Chapter 8
Source of Useful Traits

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Abstract In the late 1800s, there already was speculation that *Beta maritima* might provide a reservoir of resistance genes that could be utilized in sugar beet breeding. European researchers had crossed *Beta maritima* and sugar beet and observed many traits in the hybrid progeny. It is impossible to estimate how widely *Beta maritima* was used in the production of commercial varieties, because most of the germplasm exchanges were informal and are difficult to document. Often these crosses of sugar beet with sea beet germplasm contained undesirable traits, e.g., annualism, elongated crowns, fangy roots, high fiber, red pigment (in root, leaf, or petiole) and much lower sucrose production. It is believed that lack of acceptance of *Beta maritima* as a reservoir of genes was because most of the evaluations of the progeny were done in early generations: The reactions of the hybrids *vulgaris* × *maritima* were not impressive, and it is clear now that they were not adequately studied in the later generations.

Keywords Disease resistance · Rhizomania · Cercospora · Nematodes · Drought · Salt stress · Root rot · Curly top · Virus yellows · Powdery mildew · *Polymyxa betae*

Contrary to other species of the genus *Beta*, the evolutionary proximity between the sea beet and the cultivated types favors casual crosses (Hjerdin et al. 1994). Important characters of resistance to diseases, currently present in cultivated varieties, have been isolated from wild material (Table 8.1). According to several authors, *Beta maritima* is also an important means to increase the genetic diversity of cultivated types, now rather narrow from a domestication bottleneck and continuous selection for improvement of production and quality traits (Bosemark 1979; de Bock 1986; Doney 1998;

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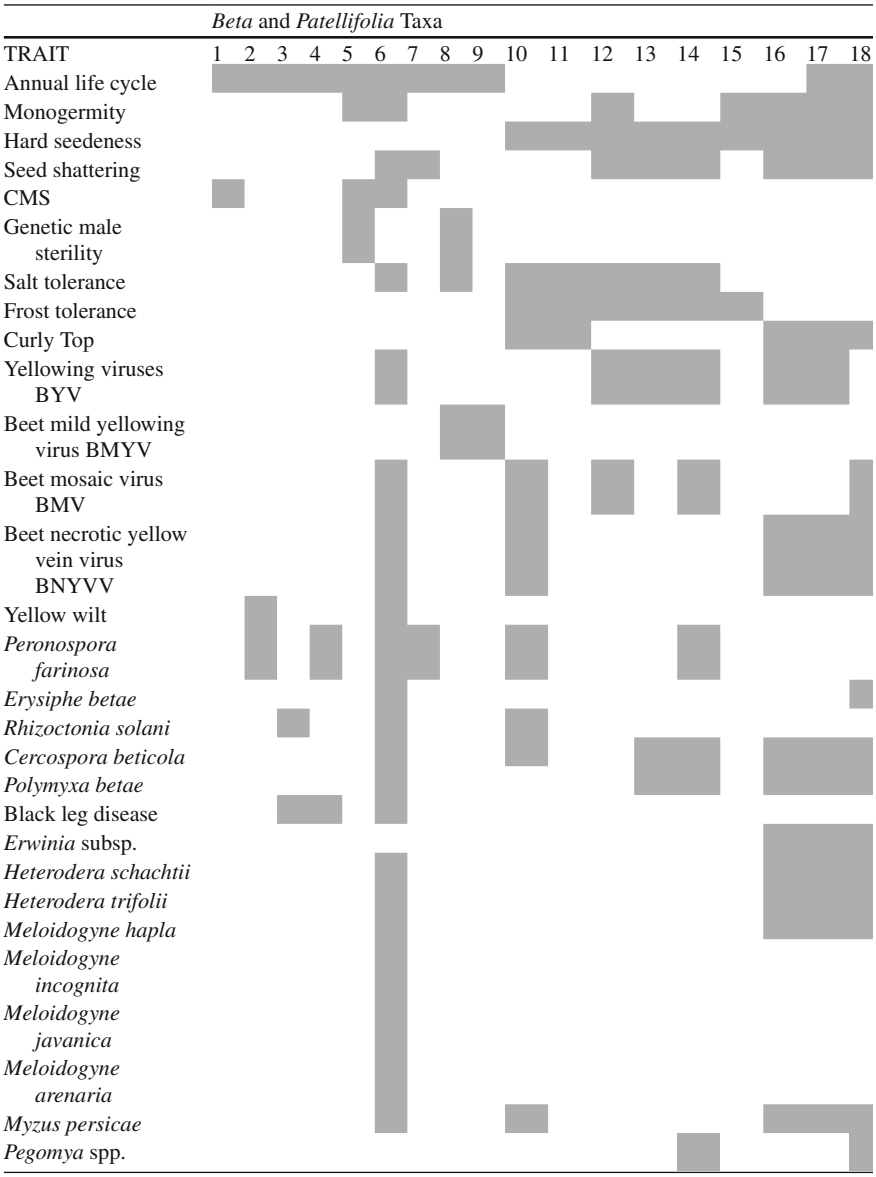
Table 8.1 Useful traits in the Genus Beta (Frese 2011, personal communication)



