SESSION 3B

MANAGEMENT OF FUNGALLY-TRANSMITTED VIRUSES OF ARABLE CROPS

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Papers: 3B-1 to 3B-4

The mosaic viruses of winter barley: problems and prospects

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ABSTRACT

Results of surveys from 1987 onwards show that both *Barley yellow mosaic virus* (BaYMV) and *Barley mild mosaic virus* (BaMMV) are widespread in central, southern and eastern Britain. Losses are difficult to measure accurately but comparisons between groups of susceptible and resistant cultivars on a site with BaMMV over several years suggested that losses of 20-45% occurred in years when the disease was severe. Delayed sowing in the autumn can mitigate the effects of the disease but the use of virus-resistant cultivars is the only reliable strategy. The distribution in the UK of a strain of BaYMV (BaYMV-2) able to overcome the commonly-used resistance gene *rym4* is reported. The prospects for effective disease management using other sources of complete or partial resistance are reviewed.

INTRODUCTION

Mosaic viruses affect winter barley crops in most temperate areas of Europe and East Asia. Symptoms appear in winter or early spring as irregular, yellow patches in the crop and affected plants have small yellow streaks on the youngest leaves. In some cultivars a chocolate-brown necrosis also occurs and the plants may assume a spiky appearance. As temperatures rise in late spring, symptoms become less obvious and they are rarely seen on the upper leaves. Infected plants may or may not be stunted but yield is usually decreased and grain quality can also be affected. There are two viruses that can cause mosaic disease either alone or together and these are named *Barley yellow mosaic virus* (BaYMV) and *Barley mild mosaic virus* (BaMMV). The viruses have similar particle morphology and genome organisation and are classified in the genus *Bymovirus* of the family *Potyviridae*. Both are transmitted by *Polymyxa graminis*, an obligate root-infecting parasite (order *Plasmodiophorales*) that is common in barley roots and which forms clusters of resting spores that remain in the soil for many years. The spores germinate to produce zoospores (swimming spores) that infect seedling roots in the autumn (see Figure 1) and the viruses are introduced at the same time. Leaf symptoms do not occur until after the plants have been stressed by cold weather. The degree of damage is probably linked in part to the severity of the winter. Because of the biology of the vector, the disease is soil-borne and very persistent, making it extremely difficult to control. This paper summarises our current knowledge of the distribution and damage done by the disease and reviews the progress and prospects for disease control, with particular reference to the UK.

DISTRIBUTION

The disease was first reported from Japan in 1940 and was thought to be confined to Asia until the late 1970s. Several European countries reported the presence of mosaic virus between 1978

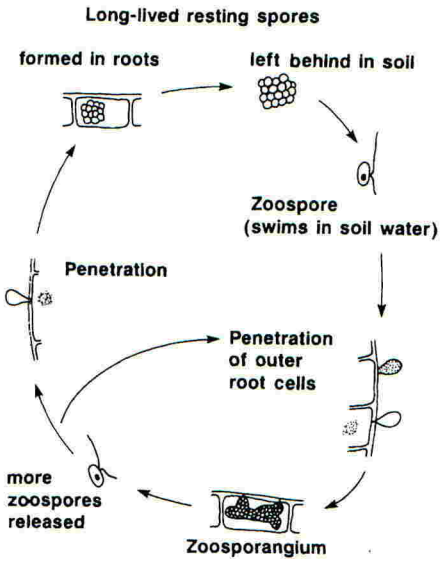

Figure 1. Diagram illustrating the life cycle of *Polymyxa graminis*, the vector of the barley mosaic viruses.

and the mid 1980s and the disease is now known to be widespread in Japan, Eastern China, Korea, Belgium, UK, France, Germany and Italy. It has also been reported from Greece and the Ukraine. All outbreaks were initially described as BaYMV but virus characterisation in Europe demonstrated that BaMMV was a distinct virus (Huth & Adams, 1990) and the two can be readily distinguished by serological tests such as enzyme-linked immunosorbent assay (ELISA). BaMMV was named because it appeared to give milder symptoms on some cultivars, but subsequent results have shown that its effects on other cultivars can be severe (see below). Although originally reported from Europe, BaMMV has since been discovered in Japan and at one site in China.

Annual surveys were begun in 1987 to determine the distribution and relative frequency of the two viruses in the UK, to detect regional or cultivar differences and to monitor the development of resistance-breaking strains. Plants with symptoms were received from farmers and advisors and leaves were tested by ELISA for the presence of both viruses as described by Adams (1991). The total numbers of samples and the viruses detected in each year are shown in Figure 2. Especially large numbers of samples were received in 1991, 1992, 1996 and 1997, when the winters were relatively protracted and cold (data not shown). BaMMV was slightly more common than BaYMV during the period 1991-1996 but seems to have been less frequent recently. The relatively small numbers of samples containing both viruses suggests that there is competition rather than synergism between them. Both viruses were widespread in central, southern and eastern Britain, the regions where most winter barley is grown although BaYMV was slightly more frequent in the west (Figure 3). A few samples infected with BaMMV were also received from Eastern Scotland.

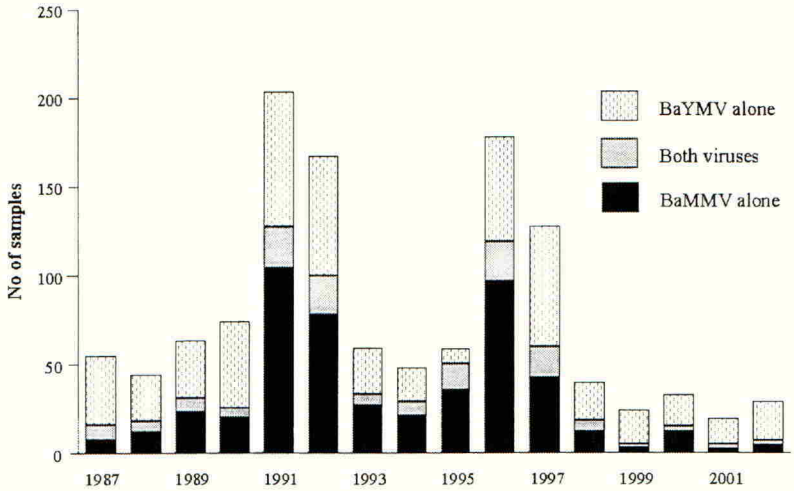

Figure 2. Numbers of mosaic virus samples received, 1987-2002.

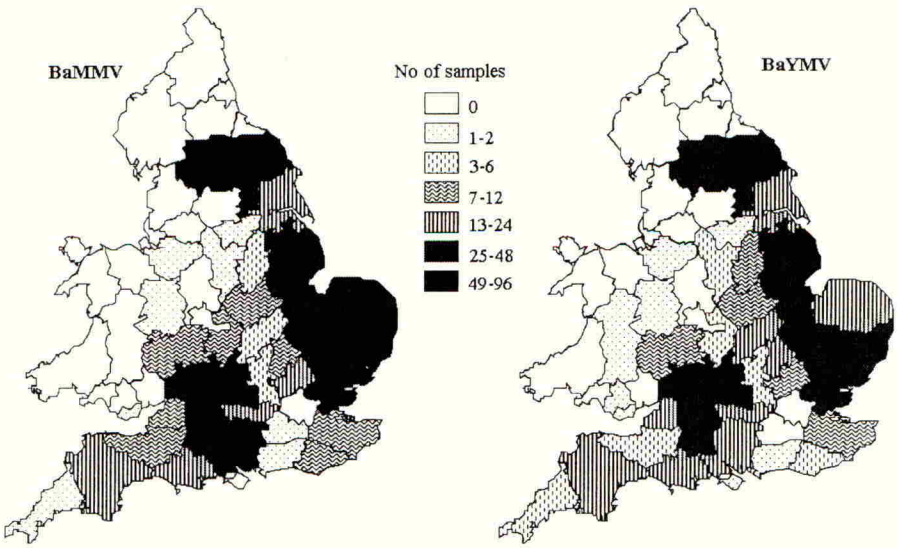

Figure 3. Maps of England and Wales showing the numbers of samples of BaMMV and BaYMV received from each region, 1987-2002.

EFFECTS ON YIELD

Measurement from infected sites in Belgium, Germany and the UK during the 1980s suggested that yield reductions of up to 50% could occur on susceptible cultivars but this is probably the maximum damage in years where climatic conditions were particularly favourable to the disease. It is difficult to measure yield loss accurately because of the patchy distribution of the disease and the difficulty of getting appropriate uninfected controls. However, approximate data can be obtained from variety trials where several susceptible cultivars can be compared

with resistant ones (that develop no disease). This helps to average out the differences between cultivars that would be expected even under virus-free conditions. Results from such experiments in the UK using a site in Gloucestershire where only BaMMV was present are summarised in Table 1. Yields on this drought-prone site were often low, but losses in the region of 20-45% occurred in years when disease was severe.

Table 1. Average disease incidence (% plants infected) in groups of three to six susceptible cultivars and average grain yields from these (S) and similar numbers of resistant cultivars (R) on a site infested with BaMMV in 9 seasons. (Data from Adams et al., 1992; 1996; Overthrow et al., 1999).

