

A controlled environment test for resistance to *Soil-borne cereal mosaic virus (SBCMV)* and its use to determine the mode of inheritance of resistance in wheat cv. *Cadenza* and for screening *Triticum monococcum* genotypes for sources of SBCMV resistance

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Several wheat genotypes, including eight with known field responses, were evaluated for their reaction to *Soil-borne cereal mosaic virus* (SBCMV, genus Furovirus) by growing in naturally infested soil under controlled environment conditions. Virus antigen titres in the foliage 8–9 weeks after sowing mostly reflected the field responses, showing that growth chamber-based tests can be used to improve the speed and reliability of germplasm screening. Such tests were used to determine the mode of inheritance of the SBCMV resistance in cv. *Cadenza*, commonly used in UK wheat-breeding programmes. One hundred and eleven doubled haploid (DH) lines derived from an F_1 of a cross between cvs *Cadenza* (resistant) and *Avalon* (susceptible) were evaluated. This DH population segregated for the reaction to SBCMV in a ratio of 1 : 1 (resistant : susceptible). This suggests that the SBCMV resistance is controlled by a single gene locus. As a first step towards identification of new sources of improved SBCMV resistance (e.g. immunity) as well as sources of the resistance to the virus vector, *Polymyxa graminis*, a set of 26 *Triticum monococcum* lines of diverse geographical origin was also screened. Most lines were susceptible to SBCMV, but one line of Bulgarian origin was resistant to the virus and possibly partially resistant to the virus vector.

Keywords: *Polymyxa graminis*, resistance, *Soil-borne wheat mosaic virus*, *Triticum aestivum*, *Triticum monococcum*, wheat

Introduction

A serious ‘mosaic-like leaf mottling’ or ‘rosette disease’ of winter wheat caused by a virus was first reported in the USA in 1919 (McKinney, 1925). The causal virus was *Soil-borne wheat mosaic virus* (SBWMV), the type member of the genus Furovirus. SBWMV is naturally transmitted only by its vector, *Polymyxa graminis*, an eukaryotic obligate biotrophic plasmodiphorid parasite of plant roots (Rao & Brakke, 1969). Virus particles are protected from the environment within *P. graminis* resting spores that may remain dormant but viable for decades, probably until a suitable host plant is encountered (Brakke & Langenberg, 1988). There are currently no efficient, inexpensive chemical agents for control of

P. graminis. SBWMV is considered to be one of the most important diseases in winter wheat, especially in central and eastern USA, because it is persistent and can practically destroy an entire crop of a susceptible cultivar when the weather conditions are particularly favourable for disease development (Myers *et al.*, 1993). SBWMV and similar viruses are also known to occur in Brazil, Argentina, China, Japan and several European countries (Brakke & Langenberg, 1988; Koenig & Huth, 2000).

The global population of furoviruses on wheat consists of genetically divergent strains, and a relatively high degree of polymorphism has been reported between virus genomes at the nucleotide and amino-acid levels (Shirako *et al.*, 2000). Importantly, a virus that is widely distributed in bread wheat, durum wheat and rye crops throughout France, Italy, Germany, Poland and Denmark shares only $\approx 70\%$ genome identity with SBWMV from the USA and Japan (Diao *et al.*, 1999; Koenig *et al.*, 1999). Some authors still consider this virus to be a European strain of SBWMV, but the proposed species name for it, *Soil-borne*

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cereal mosaic virus (SBCMV; Koenig & Huth, 2000; Yang *et al.*, 2001), has recently been approved by the International Committee on Taxonomy of Viruses. This virus was first detected in the UK at one farm in Wiltshire in 1999 (Clover *et al.*, 2001), and subsequently has been detected at several other locations in Wiltshire, Kent and on the Isle of Wight (Budge & Henry, 2002; K.K., unpublished data).