1. *Beta vulgaris* subsp. *vulgaris* (*Bv*), 2. *Bv* leaf beet group, 3. *Bv* garden beet group, 4. *Bv* fodder beet, group, 5. *Bv* sugar beet group, 6. *Beta vulgaris* subsp. *maritima*, 7. *Bv* subsp. *adanensis*, 8. *Beta* (*Beta*) *macrocarpa*, 9. *Beta patulaI*, 10. *Beta corolliflora*, 11. *Beta macrorhiza*, 12. *Beta lomatogona*, 13. *Beta intermedia*, 14. *Beta trigyna*, 15. *Beta nana*, 16. *Patellifolia* (*Patellifolia*) *procumbens*, 17. *Patellifolia webbiana*, 18. *Patellifolia patellaris*

Jung et al. 1993; McGrath et al. 1999). This is especially true of sugar beet varieties, due to the common origin from the White Silesian Beet (Achard 1803; Fischer 1989), whose variability, according to Evans and Weir (1981), could have been enhanced by crosses with North Atlantic sea beet. Moreover, this narrowing of genetic diversity was increased through the widespread use both of Owen's cytoplasmic genetic male sterility (CMS) and the monogermy trait transferred to the current varieties by means of inbred lines (Jung et al. 1993; Owen 1945; Savitsky 1952). The attempts to transfer useful traits from sea beet are still underway. In a recent paper, Campbell (2010) described the performance of four crosses between *Beta maritima* and commercial varieties, which performed quite well, both in yield and resistance to some diseases (Rhizoctonia root and crown rot, rhizomania, powdery mildew, Cercospora leaf spot, Aphanomyces root rot, and Fusarium yellows).

However, the association of negative characters with the traits to be transferred often has made the improvement of cultivated genotypes difficult (Coons 1975; Mita et al. 1991). The major problems associated with such hybridizations are (1) the dominance of the annual life cycle in some wild forms, (2) the very bad shape of the root, (3) woodiness of roots, (4) elongated and multiple crowns, (5) low sugar content, (6) poor root yield, (7) low processing quality (Oltmann et al. 1984), (8) growth habit of the seed stalk, (9) prostrate seed stalk, (10) early seed shattering, etc. (Rasmussen 1932; van Geyt et al. 1990). Similar problems also arise when crossing sea beet with fodder, leaf, and garden beets. Several backcrosses and repeated selection cycles are necessary before such hybrids can acquire a satisfactory morphology and sufficient agronomic qualities (de Bock 1986; Munerati 1932).