		Yield (t ha^{-1})		
Sown	Disease	S	R	% difference
03-Oct-88	90	6.16	8.13	24.3
27-Sep-89	73	3.63	4.86	25.2
15-Oct-90	80	5.39	7.23	25.4
09-Oct-92	16	5.08	5.54	8.4
22-Sep-93	88	4.47	5.62	20.4
23-Sep-94	91	3.16	4.51	29.8
20-Sep-95	97	4.30	7.71	44.2
20-Sep-96	67	3.30	5.54	40.4
23-Sep-97	59	5.95	7.03	15.3

Comparable experiments at a site infested with BaYMV were less easy to interpret because there was often less disease and distribution was not uniform. The best estimates indicated yield differences averaging 15-17% but this is probably an underestimate and may not reflect significant differences between the two viruses in the damage caused. Potential losses may be greater in Germany and France than in the UK, because of the typically more severe winters.

CONTROL

Various attempts to control the disease or mitigate its effects have proved ineffective or uneconomic. These include the application of fungicides to seed or soil and the adjustment of nitrogen applications. Soil sterilisation can be effective but is uneconomic and environmentally undesirable. Crop rotation should have some potential and there is evidence that disease is often more severe where barley has been grown consecutively for many years. In experiments, however, a three year break in winter wheat (which is not a host of either virus) had no measurable effect on inoculum levels of BaMMV in soil or on disease in a subsequent susceptible barley crop (Adams et al., 1992). Some mitigation of the disease and its effects may be obtained by delayed sowing as shown by some results from a BaMMV site (Figure 4). Most plants of the susceptible cultivars became infected when sown in September, but many fewer plants sown in mid-October were infected and almost none of those sown in early November. This probably reflects the diminishing opportunities for the vector, *P. graminis*, to infect roots as the temperatures decrease during the autumn. As expected, yields of resistant cultivars (which were disease-free) were greatest from the first sowing date, declining steadily as sowing was delayed. In contrast, yields of the susceptible cultivars at the first sowing date

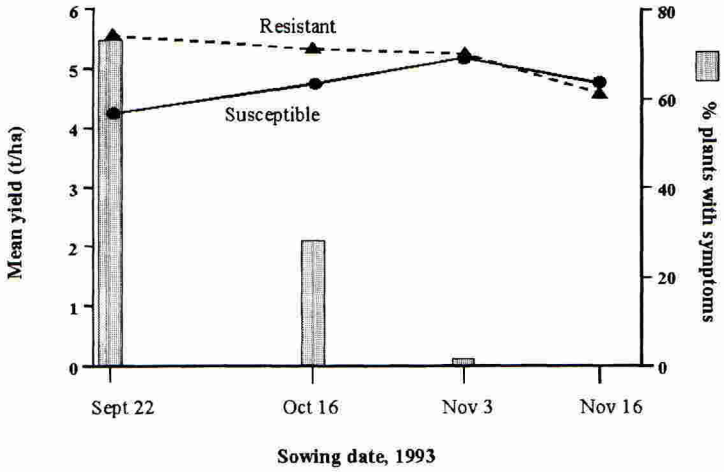

Figure 4. Mean yields of 4 susceptible and 3 resistant winter barley cultivars, and mean disease incidence on the susceptible cultivars, after sowing on 4 dates on a site infested with BaMMV.

were the lowest of any of the dates and the highest yields were at the third (Nov 3) sowing although they never reached those of the resistant cultivars at the earliest sowing. However, these effects depend upon weather conditions after sowing and have therefore not been consistent between seasons. For this reason, and because farmers are reluctant to risk a delay when soil conditions are suitable for drilling, manipulation of sowing date has limited potential as a control measure.

In practice, cultivar resistance provides the only reliable control. The resistance genes so far deployed usually confer immunity to the cultivars and in Europe almost all the resistant cultivars that have been released have (or probably have) the *rym4* gene. Through the 1980s, this provided good resistance to both BaMMV and BaYMV but since 1998 there have been increasing numbers of reports of these cultivars succumbing to a resistance-breaking strain of BaYMV, sometimes called BaYMV-2. This strain has become very widespread in France and Germany but is so far rather limited in its distribution in the UK (Figure 5).

There are no published data on yield losses from BaYMV-2 but there are some indications that it may be less damaging than the wild-type strain. There are no winter barley cultivars on the current UK Recommended List that are resistant to BaYMV-2 and farmers with this strain have either to accept the losses or avoid growing winter barley.

Plant breeders screen their winter barley lines for resistance to mosaic viruses. Some use sap (mechanical) inoculation of seedling leaves with BaMMV to screen out susceptible lines at an early stage but there is no easy laboratory test for resistance to BaYMV and field screening is always necessary, at least at the later stages of cultivar development. Although most studies and recommendations have concentrated on the genes conferring immunity, there is evidence for a degree of partial resistance amongst some other cultivars, e.g. the UK cultivar Sprite (Adams, 1994). This has not been deliberately sought in breeding programmes and its genetic basis is unknown. Results from the surveys reported above also indicate a strong association between cultivar and virus, with BaMMV much more common on a group of malting cultivars

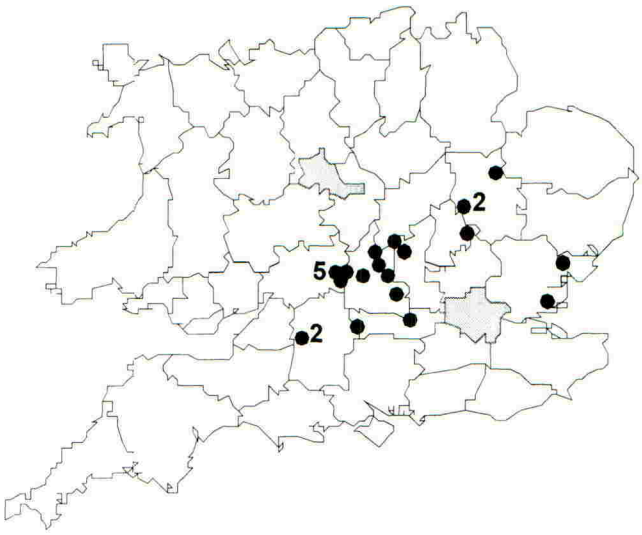

Figure 5. Map of southern Britain showing the locations of the 25 reported outbreaks of BaYMV on cultivars containing the *rym4* gene.

that share a common pedigree (Table 2). This suggests that if it is known what virus is present on a particular field, it may be possible to adjust the cultivar accordingly and minimise losses even in the presence of disease.

Table 2. The incidence of BaMMV and BaYMV on different groups of susceptible cultivars.

Cultivars	BaMMV alone	BaYMV alone	Both viruses	Total samples	Comment
Selected malting*	266	27	41	334	92% have BaMMV
Others	87	385	73	545	84% have BaYMV

*Flute, Halcyon, Maris Otter, Optic, Pipkin, Puffin, Spice

PROSPECTS AND FUTURE DEVELOPMENTS

Control of this disease is likely to depend on the use of resistant cultivars for the foreseeable future. No natural source of resistance to the vector has been detected in plants that can be readily crossed with barley and current work has therefore focussed on resistance to the virus. From the European perspective, it is clearly unwise to rely on a single resistance gene that has already been overcome. In Japan, several different strains of BaYMV and two of BaMMV have been identified from the responses of differential cultivars. Plant breeders are now mapping and characterising a number of different resistance genes (Table 3) and some of these are currently being used, singly or in combination, in programmes to produce new barley cultivars. If these resistances are to be deployed effectively, it will be important to understand their mode(s) of action. Earlier work with *rym4* cultivars demonstrated that this resistance was an immunity to the virus; fungus spores produced on the resistant varieties were virus-free and

inoculum introduced into the leaves soon decayed and became undetectable (Adams *et al.*, 1987; Schenk *et al.*, 1995). Work is now in hand to see whether similar mechanisms operate with the other genes. This is important because the use of a resistance gene that allowed the virus to multiply in the roots but without moving to the shoots, for example, could result in a continued increase in virus inoculum in the field. Alternatively, if resistance was expressed only in the roots, mechanical inoculation of BaMMV to shoots could not be used as a screening mechanism by plant breeders.

Table 3. Resistance (R) or susceptibility (S) of lines carrying different resistance genes to some of the known races of BaYMV and BaMMV.

Gene	Source	European BaYMV		Japanese BaYMV				BaMMV
		-1	-2	I	II	III	IV	
rym1	Mokusekko 3	R	R	R	R	R	?	R
rYm2	Mihori Hadaka 3	R	R	R	S	R	?	R
rym3	'Ea 52'	R	R	R	R	R	S	S
rym4	Ragusa	R	S	?	?	?	?	R
rym5	Misato Golden Resistant Ym No.1	R	R	R	R	S	?	R
rym5'	Iwate Omugi 1	S	S	S	S	S	?	R
rym6	Prior	?	?	S	R	S	S	S
rym7	HHor 3365	S	S	S	S	S	?	R
rym8	'10247'	R	S	?	?	?	?	R
rym9	Bulgarian 347	S	S	S	S	S	?	R
rym10	Hiberna	R	R	?	?	?	?	S
rym11	Russia 57	R	R	?	?	?	?	R
rym12	Muju Covered 2	R	?	?	?	?	?	R

Plant breeders are increasingly using molecular methods such as microsatellite and RFLP markers to assist selection of resistant breeding lines. Further improvements in methodology can be expected if genes are cloned and sequenced.

Amongst the farming community, the disease still sometimes goes unrecognised and may be confused with manganese deficiency or water-logging. There needs to be a greater awareness of the disease and better information on the different types of resistance available. A test kit based on lateral flow technology has recently been developed and this should provide the welcome facility to diagnose and distinguish BaYMV and BaMMV rapidly in the field.