The persistent, soilborne nature of SBCMV, SBWMV and related diseases makes the use of resistant crop cultivars currently the only practical, environmentally friendly and sustainable means of control. Wheat cultivars with resistance to these virus diseases are available; however only the inheritance of field resistance to SBWMV has been studied so far for several commercial wheat cultivars in the USA, Brazil and Japan (reviewed by Kanyuka *et al.*, 2003). It has been proposed that SBWMV resistance is controlled either by a single dominant gene (Miyake, 1938; Modawi *et al.*, 1982), or that two (Shaan *et al.*, 1966; Merkle & Smith, 1983; Barbosa *et al.*, 2001) or even three genes (Nakagawa *et al.*, 1959) are involved. This contradiction may reflect genuine differences between the different sources of resistance, but it is also possible that, at least in some studies, a proportion of susceptible individuals were incorrectly identified as resistant and *vice versa*. In most of these studies, plant reactions to SBWMV were scored simply on the basis of the presence or absence of visible leaf symptoms and the plant growth habit (e.g. stunting, rosetting etc.). However, the appearance and severity of soilborne mosaic symptoms in wheat may vary considerably depending on the plant genotype, the concentration and aggressiveness of the virus or virus strain, as well as the environmental conditions (temperature, moisture, etc.) (Budge & Henry, 2002). Also, some wheat genotypes may show no visible mosaic symptoms despite the presence of moderate to high virus titres in both leaves and roots (K.K., unpublished data). Moreover, in the field uneven distribution of fertilizer, nutrient imbalance or winter injuries may cause symptoms in the resistant genotypes that could be mistaken for the soilborne virus disease (e.g. leaf mosaic, stunting). Therefore it is very important for genetic studies to combine visual scoring of phenotypes with virus detection by enzyme-linked immunosorbent assay (ELISA) or molecular techniques, e.g. reverse transcription–polymerase chain reaction (RT–PCR).

Cultivar trials in the UK, France and Italy have shown that SBCMV can reduce grain yield of susceptible winter wheat accessions on heavily infested fields by up to 50% compared with that of resistant cultivars (Bayles & Napier, 2002; Budge & Henry, 2002). So far, fewer than a dozen UK wheat cultivars have been identified as either resistant or partially resistant to SBCMV, and only three of these (Charger, Claire and Hereward) appear on the current (2003) Home-Grown Cereals Authority Recommended List of Winter Wheat Cultivars. The genetics and the exact origin of this resistance in UK wheat cultivars are unknown, but the older cv. Cadenza has been implicated as a possible resistance source because it is a common parent occurring in the pedigree of seven of the resistant cultivars (Bayles & Napier, 2002). The roots of both

susceptible and resistant genotypes can be colonized by *P. graminis* and therefore the resistance is directed against the virus rather than its vector (Larsen *et al.*, 1985; K.K., unpublished observation). Resistant genotypes are known to contain high virus levels in the root system, and zero or low levels in the leaf tissues (Hunger & Sherwood, 1985; Driskel *et al.*, 2002). Therefore the disease resistance is likely to operate by a mechanism that either restricts virus multiplication in the leaves, or prevents or reduces virus vascular transport from roots to leaves (Hariri *et al.*, 1987; Driskel *et al.*, 2002).

The main objectives of this study were (i) to determine whether wheat genotypes can be correctly scored for their reaction to SBCMV under controlled environment conditions, to improve the speed and reliability of germplasm screening for resistance; (ii) to determine the mode of inheritance of SBCMV resistance for cv. Cadenza, commonly used in UK wheat breeding programmes, as an essential step towards developing molecular markers associated with disease resistance and cloning of the resistance gene(s); and (iii) to screen diploid *Triticum monococcum* accessions as potential new sources of resistance to SBCMV and/or its vector, *P. graminis*. A source of resistance that operates against the vector, or that efficiently prevents or reduces virus accumulation in the root system, would be invaluable for developing new wheat-breeding materials with improved resistance.

