Euphytica (2005) 141: 49–63
DOI: 10.1007/s10681-005-5231-y

Sources of resistance to diseases of sugar beet in related *Beta* germplasm: II. Soil-borne diseases

M.C. Luterbacher[1], M.J.C. Asher[1,*], W. Beyer[2], G. Mandolino[3], O.E. Scholten[4], L. Frese[5], E. Biancardi[3], P. Stevanato[3], W. Mechelke[2] & O. Slyvchenko[6]
[1]*Broom's Barn Research Station, Higham, Bury St. Edmunds, Suffolk, IP28 6NP, U.K.;* [2]*KWS SAAT AG, Postfach 1463, D-37555 Einbeck, Germany;* [3]*Istituto Sperimentale per le Colture Industriali, Via di Corticella 133, 40129 Bologna, Italy;* [4]*Plant Research International, P.O. Box 16, 6700 AA The Netherlands;* [5]*Bundesanstalt für Züchtungsforschung an Kulturpflanzen Gene Bank, Bundesallee 50, D-38116 Braunschweig, Germany;* [6]*Institute for Sugar Beet of the UAAS, Klinichna 25, 03141 Kiev, Ukraine; (*author for correspondence: e-mail: mike.asher@bbsrc.ac.uk)*

Received 11 May 2004; accepted 22 October 2004

Key words: Aphanomyces cochlioides, beet necrotic yellow vein virus, *Beta* species, *Pythium ultimum*, resistance, *Rhizoctonia solani*, Rhizomania, sugar beet

Summary

Between 580 and 700 accessions of related cultivated and wild species of the genus *Beta* were assessed for resistance to four soil-borne diseases of sugar beet: two seedling damping-off diseases caused by the fungi *Aphanomyces cochlioides* and *Pythium ultimum* and two diseases of more mature plants, Rhizoctonia root and crown rot, caused by the fungus *R. solani*, and Rhizomania, caused by *Beet necrotic yellow vein virus* (BNYVV), a furovirus transmitted by the plasmodiophorid *Polymyxa betae*. Analysis of resistance data (assessed on an international standardised 1–9 scale of Resistance Scores) indicated that the highest levels of resistance (RS $\leq$ 2) to *A. cochlioides* and *P. ultimum* were to be found amongst accessions of the more distantly related sections *Corollinae* (93% of accessions tested) and *Procumbentes* (10%), respectively; although useful levels could also be found in the more closely related, and sexually compatible, section *Beta* (1–6%). Resistance to Rhizoctonia was also found in section *Beta* (5–7%), depending on whether field or glasshouse tests were used, but there was little evidence of generally high levels of resistance to Rhizomania among accessions of this section. None of the accessions of sections *Corollinae* and *Procumbentes* exhibited any notable resistance to Rhizoctonia. However, all sections *Procumbentes* and some sections *Corollinae* (4%) accessions were highly resistant to Rhizomania. Individuals with high levels of resistance to Rhizomania were identified from within some section *Beta* and *Corollinae* accessions, in which there was evidence of segregation.

Introduction

Soil-borne diseases can have a major impact on sugar beet (*Beta vulgaris* L. ssp. *vulgaris*) at all stages of its development. They infect seedlings, leading to poor crop establishment, or damage the roots of more mature plants. In both cases, yield losses can be high and control is required where the diseases are endemic if production is to be maintained.

Two pathogens that infect sugar beet seedlings, causing 'damping-off' diseases, are the fungi *Aphanomyces cochlioides* Dreschler and *Pythium ultimum* Trow. *A. cochlioides* is commonly encountered on sugar beet throughout the world (Hall, 1989) and is often considered the most important damping-off pathogen affecting the crop (Walker, 2002). *A. cochlioides* can also cause a root rot in more mature plants although this is generally considered less

important than seedling infection, particularly in Europe (Duffus & Ruppel, 1993). Collectively, *Pythium* spp. have an ubiquitous distribution on sugar beet and *P. ultimum* has been identified on sugar beet in Ireland (O'Sullivan & Kavanagh, 1992), the Netherlands (Boerema & Verhoeven, 1976), the United States (Leach, 1986) and the UK (Williams & Asher, 1996), where *P. ultimum* and *A. cochlioides* were found in 31 and 39% of fields, respectively (Payne et al., 1994).

Although the ultimate effect of *A. cochlioides* and *P. ultimum* on sugar beet seedlings is similar, there are differences in their aetiologies. *A. cochlioides* infection of sugar beet primarily occurs 1–3 weeks post-emergence; seedling hypocotyls are invaded, eventually reducing the emergent stem to a black and thread-like structure (Duffus & Ruppel, 1993). *A. cochlioides* infection is favoured by high soil moisture and temperature, thus damage is highest where sugar beet is sown late into warm, damp soils; total crop failure can occur (Williams & Asher, 1996). More commonly, infected seedlings survive but are stunted and less vigorous (Duffus & Ruppel, 1993); the USDA estimated losses to *A. cochlioides* amounted to 1% of the entire crop (Papavizas & Ayers, 1974). In contrast, *P. ultimum* is active over a broader range of temperatures and primarily attacks sugar beet pre-emergence. This latter aspect may lessen the perceived importance of *P. ultimum* as non-emergence of seedlings may be attributed to other causes, e.g., water logging, soil capping or pest damage. Nevertheless, losses to *Pythium* can be significant; in Finland yield decreases were estimated to average 2–3 t/ha (Dunning & Heijbroek, 1981).

Rhizoctonia root and crown rot and Rhizomania are two diseases that can have a significant impact on mature sugar beet plants. The former disease, caused by the fungal pathogen *Rhizoctonia solani* Kühn (teleomorph *Thanatephorus cucumeris* (Frank) Donk), principally in its AG 2-2 form, causes wilting and chlorosis of foliage, and necrosis of petioles close to the crown: roots have a brown-black rot, often starting at the crown and extending into the root, and fissures can develop (Duffus & Ruppel, 1993). Rhizoctonia is prevalent in areas with hot climates, e.g., Egypt, Iran, Japan and Turkey and it is considered the most serious root disease of sugar beet in the United States where average losses nationally are 2%, although there is considerable variation, with losses of up to 50% recorded (Schneider & Whitney 1986). Rhizoctonia is becoming increasingly important in Europe with up to 10% of the sugar beet growing area affected in some countries (Ayala-Garcia et al., 2001).

Rhizomania is caused by *beet necrotic yellow vein virus* (BNYVV), a furovirus transmitted by the plasmodiophorid *Polymyxa betae* Keskin (Scholten & Lange, 2000). The disease has caused concern within the sugar beet industry as, once introduced, it is highly persistent and has a significant impact on yields: root losses of 50% have been observed, sugar content is reduced and some impurities, e.g., sodium, increase making sugar extraction more difficult. Rhizomania has a widespread distribution, i.e., much of continental Europe, Japan, the United States and China, and is often found in areas of intensive sugar beet growing. Despite its name, the affect of BNYVV on sugar beet is more apparent on the root than on the foliage. The virus induces prolific lateral root development ('beard' formation) and affects storage root size and development; severe infection can lead to root death. The virus can also cause chlorosis, and occasionally crinkling, wilting and necrosis within the veins of leaves (Asher, 1993; Scholten & Lange, 2000). It exists as three strain groups – designated A, B and P – each having different pattern of distribution and aggressiveness (Koenig et al., 1995; Heijbroek et al., 1999).