Finally, there remains the prospect that a biotechnological approach could introduce novel types of resistance to the viruses and/or their vector into barley cultivars. Given the current political and economic constraints, this option is not, as far as is known, being actively pursued at present. However, the research community is continuing experiments to characterise the viruses and their vectors and to determine the biochemical and molecular interactions that occur between them and their plant host. Such studies offer the prospect of devising novel

strategies that interfere with some vital component of the virus life-cycle. If such strategies were to prove effective against some or all of the other viruses transmitted in a similar way by plasmodiophorid vectors, the rewards could be considerable.

ACKNOWLEDGEMENTS

I am grateful to many farmers and consultants, and colleagues in ADAS, CSL, NIAB and the Arable Research Centres for their help in supplying samples and for collaboration in field trials. Work reported here was supported by grants from the UK Home-Grown Cereals Authority and MAFF (now DEFRA). IACR receives grant-aided support from the Biotechnology and Biological Sciences Research Council of the United Kingdom.

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The development of sugar-beet rhizomania and its control in the UK

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ABSTRACT

Sugar-beet rhizomania disease, caused by *Beet necrotic yellow vein virus*, was first recorded in the UK in 1987 and has since been detected on *c.* 7,300 ha on 211 farms. Its development has been slow, and the area affected is small, compared to other countries where it occurs. So far the disease has been found predominantly on loamy sands in the intensive production areas of East Anglia although, in recent years, outbreaks have started to appear in other regions and on heavier soil types. Its early appearance on sandy soils may be due to their capacity to warm up more rapidly following sowing. Measures to slow the spread of the disease, administered as part of the UK's Protected Zone status for rhizomania, have included extensive annual surveys to detect the disease, *in situ* destruction of affected parts of crops and prohibition of beet growing on infested fields. In recent years cultivars with high levels of partial resistance and which are otherwise suited to UK conditions have been successfully trialled. With the loss of Protected Zone status in April 2001 statutory controls have been replaced by voluntary measures, with increasing reliance on the use of resistant cultivars. This trend is likely to continue as cultivars are further improved.

INTRODUCTION

Sugar beet is grown annually on 170K ha in the UK, mainly on the light textured soils in the eastern part of the country. The crop is grown under contract by 7,100 growers, each with an individual quota, for processing at one of eight factories. A minimum two years break between crops is prescribed in the contract; the average over all growers is 3.5 years. Rhizomania disease, caused by *Beet necrotic yellow vein virus* and transmitted by the obligate root parasite, *Polymyxa betae* Keskin, was first detected in the UK in 1987 (Hill & Torrance, 1989), following its discovery in many other countries worldwide (Asher, 1993; 1999). This paper describes the progress of the disease in the UK since then, and measures adopted to slow its spread and reduce its impact.

THE DEVELOPMENT OF RHIZOMANIA IN THE UK

Prior to April 2002, rhizomania was a notifiable disease under statutory control and extensive aerial and ground surveys were carried out annually by MAFF/DEFRA to detect fields showing symptoms. Figure 1 shows the number of new farms on which the disease was detected each year. Years with a particularly high number of newly affected farms tended to be those in which soil temperatures recorded at IACR-Broom's Barn were significantly higher than the long-term average at the time the crop was sown (Asher, unpublished). This was particularly so in 2001 when the crop was sown, on average, one month later than normal. The

optimum temperature for both vector and virus multiplication is 25°C so warm soil conditions are conducive to early infection and symptom development. Clearly, however, there is also an underlying trend of increasing disease incidence over years.

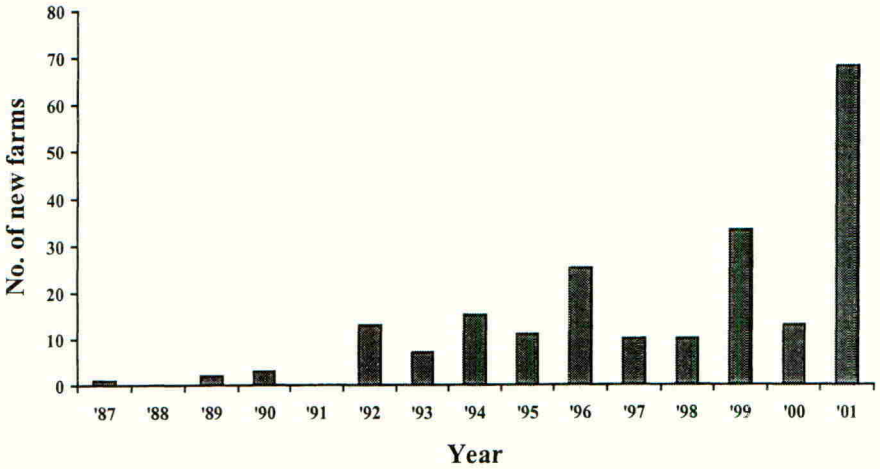

Figure 1. Number of newly affected farms detected in the rhizomania survey each year.

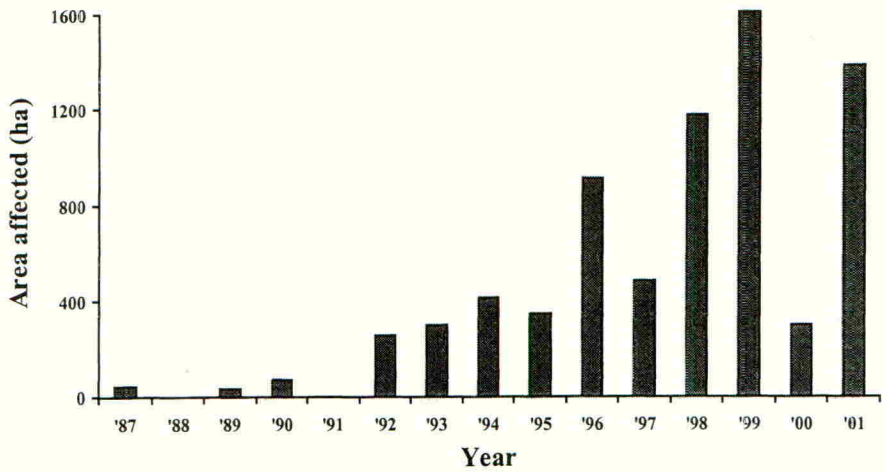

Figure 2. Total area of fields newly affected with rhizomania each year.

The total area of fields affected by the disease each year is shown in Figure 2 and, under the policy prohibiting further beet growing on infested fields, this represents the area subsequently taken out of beet cultivation, currently *c.* 7,300 ha in total. However, in most cases in the UK the areas showing symptoms in crops have been relatively discrete patches. The apparent drop in area affected in 2000 coincided with the introduction of a scheme (the Rhizomania Stewardship Scheme) allowing affected growers to lease their annual quota to those in rhizomania-free areas, rather than growing it elsewhere on a contaminated farm. Up to then, *c.* 50% of the area affected each year consisted of fields on already contaminated farms. Despite the underlying increase with time, the area affected by rhizomania in the UK is extremely

small when compared with most countries in continental Europe, where over 700K ha were infested in 2000 (Richard-Molard & Cariolle, 2001). This is illustrated by comparison with annual survey data from France (Figure 3) where *c.* 45% of the sugar beet growing area is affected each year. In contrast, development in the UK has been slow and the cumulative affected area is currently only *c.* 1% of all beet growing land.

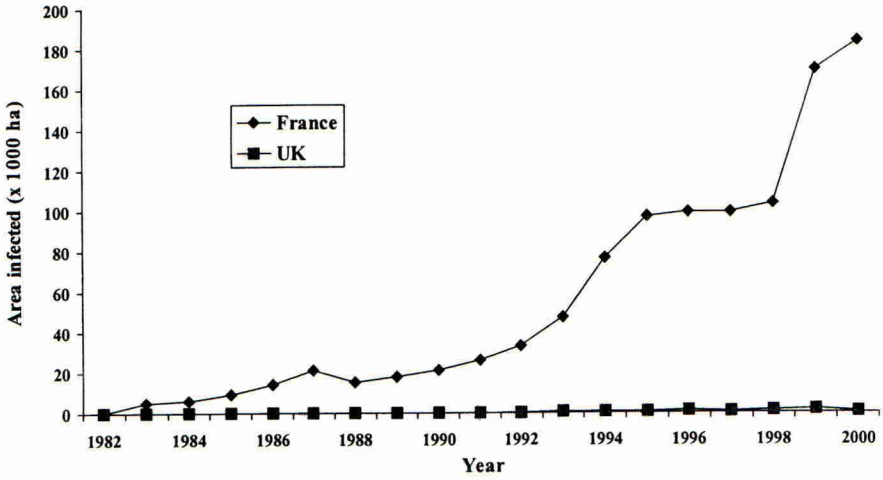

Figure 3. Annual area affected by rhizomania in France and the UK (data for France from Annual Reports of the ITB, 1984-2001).