Materials and methods

Plant material

The following hexaploid wheat *Triticum aestivum* genotypes were employed in this study: (i) UK winter type cvs Consort, Madrigal, Riband, Hereward and Avalon, obtained from reliable commercial sources; (ii) European winter type F_1 hybrid Cockpit, and its two parental cvs Piko and Phobos, provided by Volker Lein (Saaten Union Recherche, Estrées-Saint-Denis, France); (iii) UK winter type cvs Claire and Charger, USA spring type cv. Lemhi, and an Indian spring type landrace Kharchia, provided by Lesley Boyd (John Innes Centre, Norwich, UK). The population of 111 doubled-haploid (DH) individuals, derived from an F_1 progeny of a cross between cvs Avalon and Cadenza, was developed by Clare Ellerbrook and the late Tony Worland (John Innes Centre). This mapping population was originally developed to explore canopy architecture traits, following earlier discussions with Darren Lovell (Rothamsted Research), Steve Parker (Central Science Laboratory, York, UK) and the late Tony Worland. All the *T. monococcum* accessions were from the N. I. Vavilov Research Institute of Plant Industry collection.

Virus and inoculation

In June 2002, leaf samples of wheat cv. Consort displaying mosaic and yellowing were collected from a field in Kent, UK from which furovirus infection had not previously been reported. These samples tested positive for SBCMV

by ELISA (data not shown). It appeared that typical SBCMV-induced symptoms (leaf mosaic, stunting) had been observed consistently in relatively small patches at this site on several wheat cultivars for at least 15 years, but the presence of a furovirus was not tested or confirmed until this study. Soil heavily infested with viruliferous *P. graminis* was collected from the most severely affected patch of the field after harvest in September 2002.

For resistance evaluation, pregerminated wheat seeds were transplanted into 7 cm² plastic pots (three seedlings per pot) containing the infested soil mixed with sand (1 : 2), and 3.0–3.5 g L⁻¹ of the controlled release fertilizer Osmocote Plus (Scotts Europe BV, Heerlen, the Netherlands). Two replicate pots of each line or cultivar were placed into trays, generously watered every other day with a nutrient solution (Adams *et al.*, 1986), and maintained in growth rooms at 16°C (night) to 20°C (day) and a 16 h photoperiod. These conditions were chosen on the basis of data from similar, earlier glasshouse-based experiments with SBWMV (Armitage *et al.*, 1990) and *P. graminis*-transmitted bymoviruses of barley (Adams *et al.*, 1986), and preliminary experiments performed by the authors. Approximately 4–5 weeks post inoculation (wpi), when the plants had reached growth stage 4 on the Feekes scale (Large, 1954), they were trimmed to ≈ 5–7 cm from the soil level to stimulate systemic virus movement, and allowed to grow for an additional 4–8 weeks.

Detection of SBCMV

The youngest leaf of the three plants in each pot was taken 6–8 wpi, and the leaves from the same pot were combined for sample preparation. Leaf extracts were prepared using the Leaf Juice Press (Erich Pollähne GmbH, Wennigsen, Germany) in the presence of 5 vol extraction buffer (phosphate-buffered saline buffer pH 7.4 containing 0.5% Tween-20, 2% polyvinylpyrrolidone MW 44 000, and 1% nonfat dry milk) per 1 g fresh weight of leaf material. Leaf extracts were cleared by centrifugation for 1 min in a bench-top centrifuge at maximum speed, and two 200 µL aliquots of each extract were applied to a microtitre plate and incubated at 4°C overnight. These samples were tested for the presence of virus antigens by the indirect F(ab')₂ ELISA method and polyclonal antiserum to SBCMV (Rothamsted Research collection, 317) essentially as described by Chen & Adams (1991). Absorbances were measured at 405 nm ($A_{405\text{nm}}$) using an MRX microplate reader (Dynex Technologies, Chantilly, VA, USA). Roots of selected plants were also tested by ELISA as described above.