Currently, damping-off diseases caused by *A. cochlioides* and *P. ultimum* are primarily controlled by fungicides, e.g., hymexazol and thiram, applied to the surface of seeds. This, combined with cultural practices such as early planting into cool soils (Duffus & Ruppel, 1993), is relatively successful as emerging seedlings are vulnerable to these pathogens for a relatively short time only before natural resistance develops (Williams & Asher, 1996). The efficacy and relatively low cost of the fungicides has made their use commonplace, e.g., hymexazol was used to treat sugar beet seed on over 2.4 million ha in Europe in 1992 (Asher & Dewar, 1994). However, such widespread use of a limited range of fungicides could increase the potential for resistance to develop in the targeted pathogens (Walker, 2002). It has also reduced the incentive to find alternatives; currently, there are no sugar beet cultivars available in Europe that exhibit any appreciable resistance to damping-off diseases (Bosemark, 1993), although cultivars with resistance to *A. cochlioides* root rot have been developed in the United States (Schneider & Hogaboam, 1983; Duffus & Ruppel, 1993).

Adequate chemical control of diseases of mature roots is more difficult, not because of a lack of effective fungicides, but because of the need to

protect the enlarging root zone throughout the growing season (Asher, 1993). Therefore, greater emphasis has been placed on developing cultivars with resistance to Rhizoctonia and Rhizomania. Sugar beet germplasm with polygenic resistance to Rhizoctonia has been identified in selection programmes in the United States. Most resistant lines have been derived from open-pollinated sugar beet germplasm, but some have pedigrees derived from other sources including *Beta vulgaris* ssp. *maritima* (*B.v. maritima*; Panella, 1998). Several commercial cultivars with moderate levels of resistance have been developed from these sources (Duffus & Ruppel, 1993). Similarly, much effort has been put into controlling Rhizomania by developing resistance to the virus, BNYVV, or the vector, *P. betae* (Scholten & Lange, 2000). Several resistant sources have been identified within one or more of the *Beta* sections (*Beta*, *Corollinae*, *Procumbentes*): for example, *B.v. maritima* has been found to be a useful source of Rhizomania resistance (Lewellen et al., 1987; Whitney, 1989). In Europe, several partially resistant commercial sugar beet cultivars have been developed from such sources and are widely used (Asher, 1993). The earliest releases suffered from a yield penalty compared with conventional cultivars when grown in non-infested soil, but more recently available cultivars have largely overcome this problem (Scholten & Lange, 2000).

Although there have been significant advances in breeding for resistance to some soil-borne pathogens (e.g., Rhizomania), it is likely that further improvement in resistance to all these soil-borne diseases can be made through the utilization of novel resistance sources within the genus *Beta*. To achieve this, the European GENRES project '*Evaluation and enhancement of Beta collections for the extensification of agricultural production*' (GENRES-CT95-42) was initiated and subsequently managed by the BAZ Gene Bank in Germany. A consortium of 11 European agricultural research organisations and breeding companies investigated the potential for identifying resistance within the *Beta* germplasm working collection maintained by the BAZ Gene Bank during the project's lifetime (Luterbacher et al., 2004). In this paper, we report on the outcome of evaluation programmes undertaken to identify novel sources of resistance to seedling damping-off diseases caused by *A. cochlioides* and *P. ultimum*, Rhizoctonia root and crown rot, and Rhizomania. We examine the prospect of finding sources with 'multiple' resistance and how these sources are being utilised for crop improvement.

Materials and methods

The evaluation of *Beta* germplasm for resistance to soil-borne diseases was conducted by several collaborators. Seedling damping-off resistance testing was carried out at Broom's Barn Research Station, UK, whilst tests for Rhizoctonia resistance were managed by KWS SAAT AG (*KWS*), Germany. Two research institutes, the Istituto Sperimentale per le Colture Industriali (*ISCI*) in Italy and Plant Research International (*PRI*), the Netherlands, were responsible for undertaking testing for resistance to Rhizomania. The *Beta* germplasm used by each collaborator is indicated in the respective results tables (Tables 4, 6 and 7).

Seedling damping-off diseases

The methods used for evaluating resistance to the two damping-off pathogens were specially adapted from the bioassay tests of Williams & Asher (1996), to allow for comparison between very diverse *Beta* accessions. The *A. cochlioides* and *P. ultimum* cultures used in the tests were originally isolated from sugar beet seedlings growing on Brome Pin Field at Broom's Barn. For use as inoculum they were grown on a cornmeal/sand medium (Williams & Asher, 1996) for 4 and 3 weeks, respectively, at 22 °C in the dark.

Aphanomyces cochlioides

In total, 41 evaluation tests were conducted for *A. cochlioides* resistance. In each, 96 seeds (4 replicates of 24 seeds) of selected accessions were sown individually into single cells of a 36 mm deep multicellular tray (QPD104/5R 'Quick-pot propagation trays', PG Horticulture Ltd, UK) containing a commercial, partially sterilised soil (Hewitt Toptex Sportsturf, Petersfield Products, UK) thoroughly mixed with an *A. cochlioides* culture at 0.2% w/w. Subsequently, inoculated trays were placed in a controlled environment (CE) room maintained at 22 °C with a 16/8 h photoperiod and were watered daily.

Records of seedling emergence within each accession were made every 2–3 days after sowing and seedlings were thinned to a single plant per cell. After 4 weeks, seedlings were assessed for infection using the scale in Table 1, and the mean level of infection for each accession was determined from the four replicates. To compensate for any inter-experimental variation, the disease infection scores were adjusted relative to the standard sugar beet cultivar Saxon included in each test (Luterbacher et al., 2004) and

Table 1. Aphanomyces cochlioides disease assessment scale

Score	Description
0	Healthy seedlings with no lesions on aerial or subterranean tissue
1	Large seedlings with very small lesions, mostly on subterranean tissue
2	Large seedlings with large apparent lesions, either on aerial or subterranean tissue
3	Stunted seedlings with large lesions – death imminent
4	Seedling death occurred

subsequently transformed to a relative 1–9 resistance scale (RS).

Pythium ultimum

Twenty-seven tests were completed. The method of preparing test accessions was identical to *A. cochlioides* except that *P. ultimum* inoculum was incorporated at 0.75% w/w soil. Inoculated trays were maintained in a glasshouse at 22 °C with a 16/8 h photoperiod and watered daily. An uninoculated control of 48 seeds per accession (2 replications × 24 seeds) was also included so that seedling emergence (which varied between accessions) could be determined and hence estimates of pre-emergence losses from disease in inoculated trays could be made. An overall figure of % seedling loss for each accession was calculated from pre- and post-emergence infection data. These were adjusted and transformed to a relative RS value in the manner used for *A. cochlioides*.

Effect of seed vigour on A. cochlioides and P. ultimum resistance

Seedling emergence data collected for selected section *Beta* types (where >60 accessions were tested) were analysed to determine if differences in seed vigour within each influenced resistance expression. The degree of correlation between three components of seed vigour (a) seed viability (defined as the number of seeds producing seedlings), (b) seed productivity (the mean number of seedlings produced by each seedball) and (c) the rate of seedling emergence (% of seed producing seedlings in the first week after sowing), and the level of infection (using adjusted, but untransformed, mean infection scores or % seedling infection from the *A. cochlioides* and *P. ultimum* tests, respectively) was estimated for each section *Beta* type tested.

Rhizoctonia root and crown rot

Evaluation tests were conducted in the field (USA) and the glasshouse (Germany). Glasshouse testing was confined to annual types of section *Beta* plus accessions of the sections *Corollinae* and *Procumbentes*, whilst all others were tested in the field.