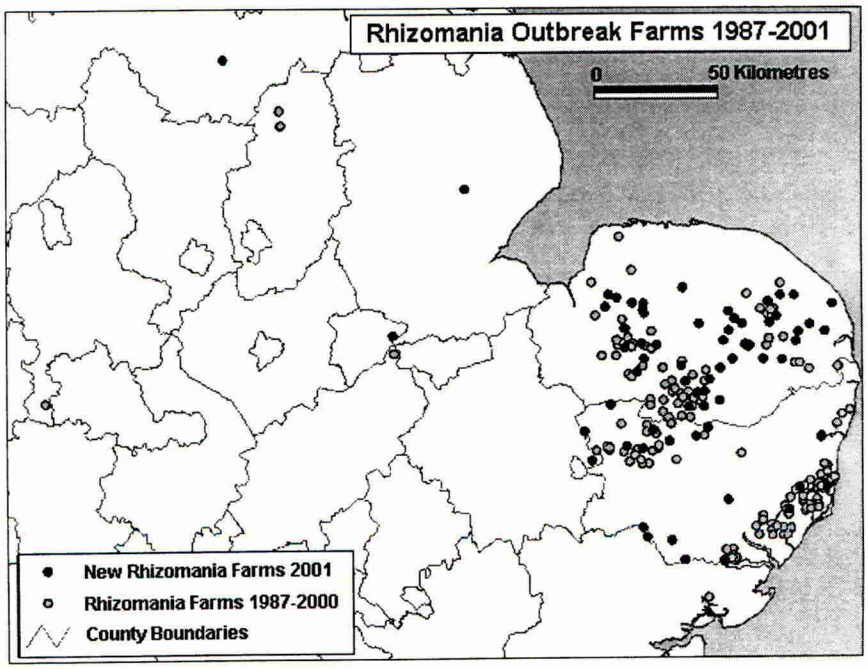

Figure 4. The location of rhizomania affected farms in the UK, 1987-2001 (Courtesy CSL, York and DEFRA).

The distribution of rhizomania affected farms is shown in Figure 4. To date, the disease has been found predominately on the light sandy soils (loamy sands) in the areas of most intensive production (Norfolk and Suffolk) in eastern England. Isolated outbreaks have also been detected on similar soils in other regions, e.g. Shropshire, Nottinghamshire, Lincolnshire and South Yorkshire. Figure 5 illustrates the close association with soil type in the UK, a feature that is not apparent in continental Europe. However, in recent years the disease has begun to show up on heavier (sandy loam, sandy clay loam) soils in East Anglia. A possible reason for the association with sandy soils is shown in Figure 6. In this experiment, soils of different textural classification (soil type) were collected and compared at one location for the rate at which they warmed during the average period of seed germination and early root growth in sugar beet (Webb *et al.*, 2000). The loamy sands warmed up significantly more rapidly than other soil types, with the black fen peats being particularly cold. Sandy soils may thus be more conducive to early infection, and hence to more rapid inoculum multiplication and disease development in successive sugar-beet crops in the rotation, than other soil types. According to this hypothesis these other soils, which are no less likely to have been contaminated with the disease, will take longer to show symptoms.

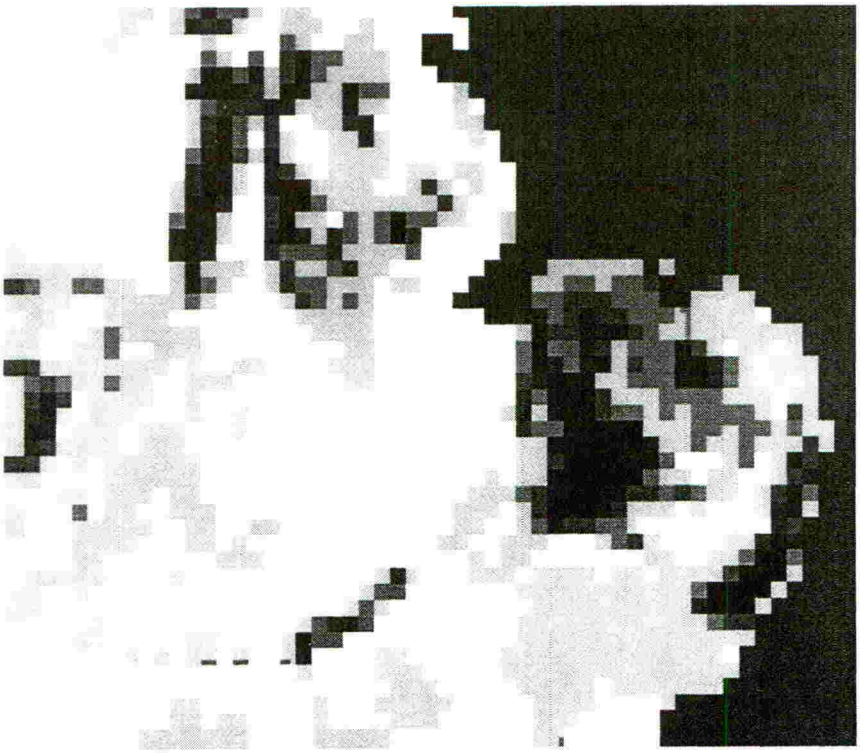

Figure 5. The distribution of soils with > 70% sand content. Increased intensity of shading indicates increased proportion of fields with these soil types within 5 x 5 km grid. Data source @ NSRI, Cranfield University, 2002.

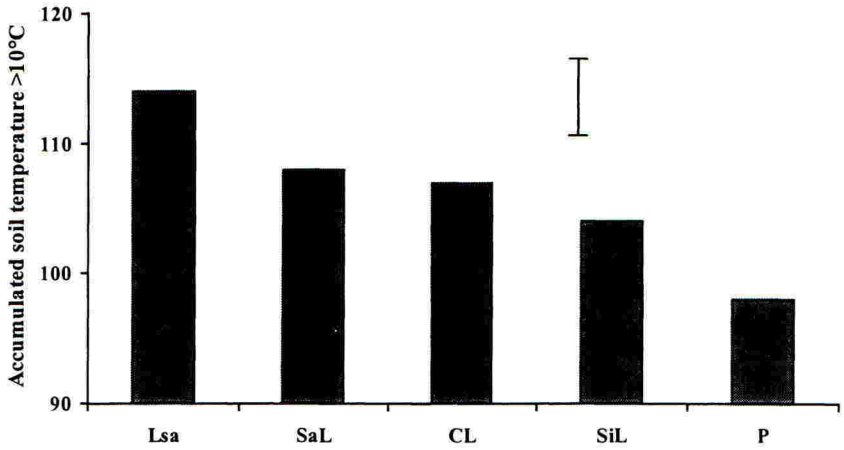

Figure 6. Accumulated hourly soil temperature (above a base temperature of 10°C) at 5cm depth in soils of different textural classifications; 29 March – 17 May 1993. LSa = Loamy sand, SaL = Sandy loam, CL = Clay loam, SiL – Silty loam, P = Black fen peat. Bar = 5% LSD between soil types.

MEASURES TO SLOW DISEASE SPREAD

Under the statutory containment policy, measures to slow the spread of rhizomania in the UK have evolved along with the development of the disease. Initial, draconian measures, such as methyl bromide fumigation and grassing down of affected fields, plus the prohibition of all root crops on the farm, have given way to a more relaxed policy with less impact on farming practices. The key measures in place in 2001 can be summarised as:

- Controls on imported planting material and soil
- Surveys to detect the disease
- Destruction of infected part of crop + compensation
- Delivery of remainder to tidal discharge factories
- Cropping controls on affected fields
- Factory waste soil disposal to non-beet land
- Rhizomania Stewardship Scheme

Under the UK's Rhizomania Protected Zone status within the EU, plant material carrying soil (e.g. seed potatoes) from rhizomania affected countries was subject to inspection and control. At the same time surveys to detect the disease, destruction of affected areas in crops (by herbicide treatment) and delivery of the remainder of the crop to coastal processing factories (to avoid contaminating inland waterways) were required by DEFRA. No further beet cropping was permitted on affected fields, the use of partially resistant cultivars being restricted to non-affected fields on farms that had already recorded the disease. In conjunction with this, the industry implemented schemes to compensate affected growers for crop losses

and to encourage the movement of beet growing away from affected areas (the Rhizomania Stewardship Scheme).

It is impossible to quantify the contribution that these measures may have made to the slow rate of disease in the UK. Our relatively cool temperature climate, with soils that are cooler or warm up more slowly following sowing than in continental or Mediterranean climates, is likely also to have contributed to this. Recent mathematical modelling of the temporal and spatial development of rhizomania (Gilligan *et al.*, unpublished) has strongly implicated machinery as the main route by which the disease is spread in the UK. Over 50% of sugar-beet crops are now harvested by contractors moving from farm to farm. The models also highlighted the difficulty of containing the spread of a disease which is so highly infectious (only very small amounts of contaminated soil are required to transmit it; Tuitert & Hofmeester, 1992) and has a long incubation period, during which further spread is unwittingly taking place.

PROGRESS WITH RESISTANT CULTIVARS

Selection for partial resistance to rhizomania was initiated in the 1970's but not until ten years later, when the 'Holly' source of resistance was discovered in the USA, was significant progress made by plant breeders. Resistance derived from this source is monogenic and thus more easy to manipulate in plant breeding programmes, and is expressed at substantially higher levels than previous sources. In the past five years this resistance has been deployed in sugar beet cultivars throughout Europe and the USA; in Europe alone such cultivars are currently grown on 690K ha (Richard-Molard & Cariolle, 2001). In the UK, however, the use of resistant cultivars to control rhizomania has not been widely adopted, both because their performance was considered inadequate under our conditions and because they would undermine the ability of surveys to detect the disease, an essential component of the containment policy.

In trials, the best resistant cultivars give very substantial yield increases on severely infested sites, compared with susceptible cultivars (Asher *et al.*, 2002). However, there has been concern that they suffer a significant yield penalty in the absence of the disease, compared with the best, widely grown susceptible cultivars. This is of particular significance in the UK, where the disease tends to be relatively restricted in its distribution, both within farms and within affected fields. In addition, other agronomic features required for the UK environment, such as resistance to bolting in our cool springs, and low levels of the impurities in roots which impede sugar extraction, have been lacking in resistant cultivars.