Results

Evaluation of selected wheat genotypes for resistance to SBCMV

The inoculation experiment involved a total of 13 wheat genotypes. Several cultivars with known field responses to SBCMV (Bayles & Napier, 2002; Budge & Henry, 2002)

Table 1 Absorbance values in ELISA tests for SBCMV using leaves of wheat genotypes grown in naturally infested soil under controlled environment conditions, and comparison with the reported field reaction of these genotypes

Genotype	Field reaction ^a	Controlled environment tests	
		$A_{405\text{nm}}$ ^b	P^c
Experiment 1			
Riband	S	> 4.0	< 0.001
Consort	S	> 4.0	< 0.001
Hereward	R	0.118	NS
Charger	R	0.110	NS
Madrigal	S	3.215	< 0.001
Kharchia	nd ^d	3.364	< 0.001
Lemhi	nd	0.233	< 0.001
Claire	R	0.101	NS
Control (ni) ^e	S	0.067	
Experiment 2			
Avalon	nd	0.706	< 0.001
Charger	R	0.054	NS
Consort	S	0.636	< 0.001
Cadenza	R	0.042	NS
Cockpit	R	0.826	< 0.001
Piko	nd	0.597	< 0.001
Phobos	nd	0.771	< 0.001
Control (ni)	S	0.055	

^aField reaction to SBCMV as determined by Bayles & Napier (2002); Budge & Henry (2002). S, susceptible; R, resistant.

^bMean absorbance values from two replicate samples, each consisting of combined leaf extracts from three individuals of the same genotype grown in the same pot. Values are smaller in experiment 2 because of a shorter incubation time with the substrate.

^cSignificance of difference from ni control; SED = 0.0323 (9 df) and 0.0305 (8 df), respectively, for experiments 1 and 2.

^dData not available.

^eControl, cv. Consort grown in virus-free soil.

were used as the controls. The cvs Avalon, Kharchia and Lemhi, with unknown responses to SBCMV, were evaluated because the DH populations derived from F_1 crosses involving these genotypes are either available, or are currently being produced (L. Boyd, John Innes Centre, personal communication). Symptoms of SBCMV, a mild leaf mosaic and green/yellow streaks, were frequently seen in most, though not all, individuals of susceptible wheat genotypes at 8–9 wpi. All genotypes known to be susceptible to SBCMV in the field ($n = 3$) were also highly susceptible in these tests, and high titres of SBCMV were detected in their leaves (Table 1). Occasionally, very mild mosaic and yellowing were seen on the leaves of resistant genotypes, but these were probably caused by nutrient imbalance or other abiotic factors, as SBCMV was not detected in their leaves and similar symptoms appeared on uninoculated controls. The susceptible disease reaction of the F_1 hybrid Cockpit was unexpected, because this genotype has been identified as resistant in field tests. Both parents of Cockpit (Piko and Phobos) were also susceptible to SBCMV in this controlled environment-based test.

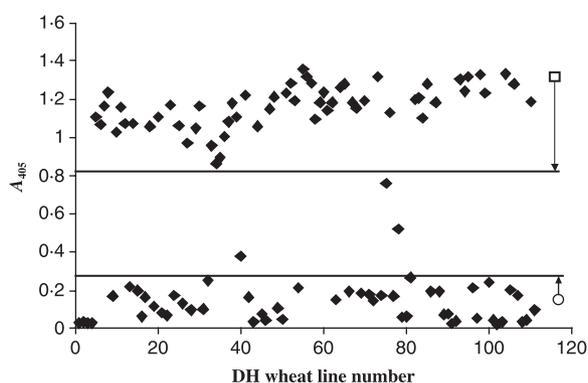


Figure 1 Absorbance values in ELISA tests for SBCMV for a population of 111 doubled-haploid (DH) lines derived from an F_1 cross between cvs Cadenza (resistant) and Avalon (susceptible), showing segregation of SBCMV resistance. Each point indicates an individual DH line. Parental cvs Cadenza and Avalon are indicated as a square and circle, respectively, with their 95% fiducial limits (derived from log-transformed data) shown by arrows and lines.