Glasshouse tests

Five glasshouse tests were carried out from 1996 to 2000. Accessions were sown initially in trays containing a standard peat-based compost and transplanted after 2 weeks into individual pots (7 cm wide × 18 cm deep) containing the same medium. Thirty-five seedlings (five randomised blocks of seven plants) of each accession were prepared. Seedlings were grown on for about 6 weeks and inoculated by placing 1/6 teaspoon (ca. 0.6 cm^3) of dried *R. solani* AG2-2 inoculum, previously prepared on sterilised barley grain medium using the method of Gaskill (1968), next to the root. Inoculated seedlings were maintained at high humidity and temperature (ca. 32 °C) and watered regularly for a further 8–10 weeks before roots were assessed for infection using the evaluation system of Hecker and Ruppel (1977) (Table 2). To ensure good root development, bolting plants of annual accessions were cut back weekly to prevent assimilates being diverted from the root.

Field tests

Five field tests were conducted, in 1996 (45 accessions tested), 1997 (90 tested), 1998 (90 tested), 1999 (59 tested) and 2000 (45 tested). Accessions were sown directly into the soil between mid-April and May, and thinned shortly after emergence. A total of 60–80 plants per accession (arranged in four replicates of 15–20

Table 2. Rhizoctonia disease assessment scale

Score	Description
0	Plants without visible symptoms; healthy
1	Blue-grey lesions observed, not only superficial – <5% of beet surface covered with lesions
2	5–10% of beet surface covered with lesions
3	10–25% of beet surface covered with lesions
4	25–50% of beet surface covered with lesions
5	50–75% of beet surface covered with lesions
6	>75% of beet surface covered with lesions
7	Beet completely rotted, leaves dead

plants) were grown on until the foliage almost covered the furrows (ca. 8–10 weeks after sowing). Plants were inoculated by placing 1/6 teaspoon (ca. 0.6 cm^3) of dried *R. solani* AG2-2 inoculum in the crown of the plant. Plants were sprinkle-irrigated for 4 h immediately after inoculation, and for 2 h per day thereafter, for 5–6 days. Individual plants were assessed for Rhizoctonia infection ca. 8–10 weeks after inoculation (Table 2). A mean (%) infection level was calculated from these values. This was subsequently adjusted relative to the susceptible control and transformed to a relative 1–9 scale. Two inbred KWS sugar beet breeding lines were used as a standard susceptible and resistant control, respectively, in all glasshouse and field trials; these were treated in the same manner as the test accessions.

Rhizomania

Evaluation tests were conducted between 1997 and 2001 at two research centres. Evaluation of predominately biennial types in section *Beta* was conducted on highly infested Rhizomania soils on land at Anzola nell'Emilia, Bologna, Italy by ISCI (1997–2000). Glasshouse evaluations, undertaken by PRI, the Netherlands (1998–2001) tested eleven biennial section *Beta* species selected from the Italian field trials, plus all section *Corollinae*, *Procumbentes* and annual section *Beta* accessions.

Field evaluations

Rhizomania field evaluations were conducted on the same severely infested field in 1997 (110 accessions tested), 1998 (74 tested), 1999 (110 tested) and 2000 (106 tested). Untreated seed of each accession was sown in the field between mid-March and mid-April (except 1999, where sowing occurred in May due to wet weather). Seedlings were thinned post-emergence to 11–15 evenly spaced plants per row (except 1997 where 28 plants were left) in 2–3 rows for each accession; on average throughout field testing, 38 plants of each accession were evaluated for Rhizomania resistance. All rows were subject to the normal agronomic practices but no artificial watering was supplied. The stalks of bolting plants were cut back in accordance with local laws to protect sugar beet seed crops growing in the Emilia-Romagna region (Luterbacher et al., 2004). The roots of plants of each accession were assessed for infection in August/September of each year (except 1999, where assessment occurred in October to compensate for the late sowing), using an assessment scale (Table 3) developed specifically to accommodate the diversity of *Beta* species. In 1997, accessions were assigned a mean score based on a collective assessment of all roots in a row. In other years, roots in each row were assessed individually and a mean resistance score calculated. In 1998, assessment was limited to a single row as severe rainfall prevented the second row from being harvested. Standard susceptible (cv. Asso) and resistant (cv. Rizor or cv. Dorotea) sugar beet cultivars were included in all trials.

Table 3. Rhizomania disease assessment scale for field evaluations

Score	Description
1	Plants with healthy roots (no 'bearding' or discoloration)
3	Roots with limited bearding and little discoloration
5	Roots with moderate bearding and discoloration
7	Roots with severe bearding, necrotic and highly discoloured
9	Plants dead; roots necrotic and rotten

Glasshouse evaluations

Ten experiments were conducted using a method adapted from Paul et al. (1992a). During 1998–1999, 24 plants (three blocks of eight plants) of each accession were tested for BNYVV resistance. In subsequent tests, 12 plants (two blocks of six plants) were used, as results indicated that numbers could be reduced without loss of accuracy. Seeds of each accession were sown in coarse sand previously heat sterilised at 105 °C. Emerged seedlings were transplanted into a mixture of heat-sterilised sand and a field soil severely infested with BNYVV at a ratio of 9:1. Seedlings were grown on for 5 weeks in a glasshouse maintained at 22 °C during the day and 17 °C at night, then sampled and assayed for virus content by ELISA using the method described by Paul et al. (1992a). Individual plants were assigned a RS using a linear 1–9 scale based on the range of ELISA readings observed in each test. Consistency between individual ELISA plates within each test was achieved by relating the results to previously identified resistant (cv. Holly) and susceptible (cv. Univers) standards on each plate: overall, the resistant and susceptible standards had mean RS values of 3 and 7, respectively.

Results from both the Rhizomania field and glasshouse tests were adjusted relative to the susceptible control to minimise any inter-experimental error between tests and analysed accordingly. Subsequently, comparisons of 18 accessions tested at both sites were made to determine how results correlated between an artificial and a natural environment.

Data interpretation and analysis

Analyses of resistance responses to each pathogen were conducted using the hierarchical approach described in Luterbacher et al. (2004). Assessments of multiple resistance were limited to the 514 accessions tested against all the soil-borne diseases. Again, the approach to analysing accessions for multiple resistance followed that used by Luterbacher et al. (2004). All statistical analyses were performed using GENSTAT version 6.

Results

Seedling damping-off

In total, 600 and 597 accessions were evaluated for resistance to *A. cochlioides* and *P. ultimum*, respectively. Results fitted a normal distribution (Figure 1a) with overall mean RS values of 5.0 and 4.6, respectively.

Different patterns of resistance were observed within the three sections of the genus *Beta* to both pathogens (Table 4); section *Corollinae* species (*B. corolliflora*, *B. macrorhiza*, *B. lomatogona*, *B. intermedia* and *B. trigyna*) were the most resistant to *A. cochlioides* with 93% of accessions having RS scores ≤ 2. Accessions of the sections *Beta* and *Procumbentes* exhibited similar levels of susceptibility. By contrast, the section *Corollinae* accessions were relatively susceptible to *P. ultimum* whilst section *Beta* and *Procumbentes* accessions exhibited greater resistance, both collectively and individually, to this pathogen. A single accession of *B. patellaris* had an RS of 2.