However, recent improvements in these traits have led to the recommendation of three resistant cultivars for use in 2003; Concept, Rayo and Dorena. Table 1 summarises their performance in trials, both in the presence and absence of the disease. Over three years of NIAB trials on a total of 41 rhizomania-free sites these resistant cultivars averaged 102% of the yield of the standard susceptible control cultivars, though only 97% of the highest yielding cultivar available. In two trials on severely infested sites, where yields of conventional cultivars were reduced by 70%, resistant cultivars were giving yields approximately 8-10% less than in the absence of disease (Asher, *et al.*, 2002). Site and soil differences are confounded with these comparisons and it is possible that the sandy soils on which rhizomania trials are conducted, and which are more nutrient depleted and drought-prone, are contributing to this yield penalty.

Table 1. Summary of yields of current rhizomania resistant and susceptible sugar-beet cultivars in the presence and absence of the disease (as percentage of those of susceptible cultivars on uninfested sites). Data from NIAB and IACR-Broom's Barn trials.

Cultivars	Sites without Rhizomania	Sites with Rhizomania
Susceptible	100	30
Resistant	102-97	90-92

FUTURE DEVELOPMENTS

Though rhizomania has been slower to develop in the UK, it is clear from the limited series of trials conducted on infested sites so far that, once established, it has the potential to be as damaging as it has been in other countries. Loss of Protected Zone status and statutory controls, largely as a consequence of the increased number of outbreaks in 2001, has precipitated greater reliance on resistant cultivars as a means of combating the disease. This has coincided with the development, trialling and subsequent recommendation of some much improved resistant cultivars suited to UK conditions and processing requirements. Surveys to detect the disease, sponsored by the industry, are to continue, not least to determine the distribution of the potentially more pathogenic 'P' strain of the virus, detected for the first time on two farms last year (Henry, C. M., personal communication). Diseased crops will in future not be destroyed, unless they are found to be infected with the 'P' strain, but instead delivered to the factory during a specified period in order to minimise contamination of other growers. Growing beet on known infested fields is no longer prohibited and the move from disease containment to control through the use of resistant cultivars is likely to be the prelude a major change in cultivar use in sugar beet in future years.

ACKNOWLEDGEMENTS

I am grateful to DEFRA's Plant Health and Seeds Inspectorate and the Central Science Laboratory, York for supplying data and to the British Beet Research Organisation for financial support.

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Assessment of the resistance of UK winter wheat varieties to the diseases caused by *Soil-borne wheat mosaic virus* and *Wheat spindle streak mosaic virus*

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ABSTRACT

Soil-borne wheat mosaic virus (SBWMV) and *Wheat spindle streak mosaic virus* (WSSMV) cause serious diseases of winter wheat. Prior to this work little was known about the resistance of UK winter wheat varieties to either virus. Replicated field trials were set up in 5 fields in France and Italy to assess the resistance of 18 varieties of UK winter wheat to infection by either SBWMV, WSSMV or a combination of both viruses. The majority of the UK varieties tested became heavily infected with SBWMV showing typical symptoms, including leaf streaking and stunting. Few or no foliar symptoms were seen on varieties Aardvark, Charger, Claire, Cockpit and Hereward. Subsequent serological testing confirmed these varieties to be resistant or partially resistant to SBWMV. At a heavily infected site, the average yield of susceptible varieties was reduced by 42% when compared to the average yield of resistant varieties. The count of heads per m^2 at harvest was significantly reduced in susceptible varieties, suggesting SBWMV infection reduces tillering in UK varieties. None of the UK varieties became infected with WSSMV, which strongly indicates that these varieties are resistant to the virus.

INTRODUCTION

Soil borne wheat mosaic virus (SBWMV), the type member of the genus *Furovirus*, was first observed in Illinois and Indiana in 1919 but has spread to at least 16 states and probably occurs throughout the winter wheat growing area of the USA (Brakke & Langenberg, 1988). The virus has since been reported from Canada, South America (Brazil), Asia (China, Japan), Africa (Zambia) and Europe (Denmark, France, Germany, Italy, Poland) (Brakke, 1971; Canova and Quaglia, 1960; Hariri *et al.*, 1987). In France, SBWMV was first reported in 1978 and is now widely distributed in central and western regions. SBWMV is also found throughout much of Italy, in particular in the northern and central areas. The virus was detected in the UK for the first time in 1999 on a farm in Wiltshire (Clover *et al.*, 2001). SBWMV has been found subsequently at two sites in Kent in 2000, and in 2002 on the Isle of Wight and one further site in Wiltshire.

Wheat spindle streak mosaic virus (WSSMV) is another soil-borne virus closely related to the barley mosaic viruses, *Barley yellow mosaic virus* and *Barley mild mosaic virus*. In Europe, WSSMV was first detected in France in 1977 (Signoret *et al.*, 1977), and is now known to occur throughout France, Germany and Italy. WSSMV is often found infecting wheat in combination with SBWMV, but this virus has not yet been reported in the UK.

The symptoms of both diseases depend on the virus strain and host cultivar and include pale green or yellow mosaics on the leaves, stunting, reduced tillering and grain yield. Symptom severity and virus titre is dependent on temperature and can fluctuate during the season. The best visual symptoms generally occur in the spring (February to April in Europe) and disappear as temperatures rise. Both viruses cause serious yield losses in susceptible varieties of winter wheat. Losses of up to 56% have been recorded in cases of mixed infection in durum wheat (V. Vallega, pers. com.). Trials at the infected site in Wiltshire have shown that SBWMV can reduce grain yield in susceptible UK cultivars of winter wheat by up to 50% (Clover *et al.*, 2001).

Both viruses are transmitted by the soil-borne plant parasite *Polymyxa graminis* Led., as are the soil-borne viruses of barley. The virus survives in the absence of host plants in the resting spores of *P. graminis*, which can remain viable in the soil for at least 15 years. Experience of the spread of SBWMV in other countries, together with the spread of barley mosaic viruses in the UK, indicate that there is a high risk of SBWMV and WSSMV becoming widespread in the UK. Once land is infected the only practicable means of control is to grow resistant cultivars. In France, cultivars with virus resistance have been identified (e.g. Cadenza and Tremie) and breeding work is in progress to produce improved cultivars for the future. Prior to this study, little was known about virus resistance in UK varieties of winter wheat. The work presented here is the first resistance data for UK cultivars of winter wheat comparing symptomatology, virus infection and final yield data.

MATERIALS AND METHODS

As the variety trials were started prior to the first UK outbreak of SBWMV, trial sites with a history of viral infection were chosen in France and Italy. Five infected fields were selected with either a single virus or a combination of both viruses during the 1999-2000 season: Ozzano, Italy (SBWMV), Chambon sur Cisse, France (SBWMV and WSSMV), and Landes Le Gaulois, France (WSSMV) and 2000-2001 season: Minerbio, Italy (SBWMV) and Chambon sur Cisse, France (SBWMV and WSSMV). Site management was carried out by the Institut Technique des Céréales et des Fourrages in France and the Università di Bologna in Italy. Resident farmers managed general crop husbandry according to local good farm practice.

Fifteen winter wheat varieties (Aardvark, Buster, Charger, Claire, Consort, Equinox, Hereward, Madrigal, Malacca, Napier, Reaper, Rialto, Riband, Savannah and Shamrock) were selected from the UK recommended and provisionally recommended national lists to represent bread-making, cake/biscuit-making and feed wheats. Poor performing or obsolete varieties (Buster, Reaper and Riband) were substituted with new varieties (Buchan, Cockpit and Eclipse) in the second season.

Local varieties of known resistance to the viruses were incorporated into each field trial to act as controls. In France, these were Aztec (susceptible to SBWMV but resistant to WSSMV), Cezanne (susceptible to both viruses) and Tremie (resistant to both viruses). In Italy, the varieties Grazia and Valnova (both susceptible to both viruses) were used.

A randomised block design with 3 or 4 replicates of each variety was used for all 5 trials. Plot size varied between countries (8m² in France and 10m² in Italy). Planting density was 400 seeds/m² in Italy and 250 seeds/m² in France and inter-row spacing was 17 cm for all sites. All data were analysed using analysis of variance (ANOVA). Friedman's test was used for analysis of symptom severity scores because these data were discontinuous (df 14). Data were correlated as required using Pearson's or Spearman rank correlation as appropriate. Data for local control varieties were removed for all statistical tests.

Symptom severity was assessed several times during the growing season using established disease indices (0-4 scale in Italy and a 0-5 scale in France where 0 = no visible symptoms and 4/5 = strong symptoms including foliar mottling and dwarfing). Assessments were grouped around times of maximum symptom expression. Data from the assessments showing highest foliar disease levels are presented.

The presence of each virus was confirmed using double antibody sandwich enzyme-linked immunosorbent assay (ELISA) essentially as described by Vallega *et al.* (1999). Absorbance values greater than 3 times the mean negative control were considered to be positive. Sampling varied slightly between sites; in France leaves were collected from 10 random plants from each plot and pooled by variety. A sub-sample of 5 leaves was then tested by ELISA. Whole plants were sampled from the site in Chambon Sur Cisse in 2000-2001 as described and roots tested. In Italy, the second youngest leaf was sampled from 10 random plants from each plot. These were pooled and tested using ELISA.

Agronomic performance was evaluated in terms of grain yield (15% moisture in France and 13% moisture in Italy), thousand grain weight (TGW), heading date (Italian trials only) and plant height at harvest. In France, detailed plant emergence data and the number of heads per m² at harvest were recorded for each plot within the same 2 x 0.34m² areas.