Inheritance of SBCMV resistance in cv. Cadenza

The resistance to SBCMV of 111 DH lines derived from an F_1 cross between cvs Cadenza (resistant) and Avalon (susceptible) was evaluated. The earlier experiments had confirmed their reaction to virus because the virus antigen was not detected in leaves of Cadenza at 8 wpi, while the A_{405nm} value for Avalon was at least 18 times that for Cadenza or uninfected control plants. Most of the DH lines gave an ELISA value that belonged in one of two groups (Fig. 1). Fifty-one lines had A_{405nm} values that were not significantly greater than those for Cadenza and uninfected control plants. The A_{405nm} values of the second group of 57 lines were 12–18 times higher than those for Cadenza and uninfected control plants, and were not significantly different from those for the susceptible parent. Therefore the first group was considered as resistant and the second group as susceptible. Three DH lines had intermediate ELISA A_{405nm} values. The reason for these intermediate values is unknown, but because the seeds of the DH wheat lines used had been multiplied in the field, it is possible that they were the result of seed impurity. These lines were omitted from further genetic analysis.

The observed phenotype segregation was compared with Mendelian expectations. The DH Avalon \times Cadenza population segregated 51 resistant and 57 susceptible lines ($\approx 1 : 1$), which is consistent with the presence of one major gene/locus controlling the resistance ($\chi^2 = 0.333$; $P = 0.56$). This gene/locus was provisionally designated *SbmCz1* (soilborne cereal mosaic virus resistance in cv. Cadenza).

Screening *T. monococcum* accessions for resistance to SBCMV

Twenty-six diploid *T. monococcum* accessions with a diverse geographical and ecological origin (Table 2) were

Table 2 Absorbance values in ELISA tests for SBCMV, using leaves of *Triticum monococcum* accessions grown in naturally infested soil under controlled environment conditions

Accession	Origin	Variety	A_{405nm}^a
K-105	Chechen-Ingushetia	<i>flavescens</i> , <i>hornemannii</i>	0.773
K-8365	Crimea, Ukraine	<i>flavescens</i> , <i>macedonicum</i>	0.727
K-8555	Crimea, Ukraine	<i>macedonicum</i> , <i>symphaeropolitanum</i>	0.767
K-18105	Nagorno-Karabach, Azerbaijan	<i>monococcum</i> , <i>macedonicum</i>	0.682
K-20399	Germany	<i>flavescens</i>	0.789
K-20491	Spain	<i>flavescens</i>	0.782
K-20589	Spain	<i>monococcum</i>	0.709
K-20994	Turkey	<i>vulgare</i> , <i>macedonicum</i>	0.469
K-21308	Italy	<i>vulgare</i>	0.900
K-23032	Yugoslavia	<i>vulgare</i>	0.698
K-23653	Armenia	<i>hornemannii</i>	0.598
K-25968	Austria	<i>vulgare</i>	0.486
K-29603	Czechoslovakia	<i>flavescens</i> , <i>monococcum</i>	0.551
K-30086	Armenia	<i>macedonicum</i>	0.667
K-30090	Armenia	<i>monococcum</i>	0.622
K-31683	Georgia	<i>hornemannii</i>	0.812
K-38079	Bulgaria	<i>macedonicum</i>	0.036
K-39417	Albania	<i>nigricultum</i> , <i>flavescens</i>	0.558
K-39471	Balkans region	<i>macedonicum</i>	0.585
K-39722	Greece	<i>vulgare</i>	0.633
K-45024	Turkey	<i>hornemannii</i>	0.550
K-45927	Denmark	<i>vulgare</i>	0.515
K-46748	Romania	<i>macedonicum</i> , <i>vulgare</i>	0.687
K-46752	Hungary	<i>macedonicum</i>	0.834
K-46753	Sweden	<i>vulgare</i>	0.743
K-58505	Iran	<i>hornemannii</i>	0.617
Control (ni) ^b			0.055

^aMean absorbance values from two replicate samples each consisting of the combined leaf extracts from three individuals of the same genotype grown in the same pot. All values except those for K-38079 are significantly different ($P < 0.001$) from the ni control (SED = 0.1065, 27 df).