Within the section *Beta*, significant differences in *A. cochlioides* resistance between types were observed ($\chi^2 = 35.8$; $P < 0.001$) with the greatest resistance

Table 4. Summary of resistance to seedling damping-off diseases within the genus *Beta*

	A. cochlioides[a]				*P. ultimum*[a]			
	BB–CE ROOM[b]				BB–GLASS[b]			
Beta section/species	*n*[c]	Mean[d] RS	Range RS	*n* RS[e] 1–2	*n*	Mean RS	Range RS	*n* RS 1–2
Sections of the genus Beta								
Corollinae	15	1.5	1–4	14	12	5.3	4–6	0
Procumbentes	10	4.2	1–9	0	10	4.1	2–6	1
Beta	575	5.1	3–7	6	575	4.5	1–9	35
Species/sub-species/cultivars of the section Beta								
Wild types								
B. macrocarpa	13	3.9	2–7	1	13	5.1	1–9	1
B. patula	1	3.0	3	0	1	4.0	4	0
B. vulgaris spp.	53	4.9	1–9	1	53	4.9	2–9	3
B.v. adanensis	13	5.2	3–7	0	13	4.4	2–9	1
B.v. maritima	159	4.6	1–9	5	159	4.2	1–9	16
Cultivated types								
Fodder beet	61	5.2	3–9	0	61	4.8	3–9	0
Garden beet	121	5.4	2–9	1	121	4.6	1–9	8
Leaf beet	123	5.5	3–9	0	123	4.5	2–9	2
Sugar beet	31	5.0	3–7	0	31	4.7	2–7	1
χ^2 value[f]		35.8				14.6		
		<0.001				<0.05		

[a]Diseases.
[b]Test centre/method: BB, Broom's Barn; CE ROOM, evaluations conducted in controlled environment room; GLASS, evaluations conducted in glasshouse.
[c]*n* = number of accessions tested.
[d]RS = mean and range of resistance scores (1–9 scale: 1 = very resistant and 9 = very susceptible).
[e]*n* RS 1–2 = number of very highly resistant accessions within section/species/subspecies/cultivar of *Beta*.
[f]χ^2 value and probability for comparisons between section *Beta* types (not including *B. patula*).

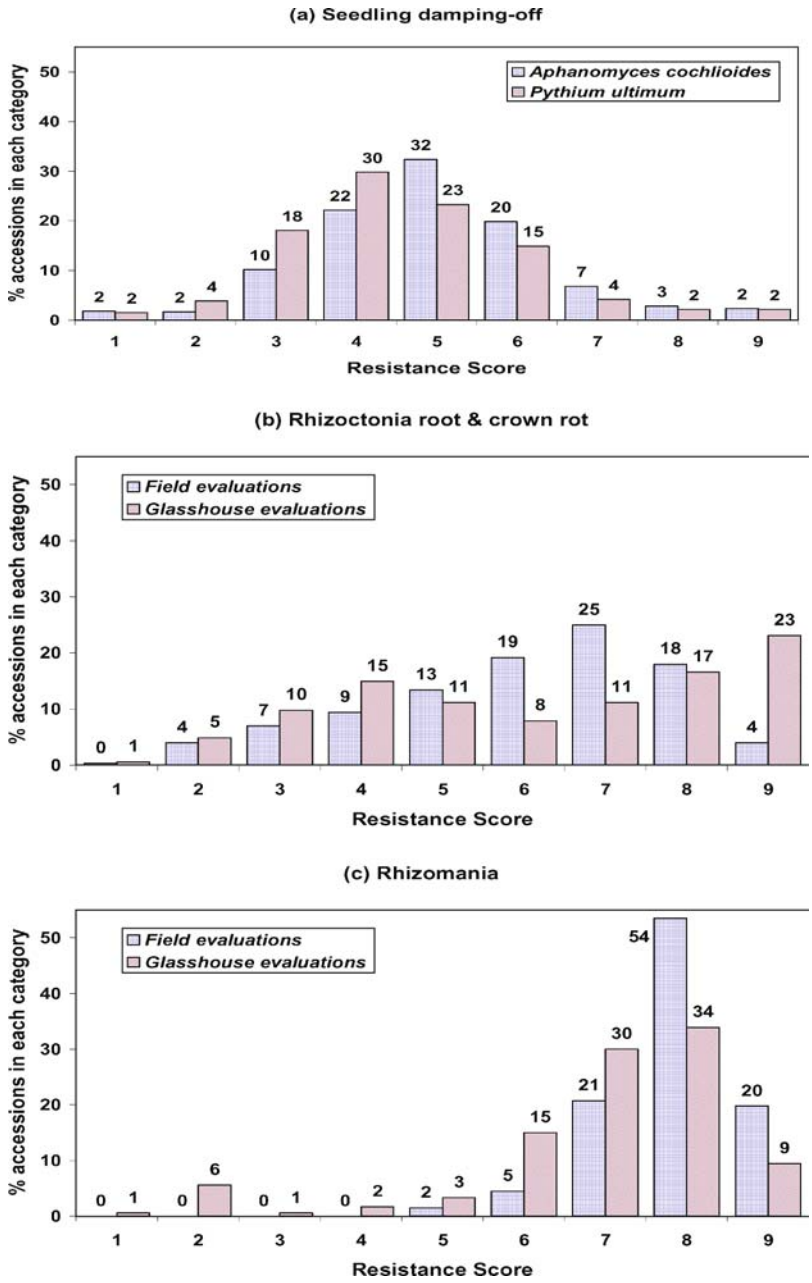

Figure 1. Frequency distribution of resistance in genus *Beta* accessions to four soil-borne diseases.

apparent in the wild species *B. macrocarpa*, *B.v. maritima* and *B. patula*. A single garden beet accession was the sole example of high resistance within the cultivated beet. Overall, there were significant differences ($\chi^2 = 14.6$; $P < 0.05$) in resistance to *P. ultimum*, with *B.v. maritima* and *B. macrocarpa* representing the most resistant and susceptible types (the lone *B. patula* accession had an RS value of 4.0): collectively there was little difference between the cultivated beets. However, these differences were not wholly reflected in the frequency of highly resistant accessions (RS $\leq$ 2) as they were more evenly distributed between section *Beta* types. *B.v. maritima* and garden beet had the highest levels of resistance in wild and cultivated

Table 5. Correlations between seed vigour and *A. cochlioides* and *P. ultimum* infection severity

Section *Beta* Type	Accession number	Correlation coefficients[a]		
		% Seed viability[b]	% Seedling emergence: Wk 1[c]	Seed productivity[d]
Aphanomyces cochlioides				
Fodder beet	61	0.264*	0.256*	−0.129
Garden beet	117	0.201*	0.191*	−0.002
Leaf beet	121	−0.006	0.093	0.268**
B.v. maritima	147	0.218**	0.257**	0.179*
Pythium ultimum				
Fodder beet	61	−0.066	−0.004	−0.087
Garden beet	121	−0.048	0.111	0.011
Leaf beet	123	−0.061	0.004	0.128
B.v. maritima	159	0.141	0.257***	−0.07

*0.05.
**0.01.
***0.001.
[a]Correlation coefficients calculated using GENSTAT 6.
[b]% seed viability = % of seeds producing viable seedlings.
[c]% seedling emergence: week 1 = % of seed producing viable seedlings one week after sowing.
[d]seed productivity = mean number of seedlings produced by each seedball.

beet respectively. The standard susceptible sugar beet cultivar Saxon had mean RS values of 5 and 4 for *A. cochlioides* and *P. ultimum* resistance, respectively

Effect of seed vigour on resistance

Overall, analyses revealed that there were a much greater number of significant correlations between measures of seed vigour and resistance expression in the *A. cochlioides* tests than in *P. ultimum* tests (Table 5). Where significance was observed, correlations were positive, i.e. the greater the measure of vigour, the higher the levels of infection observed, but in general coefficients were low.

Rhizoctonia root and crown rot

In total, 329 and 368 *Beta* accessions were tested for Rhizoctonia resistance in the field and glasshouse, respectively. The distribution of results was skewed towards susceptibility (Figure 1b) with mean RS values of 6.0 and 6.3 observed for field and glasshouse, respectively. The bi-modal appearance of glasshouse data reflects the highly disparate results from the sections *Beta*, *Corollinae* and *Procumbentes* (Table 6). In field tests, apart from in 1996, relatively consistent levels of infection were achieved over the different years. Susceptible controls had average unadjusted RS values of 3.8 (1996), 8.3 (1997), 8.8 (1998) and 7.9 (2000). Collectively, section *Beta* accessions showed similar levels of *Rhizoctonia* infection and variability in glasshouse and field tests. Although generally susceptible, most section *Beta* accessions (mean RS 5.7) were more resistant than species of the sections *Corollinae* (mean RS 8.4) and *Procumbentes* (mean RS 8.9) in glasshouse tests.