RESULTS

SBWMV

Symptom severity scores and ELISA results confirmed that susceptible local control varieties became infected with SBWMV at all sites. The most severe symptoms of SBWMV were evident in both years at the site in Chambon Sur Cisse. Strong foliar symptoms were accompanied by plant dwarfing and yield reductions in susceptible varieties (Table 1 and 3). Varieties Aardvark, Charger, Claire, Cockpit and Hereward showed little or no foliar symptoms on SBWMV-infected land at any of the sites. ELISA confirmed the absence of SBWMV in the leaves of all of these varieties except Aardvark, where the virus was found at low levels (Table 1 and 7). At Chambon Sur Cisse the average yields of resistant and susceptible varieties were respectively 8.21 and 4.76 t/ha in 2000, representing a 42% reduction in yield. The corresponding figures in 2001 were 5.68 and 3.95 t/ha, representing a 30% reduction in yield.

SBWMV infection at the site in Ozzano was mild with infection starting very late in the season. No variety was consistently infected with the virus. Whilst SBWMV uniformly infected susceptible varieties at Minerbio, symptom severity scores remained low through the season. Grain yield for the UK varieties grown on both Italian sites was not badly affected by this low level of virus infection (Table 6 and 7).

Yield positively correlated with plant height and number of heads per m^2, but negatively correlated with symptom severity, for both years at the Chambon Sur Cisse site. Also, plant height at harvest showed a significant negative relationship with symptom severity (Table 4 and 5). Significant differences were observed between varieties for TGW at all sites, including Landes Le Gaulois, irrespective of viral infection.

WSSMV

Symptom severity and ELISA of leaf material from the susceptible control variety (Cezanne) at Chambon Sur Cisse 2000-2001 and Landes Le Gaulois 1999-2000 revealed heavy infection with WSSMV at both sites. No definite symptoms of WSSMV were observed on any UK varieties at either infected site, despite strong symptoms in local susceptible control varieties (Table 2 and 3). WSSMV failed to develop at the trial site in Chambon Sur Cisse in 1999-2000, where Cezanne remained healthy (Table 1).

Table 1. Disease and plant growth measurements for varieties grown at the site in Chambon Sur Cisse 1999-2000 (SBWMV+WSSMV).

Variety	Symptom severity (0-5) 03/05/00	Leaves positive (of 5) ELISA 27/03/00		Yield (t/ha) at 15% Humidity	Height (cm)	Emergence (plants/m^2) 17/09/99	Number of heads/m^2 12/07/00
		SBWMV	WSSMV				
Aztec	5	5	0	4.64	49.6	281.0	375.0
Cezanne	5	4	0	3.46	48.3	265.0	255.9
Tremie	0	0	0	8.23	80.0	262.3	411.3
Aardvark	1.7	1	0	7.43	77.0	301.3	397.5
Charger	0.0	0	0	8.88	75.6	259.7	470.6
Claire	0.0	0	0	8.34	79.7	254.0	421.6
Hereward	0.0	0	0	8.19	80.3	274.0	483.3
Buster	5.0	4	0	5.38	53.4	239.3	344.1
Consort	5.0	4	0	4.83	51.3	257.3	292.2
Equinox	5.0	5	0	2.24	49.7	251.3	231.4
Madrigal	5.0	5	0	4.28	49.0	251.3	368.1
Malacca	5.0	5	0	5.82	51.8	250.7	382.8
Napier	5.0	3	0	4.95	51.6	238.3	353.4
Reaper	4.3	5	0	4.71	57.6	238.7	312.3
Rialto	3.7	5	0	4.93	65.5	277.3	310.3
Riband	5.0	4	0	4.54	55.0	243.0	281.4
Savannah	5.0	5	0	5.33	56.4	242.0	274.5
Shamrock	5.0	4	0	5.35	48.3	232.0	380.4
P-Value (28 df)	<0.001	-	-	<0.001	<0.001	0.09	<0.001
SED	-	-	-	0.472	4.99	19.17	39.67

Table 2. Disease and plant growth measurements for varieties grown at the site in Landes Le Gaulois 1999-2000 (WSSMV).

Variety	Symptom severity (0-5) 03/05/00	Leaves positive for WSSMV (of 5) by ELISA 27/03/00	Yield (t/ha) at 15% humidity	Height (cm)
Aztec	0.0	0	6.30	77.3
Cezanne	5.0	5	5.03	82.3
Tremie	0.0	0	6.45	77.7
Aardvark	0.3	0	4.20	73.0
Charger	0.0	0	5.76	74.0
Claire	0.0	0	4.21	81.7
Hereward	0.0	0	5.23	79.3
Buster	0.0	0	5.30	75.3
Consort	0.0	0	4.93	77.0
Equinox	0.0	0	3.87	72.0
Madrigal	0.0	0	3.60	72.3
Malacca	0.0	0	5.06	78.3
Napier	0.0	0	5.73	74.7
Reaper	0.0	0	3.81	83.0
Rialto	0.0	0	5.16	84.7
Riband	0.0	0	4.87	82.0
Savannah	0.0	0	5.11	80.0
Shamrock	0.0	0	5.38	76.3
P-Value (28 df)	0.450	-	0.015	<0.001
SED	-	-	0.612	2.66

Table 3. Disease and plant growth measurements for varieties grown at the site in Chambon Sur Cisse 2000-2001 (SBWMV+WSSMV).

Variety	Symptom severity (0-5) 29/03/01	Results for SBWMV ELISA 24/04/01 (average of 5 plants) Leaves	Roots	Results for WSSMV ELISA 24/04/01 Leaves	Yield (t/ha) at 15% humidity	Height (cm)
Aztec	4.3	+	+	-	3.35	50.9
Cezanne	4.3	+	+	+	2.72	41.8
Tremie	0.0	-	+	-	5.96	78.3
Aardvark	0.3	-	+	-	5.14	65.0
Charger	0.0	-	+	-	5.77	70.0
Claire	0.0	-	+	-	5.88	70.0
Cockpit	1.3	-	+	-	6.02	80.0
Hereward	0.0	-	+	-	5.59	70.0
Buchan	5.0	+	+	-	2.86	40.2
Consort	4.0	+	+	-	3.52	54.7
Eclipse	4.0	+	+	-	3.86	45.7
Equinox	3.7	+	+	-	3.48	51.8
Madrigal	4.7	+	+	-	3.67	43.9
Malacca	3.0	+	+	-	4.56	50.4
Napier	4.0	+	+	-	4.62	52.8
Rialto	3.0	+	+	-	3.93	41.2
Savannah	4.0	+	+	-	4.20	53.0
Shamrock	3.3	+	+	-	4.83	51.9
P-Value (28 df)	<0.001	-	-	-	<0.001	<0.001
SED	-	-	-	-	0.405	3.53

Table 4. Simple correlations between grain yield (t/ha), symptom severity (0-5) on 03/05/01 and other plant characteristics for varieties grown at the site in Chambon Sur Cisse, 1999-2000.

	Mean	Range	Yield (t/ha)	Symptom severity (0-5)†
Height at harvest (cm)	60	41-82	0.772***	-0.765***
TGW (g)	43.5	36-51	0.078	-0.136
Plant emergence (m²)	254	202-323	0.251	-0.481***
Heads per m²	354	213-559	0.770***	-0.569***
Yield (t/ha)	5.68	1.51-9.42	-	-0.694***

*P=0.05, ** P=0.01,*** P=<0.001

† Data correlation using Spearman's rank correlation

Table 5. Simple correlations between grain yield (t/ha), symptom severity (0-5) on 29/03/01 and other plant characteristics for varieties grown at the site in Chambon Sur Cisse, 2000-2001.

	Mean	Range	Yield (t/ha)	Symptom severity (0-5) †
Height at harvest (cm)	56	33-85	0.799***	-0.748***
TGW (g)	44	37-53	0.318*	-0.494***
Plant emergence (m²)	218	116-297	0.124	-0.024
Heads per m²	245	71-375	0.669***	-0.608***
Yield (t/ha)	4.58	2.47-6.66	-	-0.788***

*P=0.05, ** P=0.01,*** P=<0.001

† Data correlation using Spearman's rank correlation

Table 6. Disease and plant growth measurements for varieties grown at the site in Ozzano 1999-2000 (SBWMV).

Variety	Symptom severity (0-4) 18/04/00	Leaves positive for SBWMV by ELISA on 27/03/00 (of 4 plots)	Yield (t/ha) at 13% humidity	Height (cm)
Grazia	0.75	4	3.67	83.5
Valnova	0.56	4	4.45	82.3
Aardvark	0.38	0	4.99	70.5
Charger	0.13	0	5.35	69.0
Claire	0.31	0	4.60	72.5
Hereward	0.13	0	4.71	70.5
Buster	0.19	1	4.65	65.3
Consort	0.46	0	4.91	66.3
Equinox	0.19	0	5.15	67.3
Madrigal	0.31	1	4.54	63.3
Malacca	0.19	0	4.70	75.0
Napier	0.25	2	5.03	70.5
Reaper	0.13	0	4.89	74.8
Rialto	0.25	2	4.81	80.8
Riband	0.69	1	3.95	72.5
Savannah	0.69	0	4.29	61.0
Shamrock	0.00	0	5.11	71.0
P-Value (28 df)	0.107	-	<0.001	<0.001
SED	-	-	0.255	2.37

Table 7. Disease and plant growth measurements for varieties grown at the site in Minerbio 2000-2001 (SBWMV).