The ancestors of the modern crops are defined as "crop wild relatives" (CWR), which also include other species closely related to them (Hajjar and Hodgkin 2007). Their commercial worth is invaluable (www.biodiversityinterna-tional.org). Many wild species, including *Beta maritima*, are threatened through reduction, degradation, or fragmentation of their habitat. Therefore, we need to identify not only the species to be protected in their respective areas but also the facilities for their in situ and ex situ conservation (Frese and Germeier 2009). Maxted et al. (2006) subdivided the species of the genus *Beta* into gene pools (GP) (Harlan and de Wet 1971) according to the difficulty of using the pool as a source of traits for the beet crops: (1) primary gene pool includes the cultivated forms (GP-1A) and the wild or weedy forms of the crop (GP-1B); (2) secondary gene pool (GP-2) includes the less closely related species from which gene transfer to the crop is difficult, but possible, using conventional breeding techniques; and (3) tertiary gene pool (GP-3) includes the species from which gene transfer to the crop is impossible or requires sophisticated techniques. Consequently, *Beta maritima* was classified as explained in Table 6.2. A PGR Forum was organized both to better define CWR and to compile a list of the more endangered species (Ford-Lloyd et al. 2009).

8.1 Resistances to Biotic Stresses

Most of the breeding work with *Beta maritima* has been to use it as a source of resistance to varied pests and diseases. Lewellen (1992) theorized that because the sugar beet and the white Silesian fodder beet source were developed and produced in the temperate climate of Northern Europe, there was less pressure to maintain plant resistance to biotic stress because of the mild disease incidence and "As a consequence, this narrowly based germplasm may never have had or may have lost significant levels of genetic variability for disease resistance or the factors that condition disease resistance occur in the germplasm at low frequencies" (Lewellen 1992). However, once sugar beet production moved out of Northern Europe, east into Russia and Asia, south into Mediterranean Europe and North Africa, and west into England and North and South America, many new diseases endemic to these areas limited production of sugar beet (Lewellen 1992).

The first documented instance of successfully transferring disease resistance from sea beet to sugar beet was by Munerati using sea beet growing in the Po Delta as a source of resistance to Cercospora leaf spot (Munerati et al. 1913a). Following Munerati's success, other European researchers began working with *Beta maritima* as a source of disease resistance (Margara and Touvin 1955; Schlösser 1957; Zossimovich 1939; Asher et al. 2001a). Nonetheless, for many of the reasons enumerated by Coons (1975), it is unlikely that much of this effort resulted in commercial varieties with sea beet in their genetic background, and due to the proprietary status of commercial germplasm, this information has not found its way into the literature.

8.1.1 Yellowing Viruses

Virus yellows (VY) is an important disease of sugar beet (Fig. 8.1). It is most severe and persistent in mild maritime climates such as Pacific coastal states of the USA, Western Europe, and Chile. These climates provide a long season for sugar beet for both root and seed crops, give a potentially continuous reservoir of virus–host sources, and favor the overwinter survival of the aphid species that transmit the viruses. VY is caused by the closterovirus *Beet yellows virus* (BYV), and the poleroviruses *Beet western yellows virus* (BWYV), *Beet chlorosis virus* (BChV) (Duffus and Liu 1991; Liu et al. 1999), and *Beet mild yellows virus* (BMYV). The principal aphid vector is the green peach aphid (*Myzus persicae* Sulzer) (Watson 1940) but many other species are known to vector one or more of these viruses. BMYV, BChV, and BYV can decrease sugar yield by at least 30%, 24%, and 49%, respectively (Smith and Hallsworth 1990; Stevens et al. 2004). Breeding for resistance in sugar beet started in Europe in 1948 and in 1957 in the USA (Bennett 1960; de Biaggi 2005; Duffus 1973; Duffus and Ruppel 1993; Hauser et al. 2000; Luterbacher et al. 2004; McFarlane and Bennett 1963; Rietberg and Hijner 1956; Stevens et al. 2004, 2005, 2006).