Within the section *Beta*, leaf and sugar beets were generally more susceptible to *Rhizoctonia* than the other cultivated beets (χ^2 value 50.5; $P < 0.001$) in field tests, whilst *B. macrocarpa* and *B.v. adenensis* were the more susceptible wild species in the glasshouse (χ^2 value 44.6; $P < 0.001$). Despite the overall susceptibility of section *Beta* types, highly resistant accessions were identified, including garden beets (10% of accessions tested), fodder beets (2%) and unspecified *B. vulgaris* spp. (12%) from field tests, and *B.v. maritima* (10%) from the glasshouse tests. Interestingly, leaf beets performed better in glasshouse tests than in the field, despite evidence from the standards included in each test that the glasshouse was a more suitable environment for infection. The susceptible standards had similar mean RS values of 8, suggesting that inoculum levels were not a constraining factor in either environment, although results from the field were more variable over the 4 years of the tests, as reflected in the

Table 6. Summary of resistance to Rhizoctonia root and crown rot within the genus *Beta*

Test centre/method[a]	KWS–FIELD				KWS–GLASS			
Beta section/species	*n*[b]	Mean[c] RS	Range RS	*n* RS[d] 1–2	*n*	Mean RS	Range RS	*n* RS 1–2
Sections of the genus *Beta*								
Corollinae	nt[e]	–	–	–	54	8.4	6–9	0
Procumbentes	nt	–	–	–	12	8.9	8–9	0
Beta	329	6.0	1–9	16	302	5.7	1–9	21
Species/sub-species/cultivars of the section *Beta*								
Wild types								
B. macrocarpa	nt	–	–	–	15	8.2	6–9	0
B. patula	nt	–	–	–	1*	9.0	9	0
B. vulgaris spp.	9	5.6	2–8	1	79	6.2	2–9	3
B.v. adanensis	nt	–	–	–	12	7.8	5–9	0
B.v. maritima	1*	9.0	9	0	162	5.4	1–9	16
Cultivated types								
Fodder beet	*69*	*5.5*	*2–9*	*1*	*1**	*3.0*	*3*	*0*
Garden beet	120	5.4	1–9	12	3*	3.0	2–4	1
Leaf beet	98	6.7	3–8	0	28	4.0	2–6	1
Sugar beet	32	7.6	5–9	0	1*	4.0	4	0
χ^2 value[f]		50.5				44.6		
		<0.001				<0.001		

[a]KWS: KWS SAAT AG; FIELD: evaluations conducted in field; GLASS: evaluations conducted in glasshouse.
[b]n = number of accessions tested.
[c]RS = mean and range of resistance scores (1–9 scale: 1 = very resistant and 9 = very susceptible).
[d]nRS 1–2 = number of very highly resistant accessions within section/species/subspecies/cultivar of *Beta*.
[e]nt: not tested.
[f]χ^2 value and probability for comparisons between Section *Beta* types (*not including *Beta* types with fewer than five accessions).

coefficients of variation (field 28%; glasshouse 16%). However, the resistant standards had much higher levels of infection in the glasshouse (mean RS 5) than the field (mean RS 3), suggesting more conducive conditions for infection; again variation was greater in the field (% CV in field 56%; glasshouse 38%).

Rhizomania

In total, 400 and 180 accessions were tested for Rhizomania resistance in the field in Italy and in glasshouses in the Netherlands, respectively. Both sets of results were strongly skewed towards susceptibility (Figure 1c), with mean RS values of 7.8 and 6.8 observed in the field and glasshouse, respectively. In the field, susceptible controls had average unadjusted RS values of 8.0 (1997), 7.4 (1998), 6.9 (1999) and 6.9 (2000). Collectively, section *Beta* and *Corollinae* accessions were highly susceptible, with mean RS values of 7.2/7.8 and 6.8 respectively (Table 7), although a single accession of *B. lomatogona* had an RS of 2. However, all section *Procumbentes* accessions (*B. patellaris*, *B. procumbens* and *B. webbiana*) were found to be highly resistant (RS values $\leq$2). Despite the generally high susceptibility exhibited by section *Beta* accessions in the field, small but significant differences (χ^2 value 62.3; $P < 0.001$) were observed. *B.v. maritima* was less susceptible than the other types and, although no highly resistant (RS < 2) forms were observed, there were accessions which performed as well as the resistant standard (mean RS 5) in the field trials. There was also a single leaf beet accession that had a RS value of 5. The susceptible standard in the field trials had a RS of 8.

Significant differences in susceptibility were detected in the glasshouse tests, with leaf beets and two annual wild species, *B. macrocarpa* and *B.v. adenensis*, performing relatively better and worse, respectively, (χ^2 value 14.5; $P < 0.01$) than others.

Table 7. Summary of resistance to Rhizomania within the genus *Beta*

Test centre/method[a]	ISCI–FIELD				PRI–GLASS			
Beta section/species	*n*[b]	Mean[c] RS	Range RS	*n* RS[d] 1–2	*n*	Mean RS	Range RS	*n* RS 1–2
Sections of the genus *Beta*								
Corollinae	nt[e]	–	–	–	28	6.8	2–8	1
Procumbentes	nt	–	–	–	10	1.9	1–2	10
Beta	400	7.8	5–9	0	142	7.2	3–9	0
Species/sub-species/cultivars of the section *Beta*								
Wild types								
B. macrocarpa	1*	7.0	7	0	9	7.9	7–9	0
B. patula	nt	–	–	–	1*	8.0	8	0
B. vulgaris spp.	33	8.2	7–9	0	22	7.3	6–8	0
B.v. adanensis	nt	–	–	–	9	8.3	7–9	0
B.v. maritima	78	7.3	5–9	0	76	7.2	3–9	0
Cultivated types								
Fodder beet	*67*	*7.6*	*6–9*	*0*	*1**	*6.0*	*6*	*0*
Garden beet	99	8.1	7–9	0	6*	7.7	7–9	0
Leaf beet	99	8.1	5–9	0	18	6.9	5–8	0
Sugar beet	23	7.8	6–9	0	nt	–	–	–
χ^2 value[f]	62.3				14.5			
	$P < 0.001$				$P < 0.01$			

[a]ISCI: Istituto Sperimentale per le Colture Industriali, Italy; PRI: Plant Research International, the Netherlands; FIELD: evaluations conducted in field; GLASS: evaluations conducted in glasshouse.
[b]n = number of accessions tested.
[c]RS = mean and range of resistance scores (1–9 scale: 1 = very resistant and 9 = very susceptible).
[d]n RS 1–2 = number of very highly resistant accessions within section/species/subspecies/cultivar of *Beta*.
[e]nt: not tested.
[f]χ^2 value and probability for comparisons between section *Beta* types (*not including *Beta* types with fewer than five accessions). Data for *B. macrocarpa* and *B.v. adenensis* combined for analysis as no significant differences in resistance distribution were observed (Fisher exact test $P = 0.603$).

However, only within *B.v. maritima* were there individual accessions that had RS scores similar to that of the resistant standard (Holly). A more detailed analysis of the individual plant data from glasshouse tests indicated that there were high levels of segregation in some types and species within the sections *Beta* and *Corollinae*. Consequently, despite having relatively poor mean RS values, they contained a significant proportion (>30%) of highly resistant plants with RS $\leq$ 3. In total, 18 accessions from these sections were identified with this trait and included *B. vulgaris* spp. (two accessions), *B.v. vulgaris* 'leaf beet' (3), *B.v. maritima* (7), *B. corolliflora* (1), *B. macrorhiza* (1), *B. lomatogona* (3) and *B. intermedia* (1).