Variety	Symptom severity (0-4) 05/03/01	Leaves positive for SBWMV by ELISA on 21/03/01 (of 4 plots)	Yield (t/ha) at 13% humidity	Height (cm)
Grazia	2.75	4	3.80	72.5
Valnova	3.00	4	2.02	71.8
Aardvark	0.50	1	8.19	85.0
Charger	0.13	0	6.85	83.8
Claire	0.00	0	8.29	92.5
Cockpit	0.10	0	6.47	112.5
Hereward	0.25	0	6.24	86.3
Buchan	1.00	2	7.34	79.0
Consort	1.13	2	3.90	79.5
Eclipse	1.19	1	7.27	86.3
Equinox	0.94	3	7.78	76.3
Madrigal	0.63	2	6.71	78.6
Malacca	0.56	1	5.29	85.0
Napier	1.06	4	7.03	81.0
Rialto	0.38	1	6.20	96.3
Savannah	0.88	1	4.99	82.0
Shamrock	0.06	1	2.56	79.3
P-Value (28 df)	<0.001	0.05	<0.001	<0.001
SED	-	0.2321	0.395	2.27

DISCUSSION

The majority of UK winter wheat varieties tested during the study were susceptible to infection by SBWMV in France and Italy. At heavily infected sites, e.g. Chambon Sur Cisse in 1999-2000, virus infection significantly reduced the average yield for susceptible varieties by 42% when compared to the average yield for resistant varieties. Furthermore, since the plants were not infected with WSSMV, this yield loss can be attributed to SBWMV infection alone. This large yield reduction was caused by a significant reduction in the number of heads per m^2. For example, at Chambon Sur Cisse, yield was positively correlated with the number of heads per m^2 and negatively correlated to symptom severity in both seasons. These results suggest that SBWMV reduces tillering in UK varieties of winter wheat. Previous studies have implicated a reduction in tillering as the main cause of yield loss in SBWMV-infected wheat (Kucharek and Walker, 1974; Campbell *et al.*, 1975).

At the two sites in Italy, UK varieties were not challenged sufficiently with the virus to determine resistance status. However, symptomatology and ELISA data correlated with the results obtained at the sites in France, showing that the cultivars Charger, Claire, Cockpit, Hereward, were consistently resistant to SBWMV infection. Aardvark, whilst susceptible to SBWMV infection, showed little or no reduction in yield suggesting this variety is partially resistant.

The origin of resistance to SBWMV is unknown, although the variety Moulin has been implicated as a possible resistance source. The mechanisms for resistance are also unclear, however, the lack of virus in leaf material of resistant varieties suggests resistance may act by

reducing virus movement to the aerial parts. Driskel *et al.* (2002) recently concluded that virus resistance in hard red winter wheat probably operates in the roots to block virus movement to the leaves. Clearly there is a need to research resistance mechanisms more thoroughly in the future. The large variation in virus infection between sites, even between the same site in different years, highlights the importance of the environment (e.g. temperature, soil moisture etc.) in determining whether severe disease will occur. Future work will analyse meterological data collected at these sites and also replicate these trials on an infected site in the UK, to investigate whether similar yield reductions can be recorded in the UK climate.

All 18 UK varieties tested are resistant to WSSMV. The virus was not detected in the leaves or roots of any of the varieties. It has previously been observed that a mixed infection of SBWMV and WSSMV can enhance viral infection and break down varietal resistance to SBWMV (Brakke & Langenberg, 1988) but this was not found to be the case in these trials.

ACKNOWLEDGEMENTS

We thank the Home Grown Cereal Authority (HGCA) for funding this work (project no 2182) and Victor Vallega for critically reading the manuscript.

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Potential for chemical control of *Spongospora subterranea*, cause of powdery scab of potatoes and vector of *Potato mop-top virus*

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ABSTRACT

In a series of trials across northeast Scotland, the control of powdery scab incidence and severity was highly variable with both seed tuber and soil treatments. Maneb plus zinc oxide seed treatment was more effective in controlling severity than incidence. Soil treatment with fluazinam at high doses was more consistent. An inability to quantify inoculum inhibits effective understanding of the variability in trial data. Chemical treatment alone is insufficient for the control of powdery scab and *Potato mop-top virus* and should be integrated with other control measures.

INTRODUCTION

Powdery scab, caused by the plasmodiophorid *Spongospora subterranea f.sp. subterranea,* is a serious disease of potatoes in northern Europe and elsewhere in the world (Wale, 2001). *S subterranea* is also the only vector of *Potato mop-top virus* (PMTV). This virus causes brown arcs or lines in tubers of susceptible potato cultivars. *S. subterranea* survives for many years as cystosori, aggregations of spores. When stimulated to germinate, spores within the cystosori germinate to release a biflagellate zoospore, which swims in free water in the soil matrix to the host, where infection occurs. If the zoospore is carrying PMTV, it is transmitted to the host tissue.

As with other plasmodiophorid pathogens, *S. subterranea* has proved difficult to control. With standards for seed, pre-pack and processing potato crops becoming ever stricter, a great deal of effort has been put into identifying effective control measures. Disease resistance to the pathogen is likely to be the most important means of control but almost three quarters of the cultivars grown in GB are either susceptible (rating 1, 2 or 3) or moderately susceptible (rating 4 or 5) to powdery scab (Anon., 2002). Soil contamination by cystosori is believed to be widespread in soils in GB potato growing regions, nevertheless disease avoidance is practised as a control measure, particularly by seed growers.

The use of chemicals to control powdery scab has been evaluated since the early part of the century (Harrison, 1997). In recent years, the growing importance of powdery scab as a disease of potatoes and the absence of other effective control measures, has led to the evaluation of a wide range of chemicals. These have been evaluated in two ways, as tuber treatments to control tuber-borne inoculum and as soil treatments to control soil-borne inoculum.

This paper presents the results for two fungicides, one used as a seed tuber treatment and the other as a soil treatment, extracted from a series of trials carried out over 14 years. The results will be used to demonstrate the issues relating to the effective use of chemicals for control of powdery scab and by inference PMTV.

MATERIALS AND METHODS

Trials were carried out in fields across northeast Scotland. Sites were selected where soil contamination by cystosori of *S. subterranea* was believed present at high levels or in fields believed to be uncontaminated. Where high levels of powdery scab were found on a prior potato crop, fields were considered contaminated. Uncontaminated fields were locations where either potatoes had not been grown in memory and where manure from stock fed with potatoes had never been spread or where powdery scab had never been found on previous potato crops.

Trials evaluated a range of chemical treatments for efficacy against *S. subterranea* compared to untreated controls. They were usually of a randomised block design with four to six replicates. Plot sizes were mostly 4 drills wide (c. 2.7m) by 6.25m long and planted with 100 seed tubers. A few unyielded trials were 2 drills wide and planted with 30 to 50 seed tubers.

Seed tuber treatments targeted at control of tuber-borne inoculum were applied either as a dust or sprayed onto tubers on a roller table. In either case, tubers in all treatments received the same physical handling. The seed tuber treatment reported in this paper was a fungicide containing the active ingredients maneb and zinc oxide (80% + 2.5% w/w a.i., Mazin, Universal Crop Protection Ltd. 4 kg product/tonne). This fungicide received provisional approval for use as a seed tuber treatment from 1987 to 1996. As the only product receiving approval it was frequently used as a treatment in commercial trials evaluating chemicals even where control of soil-borne inoculum was evaluated. It was applied immediately prior to planting by placing seed for the trial in a clean paper sack, adding the pro-rata quantity of fungicide and gently rolling the dust and seed tubers in the bag. This achieved a uniform coverage of tubers. Seed for the trials was of a susceptible cultivar and taken from a certified stock. The levels of powdery scab were thus within the group tolerance of no more than 3% tubers having a surface area greater than 12.5%. Frequently, a greater percentage of tubers exhibited low levels of infection.

Soil treatments targeted at soil-borne inoculum were applied immediately prior to planting either by spraying a chemical solution into the furrow over the seed tubers before ridging up or by spraying a chemical solution onto the destoned bed and incorporating into the bed prior to planting. The soil treatment reported in this paper was a fungicide containing the active ingredient fluazinam (0.5 kg a.i./litre, Shirlan, Syngenta Crop Protection UK). Various doses of fluazinam are reported here. Seed tubers used for trials evaluating control of soil-borne inoculum were of susceptible cultivars and selected from stocks where no powdery scab could be detected.

At harvest 100 tubers (>35mm) were sampled at random from each plot and assessed individually for presence of powdery scab (incidence) and percentage surface area (severity). The percentage of tubers with cankerous outgrowths was also assessed.

RESULTS

Only those trials where the incidence of powdery scab on the untreated control were greater than 25% are reported. The results for cankers are only shown where the incidence exceeded 5%. The control achieved by maneb plus zinc oxide or fluazinam is expressed as a percentage of the untreated control.

The results of the evaluation of maneb plus zinc oxide in 27 trials are shown in Table 1. The control achieved by maneb plus zinc oxide in the situations where inoculum was seed-borne, soil-borne or seed- and soil-borne was highly variable. Statistically significant reductions in disease were infrequently recorded. As might be expected, the overall control where soil was the source of inoculum was less than where seed tuber were the source. The percentage control of severity was usually greater than that for incidence. Complete control was never achieved.