^bControl, cv. Consort grown in virus-free soil.

screened for SBCMV resistance as above. Leaves of inoculated plants were tested at 8 wpi for the presence of SBCMV antigen using ELISA. Twenty-five accessions displayed high A_{405nm} values compared to cv. Consort, and were considered to be fully susceptible (Table 2). However, one accession (K-38079) of *T. monococcum* var. *macedonicum* from Bulgaria showed the lowest absorbance value (0.036) in an ELISA test of leaf tissue (Table 2), and no more than very low levels in the root tissue (Table 3). Roots of this, and several other *T. monococcum* accessions and wheat cultivars, were inspected under the microscope for the presence of *P. graminis* after staining with 0.1% acid fuchsin (Hooper, 1986). Numerous

Table 3 Absorbance values in ELISA tests for SBCMV, using leaves and roots of selected *T. monococcum* accessions grown in naturally infested soil under controlled conditions

Accession	Roots		Leaves	
	A_{405nm}^a	P^b	A_{405nm}	P
K-38079	0.210	NS	0.046	NS
K-39722	2.895	< 0.001	0.633	< 0.001
Consort	nd ^d		0.636	< 0.001
Control (ni) ^c	0.050		0.055	

^aMean absorbance values from two replicate samples each consisting of the combined leaf or root extracts from three individuals of the same genotype grown in the same pot.

^bSignificance of difference from ni control; SEDs 0.1233 (3 df) and 0.0210 (4 df), respectively, for root and leaf samples.

^cControl, cv. Consort grown in virus-free soil.

^dData not available.

mature resting spores (cystosori) were detected in roots of all susceptible genotypes as well as in SBCMV-resistant wheat cvs Charger and Cadenza, while *P. graminis* cystosori were less abundant in the roots of *T. monococcum* var. *macedonicum* (K-38079) (data not shown).

Discussion

This study has demonstrated that wheat genotypes can be tested efficiently for their resistance to SBCMV under controlled environment conditions using soil naturally infested with viruliferous *P. graminis*. This relatively low-cost technique can be used to improve the speed and reliability of screening wheat germplasm for resistance to SBCMV in breeding programmes. In these tests, all wheat cultivars previously scored for resistance under field conditions (Bayles & Napier, 2002; Budge & Henry, 2002) were correctly identified as either resistant or susceptible, except for the F_1 hybrid Cockpit (see below). The main criterion for scoring disease resistance in this study was the absence of the SBCMV antigen in the leaf tissues, rather than symptomatology or effects on crop yield. This is preferable for a breeding programme because lines with few symptoms or good yield, but high virus titres, would certainly increase the virus inoculum concentration in the soil, which is undesirable as a long-term sustainable management strategy. Reliance on visual symptoms alone is also unreliable because leaf yellowing and plant stunting can have other causes (especially in the field), and because some individuals of the same susceptible genotype did not develop typical SBCMV symptoms despite having high titres of virus antigen in leaves. In the growth chamber-based resistance tests, the plant reaction to SBCMV was determined within 6–9 weeks of sowing. This contrasts with field-based tests that require at least two growing seasons to complete, and provides the advantage that the viruliferous *P. graminis* is more uniformly distributed. The susceptibility of the F_1 hybrid cv. Cockpit in this study was unexpected as it was scored as resistant to SBCMV in

earlier field trials (Bayles & Napier, 2002). The reasons for this require further investigation, but any field resistance that it possesses is likely to be of a different type to that found in the other genotypes. It is also possible that the pathogenicity of the virus isolate used in this study differs from that in the field tests.

The resistance to SBCMV in the UK cv. Cadenza is determined by a single gene locus, provisionally designated as *SbmCz1*. This is the first report of a genetic analysis of resistance to SBCMV in UK wheat cultivars. Current work is progressing to identify and develop molecular markers linked to this locus. These will assist selection in current European wheat breeding programmes, and will also be used to confirm whether the same gene is carried by all the resistant European wheat cultivars, as has been suggested (Bayles & Napier, 2002). In previous genetic studies it was concluded that the resistance reaction to SBWMV in several wheat cultivars from the USA and Japan is also controlled by a single dominant gene (Miyake, 1938; Dubey *et al.*, 1970; Modawi *et al.*, 1982). The genome sequence of the virus isolate used in this study has not been determined, but is expected to be closely related to other UK and European SBCMV isolates. If so, it will be at least 30% divergent from the SBWMV isolates in the USA and Japan (Diao *et al.*, 1999; Koenig & Huth, 2000; Clover *et al.*, 2001). Therefore it will be interesting in future experiments to determine whether the SBWMV-resistant germplasms from the USA and Japan are also resistant to SBCMV, and whether these genotypes carry a gene that is allelic to *SbmCz1*.