Comparisons of results of 18 accessions tested in both the field and glasshouse indicated that there was no correlations between RS values, either absolute (R^2 0.028; $P = 0.51$) or relative (Spearmans rank correlation: 16 d.f; $P = 0.558$), obtained in the two types of test.

Multiple resistance

The prospects of finding multiple resistance in a single accession are very limited at the highest resistance levels (RS $\leq$ 2; Table 8). No accession of any section within the genus *Beta* had resistance to more than two diseases at these levels and the numbers available, within the sections *Beta* and *Procumbentes*, were low (1–2%; Figure 2). No accessions within the section *Corollinae* had high resistance to more than one disease. At RS $\leq$ 4, it was possible to identify accessions (3–10%) within sections *Beta* and *Procumbentes* that showed resistance to three diseases (data not shown).

Table 8. Accessions showing high levels of resistance (RS $\leq$ 2) to more than one disease

IDBB Accession number	Section	Species or type	Country of origin	Resistance to:
2196	Beta	*maritima*	Greece	Pythium; Rhizoctonia
2220	Beta	*vulgaris*	Italy	Pythium; Rhizoctonia
3092	Beta	*maritima*	Greece	Pythium; Rhizoctonia
5763	Beta	*maritima*	France	Aphanomyces; Pythium
6801	Beta	*garden beet*	UK	Pythium; Rhizoctonia
8472	Beta	*maritima*	Turkey	Pythium; Rhizoctonia
6542	Procumbentes	*patellaris*	Spain	Pythium; Rhizomania

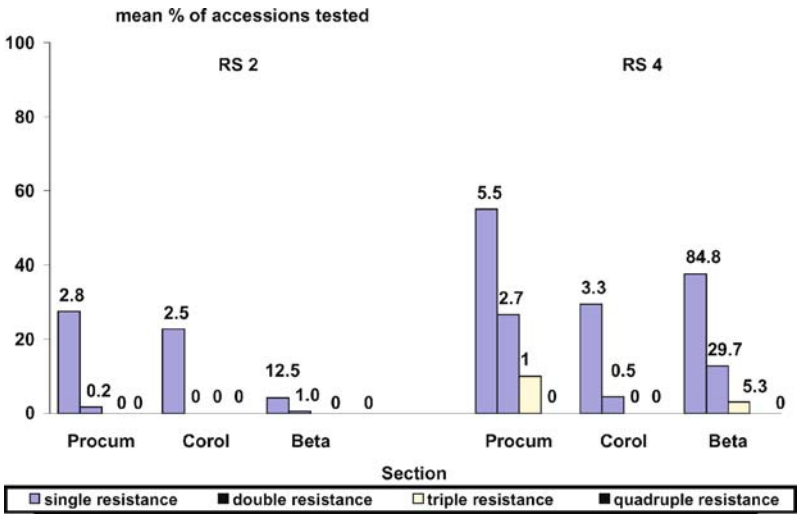

Figure 2. Multiple resistance to soil-borne diseases within the genus *Beta*. RS2 = mean % of accessions scoring RS $\leq$ 2; RS 4 = mean % of accessions scoring RS $\leq$ 4. Procum = Section *Procumbentes* accessions; Corol = Section *Corollinae* accessions; Beta = Section *Beta* accessions. Numbers heading each column are the mean number of accessions in that category.

No accessions of section *Corollinae* were found to be resistant to more than two diseases at this level.

Discussion

These evaluation tests have demonstrated that resistance to soil-borne diseases of sugar beet can be found within related species of the genus *Beta*, thus providing breeders with the opportunity to extend the gene pool utilised in modern breeding programmes. Resistant accessions of the section *Beta*, which can be utilised immediately in breeding programmes because of their compatibility with sugar beet, will be of greatest value. The relative success rate across all tests in selecting for resistance to soil-borne diseases within the section *Beta* was comparable to that achieved with foliar diseases (Luterbacher et al., 2004).

The most resistant of the cultivated beets were the garden beets which, apart from Rhizomania resistance, contained the largest number of very highly resistant (RS $\leq$ 2) accessions; this group appeared particularly promising as a novel source of Rhizoctonia resistance. Garden (or 'red') beet has been used as a source of resistance to Rhizoctonia before (Van Geyt et al., 1990). The leaf beets also contained a few examples that exhibited high levels of resistance to both *P. ultimum* damping-off and Rhizoctonia: three leaf beet accessions also contained highly resistant plants within populations segregating for Rhizomania resistance. No other cultivated type from the section *Beta* exhibited any significant resistance to Rhizomania.

Several accessions of *B.v. maritima* were identified as highly resistant to seedling damping-off (3–10% of accessions tested) and Rhizoctonia (10%). The value of

B.v. maritima has been noted before and it has already been widely used in breeding programmes in Europe and the United States (Van Geyt et al., 1990; Panella, 1998). This species also offered the most promise in relation to Rhizomania resistance within the section *Beta*; one accession was identified with a mean RS of 3, equivalent to that of the resistant control, but several more had a significant proportion of highly resistant plants (>30%) within segregating populations. This latter observation reinforces the need to use indicators of heterogeneity, e.g., standard deviations, or coefficients of variation, in conjunction with RS values to evaluate the worth of accessions, otherwise valuable resistance sources could be overlooked in such an outbreeding species (Luterbacher et al., 2004). The relative success of *B.v. maritima* in these tests confirms the usefulness of this species as a source of resistance to Rhizomania (Lewellen et al., 1987; Whitney, 1989; Lewellen and Whitney, 1993). One of the earliest partially resistant sugar beet cultivars, Alba, was derived from this wild species (Biancardi et al., 2002) and, more recently, the resistant *B.v. maritima* accession WB42 has been used in breeding programmes (Paul et al., 1993b; Scholten & Lange, 2000).

Other wild species within the section *Beta* exhibited a wider range of responses to soil-borne diseases. Some, such as *B. macrocarpa* and *B.v adenensis*, contained useful levels of resistance to the damping-off diseases. However, it is uncertain if these groups, which have relatively extreme features such as small size, relatively short annual life cycles (Buttler, 1977) and no agronomic characters of interest, would find favour with breeders, particularly if other sources are available. Another interesting group is the collection of highly variable accessions identified collectively as *B. vulgaris* spp. Some of these exhibited significant levels of resistance to seedling damping-off diseases and, more importantly, showed very useful resistance to Rhizoctonia. Most are largely uncharacterised, although some might be classified as 'weed' populations (as they were identified as *B. vulgaris* spp. *vulgaris*) derived from crosses between cultivated beet and *B.v. maritima*, which are common in some parts of Europe (Lange et al., 1999).