Table 1. Evaluation of maneb plus zinc oxide seed tuber treatment for the control of *S. subterranea*

Year	Cv[1]	Source of inoculum	Incidence			Severity			Cankers		
			UT	%[2]	Sig.	UT	%[2]	Sig.	UT	%[2]	Sig.
1988	E	Seed	-	-	-	8	-23	Ns	-	-	-
1989	E	Seed	100	0	Ns	6	46	*	-	-	-
1990	E	Seed	100	0	Ns	14	37	*	-	-	-
1990	E	Seed	74	7	Ns	8	32	Ns	-	-	-
1992	E	Seed	72	5	Ns	4	9	Ns	-	-	-
1992	E	Seed	55	-28	Ns	2	-26	Ns	-	-	-
1992	E	Seed	91	2	Ns	11	21	Ns	-	-	-
1993	E	Seed	29	63	*	6	75	*	-	-	-
1993	E	Seed	40	31	Ns	5	81	**	-	-	-
1994	E	Seed	26	21	Ns	1	36	Ns	3	100	Ns
Mean % control				11			29			100	
1993	E	Seed+Soil	98	32	*	22	41	Ns	-	-	-
1994	E	Seed+Soil	56	25	Ns	6	20	Ns	-	-	-
1997	D	Seed+Soil	73	36	Ns	5	62	*	-	-	-
Mean % control				31			42			-	
1987	C	Soil	40	20	Ns	4	75	*	6	89	**
1988	E	Soil	100	0	Ns	13	-7	Ns	57	-4	Ns
1989	E	Soil	100	0	Ns	9	16	Ns	-	-	-
1989	E	Soil	97	6	Ns	17	62	*	31	60	*
1990	E	Soil	70	39	*	2	48	Ns	-	-	-
1991	E	Soil	90	-5	Ns	12	-22	Ns	-	-	-
1991	E	Soil	89	4	Ns	8	5	Ns	-	-	-
1992	E	Soil	48	-5	Ns	9	9	Ns	31	16	Ns
1994	K	Soil	68	30	Ns	5	24	Ns	2.3	26	Ns
1994	C	Soil	55	-24	Ns	3	-49	Ns	-	-	-
1994	M	Soil	59	32	Ns	9	42	Ns	12	73	**
1995	M	Soil	81	-4	Ns	2	56	Ns	-	-	-
1996	E	Soil	53	-11	Ns	6	-24	Ns	13	-40	Ns
1996	M	Soil	39	-27	Ns	1	-14	Ns	11	36	Ns
Mean % control				4			16			32	

[1]Cultivars: C = Cara, D = Desiree, E = Estima, K = Kennebec, M = Maris Piper

[2]% = % control compared to untreated (UT).

Ns = Not significant, *, ** = significant at $p < 0.05, 0.01$

Fewer trials were carried out evaluating fluazinam as a soil treatment. A clear dose response was apparent, particularly in the 1995 trial (Table 2). The control of *S. subterranea* increased as the dose applied increased to 4.0 kg a.i./ha. This trial also indicated that incorporation was more effective than application in-furrow. The most effective incorporation was achieved when the chemical was applied during de-stoning (2000, seed+soil inoculum).

Although the results were variable, at higher doses reductions were more frequently recorded, irrespective of the source of inoculum than for the maneb plus zinc oxide seed tuber treatment. The percentage reduction in severity was usually greater than that in incidence. Complete control was never achieved.

Table 2. Evaluation of fluazinam soil treatment for the control of *S. subterranea*

Year	Cv[1]	Source of inoculum	Fluazinam dose (kg a.i./ha) and application method	Incidence			Severity		
				UT	%[2]	Sig.	UT	%[2]	Sig.
1994	E	Seed	0.5 – incorporated[4]	29	0	Ns	1	24	Ns
			1.0 – incorporated[4]	29	-3	Ns	1	-7	Ns
			2.0 – incorporated[4]	29	-45	Ns	1	-15	Ns
			4.0 – incorporated[4]	29	-17	Ns	1	-18	Ns
1994	E	Seed	4.0 – incorporated[4]	35	62	**	1	81	**
2000	E	Seed	1.5 - incorporated	27	4	Ns	2	29	Ns
			1.5 - in furrow	27	-19	Ns	2	8	Ns
1994	E	Soil	0.5 – incorporated[4]	47	10	Ns	12	-15	Ns
			1.0 – incorporated[4]	47	10	Ns	12	-4	Ns
			2.0 – incorporated[4]	47	8	Ns	12	-8	Ns
			4.0 – incorporated[4]	47	9	Ns	12	21	Ns
1994	E	Soil	4.0 – incorporated[4]	53	27	Ns	4	59	Ns
1995	E	Soil	0.25 – in furrow	81	25	**	41[3]	47	**
			0.5 – in furrow	81	20	**	41[3]	40	**
			1.2 – in furrow	81	29	**	41[3]	57	**
			2.0 – in furrow	81	27	**	41[3]	51	**
			4.0 – in furrow	81	47	**	41[3]	61	**
			4.0 - incorporated	81	57	**	41[3]	86	**
2000	E	Seed+Soil	1.5 - incorporated	45	5	Ns	10	59	*
			1.5 - in furrow	45	-2	Ns	10	37	Ns
2000	E	Seed+Soil	1.5 – incorporated[5]	51	49	***	2	70	*

[1]Cultivar: E = Estima

[2] % = % control relative to untreated (UT)

[3] Severity expressed as % 'unmarketable' tubers i.e. >5% surface area affected

[4] Incorporated to at least 20cm after spraying onto destoned bed

[5] Incorporated by spraying soil passing through destoner

Ns = Not significant; *, **, *** = significant at $p < 0.05, 0.01, 0.001$

DISCUSSION

There is evidence from these results that both chemicals can have a substantial effect on *S. subterranea* but their consistency of effect is poor. This variability in efficacy of both seed tuber and soil treatments is unhelpful when attempting to provide guidance to a potato grower about chemical control of *S. subterranea.* The results suggest that consistent control cannot be guaranteed and the use of a chemical as a sole control measure will depend on a financial analysis of the proportion of occasions a cost effective response is achieved.

In assessing the effectiveness of control treatments, it is important to have an understanding of inoculum. Because *S. subterranea* cannot be cultured in vitro, the initial inoculum in the trials described in this paper could not be quantified. Thus, one reason for the variability in results could be differences in inoculum between trials. Whilst every effort was made to identify trial sites for control of tuber-borne inoculum free from soil-borne inoculum, the absence of soil contamination could not be guaranteed. Conversely, where control of soil-borne inoculum was being tested, the absence of contamination of seed tubers by cystosori was not possible to confirm. Recent studies have shown that symptomless but contaminated tubers can lead to disease development equivalent to that of heavily infected tubers (Wale *et al.*, 2002).

Another complicating factor in interpreting chemical control trials, is that inoculum can increase in the roots of potato crops and, as a result, even low levels of initial inoculum can lead to severe tuber infection (Burnett, 1991). Conducive conditions for inoculum multiplication early in crop growth may increase the disease pressure for subsequent tuber infection and reduce effectiveness of chemical control. Ideal conditions for infection are free moisture in the soil matrix to permit the zoospores to swim to the host tissue and a temperature between c. 9° and 17°C. When soil moisture is high, the conditions may be suitable for the pathogen but they may also improve release of the chemical control agent in the soil, although chemical release in soil may be under the control of many factors (e.g. pH). As the zoospores are the phase of the life cycle susceptible to chemical control, the chemical control agent needs to be in the free moisture in soil in sufficient quantity to interact with zoospores. An *in vitro* experiment reported by Burnett & Wale (1993), demonstrated that zinc was only effective when inoculum was low and zinc concentrations high.

In vitro studies by Fournier (1997) on the release of primary zoospores after 5 days incubation in the presence of 1ppm of a range of fungicides, demonstrated that the four fungicides tested inhibited release. Zinc oxide significantly reduced the release to 512 zoospores/ml and fluazinam to 860 zoospores/ml from 1327/ml in the water control. At 10ppm the release of zoospores was totally inhibited by these two fungicides. However, measurement of zinc in soil solution *in vivo* (Burnett, 1991) after zinc applications of 10 or 15 kg/ha found it rose to just 0.5-1.1 ppm.

These results suggest that chemical treatment is unlikely ever to be fully effective except where inoculum pressure is low. Since inoculum may be found at any point within the soil profile, an even distribution of a chemical within the soil profile is likely to result in more effective control of soil-borne *S. subterranea*. The results with fluazinam confirm this.

Powdery scab is such a difficult disease to control and can have such a major impact on profitability, even small reductions in disease can be valuable. Thus, for example, seed growers who can increase the proportion in a seed fraction by 2.5 tonne/ha will more than

cover the cost of most treatments. For the seed grower, a reduction in severity to bring a higher percentage of tubers below the surface area tolerance for certification can be important. In these trials chemical control appears to be more consistent in reducing severity. For the ware grower, however, a reduction in incidence is more beneficial and this seems to be difficult to achieve.

As far as control of PMTV is concerned, it can be assumed that the degree of control of the virus is directly related to that of the vector

Effective control of powdery scab and PMTV will depend on a combination of measures – integrated control – where chemical control is combined with disease escape, judicious use of disease resistance in cultivars and cultural control.

Neither maneb plus zinc oxide or fluazinam are currently approved for the control of powdery scab.

ACKNOWLEDGEMENTS

I would like to thank SAC staff who have carried out the trials reported in this paper, in particular Drs McKenzie Munro, Fiona Burnett, Phillip Burgess and Alex Hilton. Thanks are due to Dr Richard Falloon for permission to reproduce results of trials carried out with SAC funded by Potato Sector of New Zealand Foundation for Research, Science & Technology (Falloon, 1997). Thanks are also due to Mr Willie Kyle for allowing reproduction of results from a trial investigating development of the Spraymax® sprayer system and to the Potato Marketing Board / British Potato Council for funding projects, from which some of the results are taken.

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