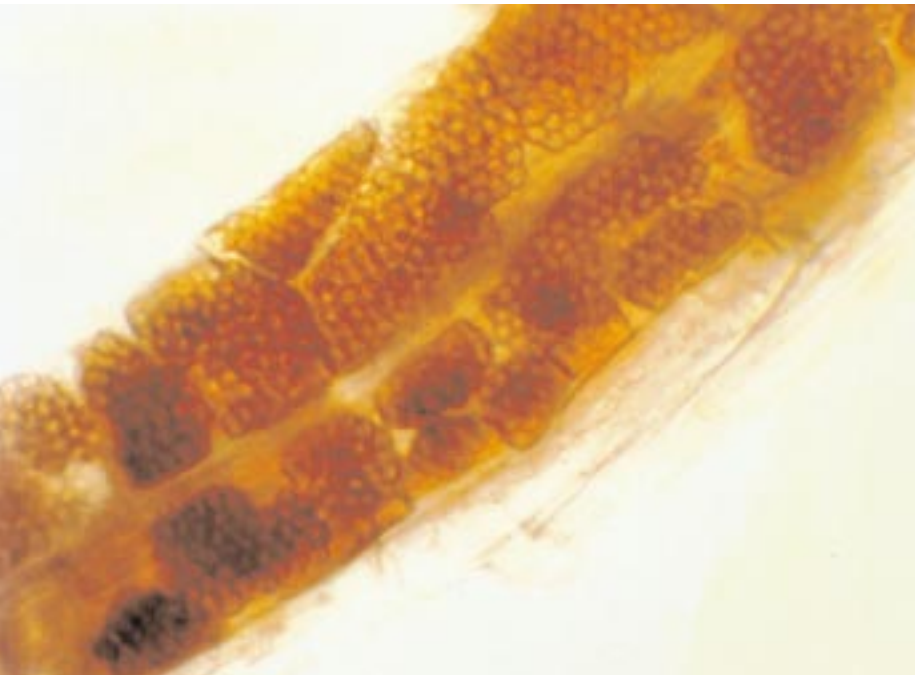


Fig. 4. Cystosori in sugar-beet root cells.  
PHOTO COURTESY MIKE ASHER

are considered essential since evidence from studies of molecular variability within the virus genome suggests that the isolates present in this country are likely to have originated from at least two different areas in continental Europe. Within the UK, extensive surveys are carried out each year and any infected sugar-beet crops, or parts of crops, are destroyed *in situ*. Further sugar-beet cropping on affected fields is prohibited. These measures, along with the very limited use of irrigation on the crop and the disposal of factory waste soil only to non-arable land, have made a substantial contribution to reducing the rate of spread of the disease. Only 4000 hectares, less than 1% of the total UK sugar-beet-growing area, have been affected since the disease was first recorded 12 years ago, and these are largely restricted to the East Anglia region.