Overall, the sections *Corollinae* and *Procumbentes* showed less promise as resistance sources for soil-borne diseases than for leaf diseases (Luterbacher et al., 2004). However, accessions of the section *Corollinae* were generally highly resistant to *A. cochlioides*, whilst those of the section *Procumbentes* were highly resistant to Rhizomania. On first examination, the performance of section *Corollinae* (*B. corolliflora*, *B. intermedia*, *B. lomatogona*, *B. trigyna*, *B. macrorhiza*) to Rhizomania could be considered poor, particularly as others have reported this group to be resistant to both BNYVV and its vector *Polymyxa betae* (Fujisawa & Sugimoto, 1979; Paul et al., 1993a; Mesbah et al., 1997). Only one accession, of the species *B. lomatogona*, had a RS $\leq$ 2. However, closer examination of results within the section *Corollinae* revealed clear segregation within some accessions, with approximately 20% of accessions containing a significant proportion of plants (>30%) with RS values of 3 or better; therefore, accessions in this group cannot be disregarded as potential resistance sources. In the section *Procumbentes* resistance was more uniform within each accession, confirming previous observations (Fujisawa & Sugimoto, 1979; Paul et al., 1992b). No attempt was made here to determine whether the resistance was to the virus or its vector. Others have observed resistance to *P. betae* within the section *Procumbentes* (Paul et al., 1992b; Barr et al., 1995). However, the immediate use of section *Corollinae* and *Procumbentes* accessions for breeding purposes is unlikely due to their sexual incompatibility with sugar beet. This could be overcome by using genetic transformation techniques if the technical challenges of isolating and cloning the resistance genes involved could be surmounted (Scholten & Lange, 2000). Such an approach has reportedly been used successfully to isolate resistance genes to beet cyst nematode from a section *Procumbentes* accession (Cai et al., 1997)

The identification of a relatively large number of novel resistance sources to seedling damping-off caused by *A. cochlioides* and *P. ultimum* could initiate greater efforts to develop resistance to these diseases, particularly in Europe. The observation that sugar beet cultivars that germinate rapidly and show vigorous seedling growth suffer less from seedling diseases than slow-growing types (Leach, 1986) prompted the study on seed vigour characteristics and their influence on resistance to *A. cochlioides* and *P. ultimum*. Statistically significant, though generally low ($R < 0.3$), positive correlations were observed between some seed vigour characteristics and the level of infection by *A. cochlioides*, particularly among *B.v. maritima* accessions. With one exception this association was not observed with *P. ultimum* infection. Clearly, poor seed vigour was not leading to enhanced disease susceptibility in these tests; if anything, the reverse was the case, though the relationship was weak and may not be directly causal.

Given the importance of Rhizoctonia root and crown rot in the United States, and the increasing prevalence of the pathogen in Europe (Ayala-Garcia et al., 2001), the discovery of novel sources of resistance, particularly in related cultivated beets, may be valuable. As with the damping-off diseases, the morphology of *Beta* types had the potential to influence disease infection during testing: successful *Rhizoctonia* infection requires a well developed tap root whereas in annual species most assimilates are directed towards flowering rather than root formation, thus possibly modifying the nature of infection. This potential problem was minimised by conducting evaluation tests in the glasshouse where annual plants could be pruned to prevent flowering. Importantly, unpublished data (W. Beyer, personal communication) on fodder beet resistance indicated good correlations between glasshouse and field tests, although the evidence from the resistant and susceptible controls showed that the former environment generated more severe disease. Glasshouse tests were also more consistent than field tests, making comparisons between years more reliable.

Results of the Rhizomania evaluations indicated that little useful resistance could be detected from field or glasshouse tests using mean RS values alone. However, the use of a quantitative ELISA method in glasshouse tests provided much clearer evidence of segregation between individual plants of accessions than was achieved by visual assessments of root infection in the field, a feature that makes the former method valuable in detecting resistance in a potentially limited range of material. Unlike Rhizoctonia, comparison of results from field and glasshouse tests for Rhizomania resistance proved difficult; there was no correlation between resistance observed in 18 section *Beta* accessions tested in both environments. This has been shown before by Paul et al. (1993c), who demonstrated that the relative performance of susceptible and partially resistant sugar beet cultivars in infested fields can best be estimated by (1) the resistance observed in glasshouse experiments, (2) the yield and quality obtained in non-infested fields, and (3) the level of infection in the field. The disparity between glasshouse and field tests was probably related to the difficulty of ensuring uniform and optimum conditions of inoculum distribution, temperature and water levels in the field, factors that influence Rhizomania infection. Such influences can be more closely controlled in the glasshouse and it is clear that Rhizomania evaluation programmes are best conducted under controlled conditions to optimise disease pressure, and using quantitative ELISA to discriminate individual plants.

The goal of a universal resistance source for all important sugar beet diseases is not realistic. However, the results of this work on root diseases, and the previous study on leaf diseases (Luterbacher et al., 2004) have shown that breeders should be able to find single accessions with high resistance (RS $\leq$ 2) to at least two diseases. A minor reduction in the desired level of resistance will greatly expand the choice available. Such choices, combining resistance to particular foliar and soil-borne diseases, can be made from the data held on the International Database for Beta (IDBB) website: www.genres.de/idb/beta. Most importantly, it should be possible to find multiple resistances within the section *Beta*, a group which are sexually compatible with sugar beet (Frese et al., 2001), thus making it possible to introgress genes using conventional methods. The success of such crosses will depend on the linkage relationships between the disease resistance genes and those responsible for other agronomic traits, e.g., yield. Prospects for using accessions of the sections *Corollinae* and *Procumbentes* are more distant because of problems of sexual incompatibility, but they may prove a valuable source of highly expressed resistance to some diseases in the future.

Acknowledgments

The technical assistance of Jo Smith, Kathy Bean and Kevin Sawford is gratefully acknowledged. This work was supported by the Commission of the European Communities and funded through the Council Regulation (EC) No. 1467/94 on The Conservation, Characterisation, Collection and Utilisation of Genetic Resources in Agriculture. Additional funding in the UK was provided by the British Beet Research Organisation. Broom's Barn Research Station is a division of Rothamsted Research and receives grant-aided support from the Biotechnology and Biological Sciences Research Council, UK.

References

Asher, M.J.C., 1993. Rhizomania. In: D.A. Cooke & R.K. Scott (Eds.), The Sugar Beet Crop, pp 311–346. Chapman & Hall, London.

Asher, M.J.C. & A.M. Dewar, 1994. Control of pests and diseases in sugar beet by seed treatments. In: T. Martin (Ed.), Seed

Treatment: Progress and Prospects, pp 151–158. BCPC monograph 57. BCPC publications, UK.

Ayala-Garcia, J., G. Büttner, H. Gutiérez, W. Heijbroek, P. Ioannides, M. Nihlgaard, M. Richard-Molard, L. Panella, V. Rossi, H. Rössner, J.H.M. Schneider & A. Wauters, 2001. Integrated control of *Rhizoctonia* root rot – First results of an I.I.R.B. trial series. In: Proceedings of the 64th Congress of the Institut International de Recherches Betteravières, pp 397–400. IIRB, Brussels.

Barr, K.J., M.J.C. Asher & B.G. Lewis, 1995. Resistance to *Polymyxa betae* in wild *Beta* species. Plant Pathol 44: 301–307.

Biancardi, E., R.T. Lewellen, M. De Biaggi, A.W. Erichsen & P. Stevanato, 2002. The origin of Rhizomania resistance in sugar beet. Euphytica 127: 383–397.

Boerema, G.H. & A.A. Verhoeven, 1976. Check-list for scientific names of common parasitic fungi. Series 2a. Fungi of field crops: Beet potatoes, caraway, flax and oilseed poppy. Neth J Plant Pathol 82: 193–214.

Bosemark, N.O., 1993. Genetics and breeding. In: D.A. Cooke & R.K. Scott (Eds.), The Sugar Beet Crop, pp 66–119. Chapman & Hall, London.

Buttler, K.P., 1977. Variation in wild populations of annual *Beta* (*Chenopodiaceae*). Plant Syst Evol 128: 123–136.

Cai, D., M. Kleine, S. Kifle, H.-J. Harloff, N.N. Sandel, K.A. Marcker, R.M. Klein-Lankhorst, E.M.J. Salentijn, W. Lange, W.J. Stiekema, U. Wyss, F.M.W. Grundler & C. Jung, 1997. Positional cloning of a gene for nematode resistance in sugar beet. Science 275: 832–834.