If there is only one resistance gene in current European wheat cultivars, it is reasonable to predict that this resistance could be overcome by new strains of SBCMV or by strains imported from other geographical regions. Plant RNA viruses are known to have high rates of mutation, and new strains of viruses with altered pathogenicity have been reported to evolve frequently, especially when only one resistance gene source is employed extensively. This has happened recently in Europe with the *P. graminis*-transmitted bymoviruses *Barley yellow mosaic virus* and *Barley mild mosaic virus*, where pathotypes have emerged that overcome the resistance genes *rym4* and *rym5* (Hariri *et al.*, 1990; Huth, 1991; Adams, 2002; Hariri *et al.*, 2003; McGrann & Adams, 2004) that are used exclusively in all European barley breeding programmes. Therefore to ensure sustainable disease control via the deployment of resistant germplasms, other novel sources of SBCMV resistance will need to be identified.

Screening of a representative set of hexaploid bread wheat from the main world collections for new sources of resistance to SBCMV is currently in progress. Field screens for resistance to the related virus, SBWMV, in the USA have identified potential novel sources of resistance (Bockus *et al.*, 2001). Such new resistance sources could be used directly in breeding programmes, but the reported resistance to SBWMV in hexaploid bread wheat appears to operate by preventing or reducing virus accumulation in the foliar tissues, while virus accumulation in the root systems is unaffected (Hunger & Sherwood, 1985; Driskel

et al., 2002). It is likely that the SBCMV resistance in European wheat cultivars operates using a similar mechanism (Hariri *et al.*, 1987; Rumjaun *et al.*, 1996). Such genotypes are therefore probably good hosts for the virus vector *P. graminis*, and roots of these plants will provide a source of virus inoculum in the field. This increases the need to search for novel and better sources of virus resistance.

Germplasms of related wild species of wheat are an excellent source for resistance against various diseases, and they are being used worldwide for bread wheat germplasm enhancement. However, these germplasms largely remain unexplored for the resistance to *P. graminis* and the cereal viruses it transmits. As a first step towards identification of better sources of SBCMV resistance (e.g. possible immunity or significant reduction of virus accumulation in the plant root system), as well as resistance to *P. graminis*, a representative set of *T. monococcum* lines from the main wheat collection at the N. I. Vavilov Research Institute of Plant Industry was screened. *Triticum monococcum* was chosen for screening for the following reasons: (i) it is a cultivated species that is closely related to the progenitor of the AA genome of hexaploid bread wheat; (ii) it is considered a rich source of novel genes and variant alleles (Cadle *et al.*, 1997; Shi *et al.*, 1998); (iii) it is accessible to wheat breeders as a gene/trait source via established sexual crossing procedures using specific *T. aestivum* chromosome deletion lines; and (iv) its diploid ($2n = 2x = 14$) genome is ideal for genetic studies. One *T. monococcum* line out of 26 tested contained no SBCMV antigen in the leaves and significantly lower levels of virus antigen in the roots. Resting spores of *P. graminis* were also less abundant in the roots of this resistant line. Further detailed studies are required to identify the exact mechanism and mode of inheritance of novel resistance to *P. graminis* and SBCMV in this *T. monococcum* accession.

The growth chamber-based tests used soil naturally infested with viruliferous *P. graminis*. Sequencing the exact strain(s) of SBCMV present in these tests is in progress. Several related types of *P. graminis* exist in the UK (Ward & Adams, 1998), and it will be important to characterize those present in the infested soil in tests. This will provide reference strains for future characterizations of the resistance sources.

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