Dunning, R.A. & W. Heijbroek, 1981. Improved plant establishment through better control of pest and disease damage. In: Proceedings of the 44th Congress of the Institut International de Recherches Betteravières, pp 37–59. IIRB, Brussels.

Duffus, J.E. & E.G. Ruppel, 1993. Diseases. In: D.A. Cooke & R.K. Scott (Eds.), The Sugar Beet Crop, pp 346–427. Chapman & Hall, London.

Frese, L., B. Desprez, & D. Ziegler, 2001. Potential of genetic resources and breeding strategies for base-broadening in *Beta*. In: H.D. Cooper, C. Spillane & T. Hodgkin (Eds.), Broadening the Genetic Base of Crop Production, pp 295–309. IPGRI/FAO, Rome.

Fujisawa, I. & T. Sugimoto, 1979. The reaction of some beet species of the sections *Patellares*, *Corollinae* and *Vulgares* to Rhizomania of sugar beet. Proc Sugar Beet Res Assoc Jpn 21: 31–38.

Gaskill, J.O., 1968. Breeding for Rhizoctonia resistance in sugar beet. J Am Soc Sugar Beet Technol 15: 107–119.

Hall, G., 1989. *Aphanomyces cochlioides*. CMI Descriptions of pathogenic fungi and bacteria, No. 972. Mycopathology 106: 185–186.

Hecker, R.J. & E.G. Ruppel. 1977. Rhizoctonia root-rot resistance in sugar beet: Breeding and related research. J Am Soc Sugar Beet Technol 19: 246–256.

Heijbroek, W., P.M.S. Musters & A.H.L. Schoone, 1999. Variation in pathogenicity and multiplication of beet necrotic vein virus (BNYVV) in relation to resistance of sugar-beet cultivars. Eur J Plant Pathol 105: 397–405.

IDBB, 2003. International Database for Beta (IDBB). Website: www.genres.de/idb/beta.

Koenig, R., P. Ludigr ddecke & A.M. Haeberle, 1995. Genome differences between beet necrotic yellow vein virus (BNYVV) sources from different parts of the world. In: Proceedings of the 58th Congress of the Institut International de Recherches Betteravières, pp 271–278. IIRB, Brussels.

Lange, W., W.A. Brandenburg & Th.S.M. De Bock, 1999. Taxonomy and cultonomy of beet (*Beta vulgaris* L.). Bot J Linn Soc 130: 81–96.

Leach, L.D., 1986. Seedling diseases. In E.D. Whitney & J.E. Duffus (Eds.) Compendium of Beet Diseases and Insects, pp 5–8. American Phytopathological Society, St. Paul, Minnesota.

Lewellen, R.T. & E.D. Whitney, 1993. Registration of germplasm lines developed from composite crosses of sugar beet × *Beta maritima*. Crop Sci 33: 882–883.

Lewellen, R.T., I.O. Skoyen & A.W. Erichsen, 1987. Breeding sugar beet for resistance to Rhizomania: Evaluation of host-plant reactions and selection for, and inheritance of, resistance. In: Proceedings of the 50th Congress of the Institut International de Recherches Betteravières, pp 139–156. IIRB, Brussels.

Luterbacher, M.C., M.J.C. Asher, E. DeAmbrogio, E. Biancardi, P. Stevenato & L. Frese, 2004. Sources of resistance to diseases of sugar beet in related *Beta* germplasm: I. Foliar diseases. Euphytica 139: 105–121.

Mesbah, M., O.E. Scholten, Th.S.M. De Bock & W. Lange, 1997. Chromosome localisation of genes for resistance to *Heterodera schachtii, Cercospora beticola* and *Polymyxa betae* using sets of *Beta procumbens* and *B. patellaris* derived monosomic additions in *B. vulgaris*. Euphytica 97: 117–127.

O'Sullivan, E. & J.A. Kavanagh, 1992. Characteristics and pathogenicity of *Pythium* spp. associated with damping-off of sugar beet in Ireland. Plant Pathol 41: 582–590.

Panella, L., 1998. Screening and utilizing *Beta* genetic resources with resistance to Rhizoctonia root rot and Cercospora leaf spot in a sugar beet breeding programme. In: L. Frese, L. Panella, H.M Srivastava & W. Lange (Eds.), Report of the 4th International Beta Genetic Resources Workshop & World Beta Network Conference, Izmir, Turkey. IPGRI, Rome.

Papavizas, G.C. & W.A. Ayers, 1974. *Aphanomyces* species and their root diseases in pea and sugar beet – A review. USDA Technical Bulletin 1845. Washington, DC.

Paul, H., B. Henken & M.F.J. Alderlieste, 1992a. A greenhouse test for screening sugar-beet (*Beta vulgaris*) for resistance to beet necrotic yellow vein virus (BNYVV). Neth J Plant Pathol 98: 65–75.

Paul, H., B. Henken, Th.S.M. De Bock & W. Lange, 1992b. Resistance to *Polymyxa betae* in *Beta* species of the section *Procumbentes*, in hybrids with *B. vulgaris* and in monosomic chromosome additions of *B. procumbens* in *B. vulgaris*. Plant Breed 109: 265–273.

Paul, H., B. Henken, O.E. Scholten, Th.S.M. De Bock & W. Lange, 1993a. Variation in the level of infection with *Polymyxa betae* and its effect on infection by beet necrotic vein virus in beet accessions of the section *Beta* and *Corollinae*. In: Proceedings of the Second Symposium of the International Working Group on Plant Viruses with Fungal Vectors, pp. 133–136. Montreal, Canada.

Paul, H., B. Henken, O.E. Scholten & W. Lange, 1993b. Use of zoospores of *Polymyxa betae* in screening beet seedlings for resistance to beet necrotic vein virus. Neth J Plant Pathol 99 (3 Suppl): 151–160.

Paul, H., F.A. Van Eeuwijk & W. Heijbroek, 1993c. Multiplicative models for cultivar by location interaction in testing sugar beets for resistance to beet necrotic yellow vein virus. Euphytica 71: 63–74.

Payne, P.A., M.J.C. Asher & C.D. Kershaw, 1994. The incidence of *Pythium* spp. and *Aphanomyces cochlioides* associated with the sugar beet growing regions of Britain. Plant Pathol 43: 300–308.

Schneider, C.L. & G.J. Hogaboam, 1983. Evaluation of sugarbeet breeding lines in greenhouse tests for resistance to *Aphanomyces cochlioides*. J Am Soc Sugar Beet Technol 22: 101–107.

Schneider, C.L. & E.D. Whitney, 1986. Rhizoctonia root and crown rot. In: E.D. Whitney & J.E. Duffus (Eds.), Compendium of Beet Diseases and Insects, p. 21. American Phytopathological Society, St. Pauls, Minnesota.

Scholten, O.E. & W. Lange, 2000. Breeding for resistance to Rhizomania in sugar beet: A review. Euphytica 112: 219–231.

Van Geyt, J.P.C., W. Lange, M. Oleo & T.S.M. De Bock, 1990. Natural variation within the genus *Beta* and its possible use for breeding sugar beet: A review. Euphytica 49: 57–76.

Walker, R., 2002. Development of bacterial seed treatments for the control of *Aphanomyces cochlioides* on sugar beet. Ph.D Thesis, University of Nottingham, UK.

Whitney, E.D., 1989. Identification, distribution, and testing for resistance to Rhizomania in *Beta maritima*. Plant Dis 73: 287–290.

Williams, G.E. & M.J.C. Asher, 1996. Selection of rhizobacteria for the control of *Pythium ultimum* and *Aphanomyces cochioides* on sugar-beet seedlings. Crop Prot 15: 479–486.