Fig. 8.1 Vein of beet yellows virus on sugar beet

Likely, the agents that cause VY have coevolved with *Beta* spp. It would seem then that a desirable place to search for high host–plant resistance to one or more of the viruses would be in the primary and secondary germplasms (Luterbacher et al. 2004; Panella and Lewellen 2007). Conventional breeding for resistance to VY has been moderately successful within sugar beet, but most sources of resistance are quantitatively inherited and have low heritabilities. This makes transfer from exotic sources to elite breeding lines and parents of hybrids very difficult. Other than the cultivated beet crops, *Beta maritima* would be the most logical place to find the desired genetic variability. However, little known research has been done within *Beta maritima* for VY resistance.

Grimmer et al. (2008a) reported that resistance to BMYV was identified in wild accessions and successfully transferred to early generation backcrosses with sugar beet. Luterbacher et al. (2004) assessed resistance to BYV in 597 *Beta* accessions collected worldwide and identified highly resistant individual accessions. Resistant individual plants were crossed with sugar beet plants to generate populations for mapping (Francis and Luterbacher 2003). The results from mapping these populations were reported by Grimmer et al. (2008b). Using AFLP and SNP markers, a locus controlling vein-clearing (Fig. 8.2) or mottling symptoms caused by incipient BYV infection was mapped to chromosome IV and given the name *Vc1*. Three BYV resistance QTLs were identified and mapped to chromosomes III, V, and VI. QTLs on chromosomes III and V acted only in plants showing mottled symptoms. Vein-clearing symptoms were controlled only in plants with allele *Vc1* on Chromosome VI. These results and concurrently run ELISA tests for BYV suggest that BYV resistance breeding can be facilitated by employing molecular marker techniques (Grimmer et al. 2008b) but the inheritance of resistance is still rather complex with unknown outcomes in the field.

Breeding for VY resistance at Salinas, CA had been one of the long-term objectives of the sugar beet breeding program starting in 1957 for BYV (McFarlane and Bennett

Fig. 8.2 Virus yellows inoculated trials at Salinas

1963), then changing to BWYV (Lewellen and Skoyen 1984), and then to BChV (Lewellen et al. 1999). Despite preliminary tests with wild beet species that suggested "It seems unlikely that any of the wild species tested will be of value in the program of breeding for resistance to beet yellows" (McFarlane and Bennett 1963), it seemed important to determine if higher, more heritable resistance could be found in *Beta maritima*. Several lines with resistance have been released from this later work, including C927-4 (Lewellen 2004d).

The development and traits of line R22 also called C50 and C51 (Lewellen 2000b) are discussed in Sects. 8.1.3 and 8.1.11.1. Other populations, for example, C26 and C27, containing *Beta maritima* germplasm also were developed (Lewellen 2000b). One of the objectives in breeding R22, C26, and C27 was to find higher resistance to VY from *Beta maritima.* Advanced cycle synthetics of R22 were further backcrossed into sugar beet and reselected for VY resistance (Lewellen 2004c). Spaced plants grown in the field were inoculated with BYV, BWYV, and/or BChV and selected on the basis of individual sugar yield and freedom from yellowing symptoms.

Trials in the UK with BChV were run to show that BChV caused significant losses (Stevens and Hallsworth 2003). At Salinas, compared to susceptible, unselected sugar beet, germplasm lines with *Beta maritima* had reduced losses to BChV (Table 8.2). However, in developing R22 and its backcrosses, moderately VY-resistant/tolerant sugar beet parents were used that showed similar responses to VY. It is unclear if any additional genetic variation for resistance was introduced from the *Beta* maritima sources. These tests did suggest, however, that mass selection for VY resistance based on components of sugar yield lead to higher sugar yield and percentage sugar performance than what might be expected for lines with up to 50% of their germplasm from *Beta maritima*.

Table 8.2 One component of virus yellows is *Beet chlorosis virus* (BChV). Comparison of breeding lines under BChV inoculated and non-inoculated conditions at Salinas, CA, including lines with germplasm from *Beta maritima*

Variety	References	Description	BChV Inoculated		% Loss[2]	Yellows score[3]
			SY[1] (kg/ha)	% Sugar		
Susceptible checks						
SP6322-0	Coe and Hogaboam (1971)	Selected without exposure to VY[4]	9860	14.3	36	6.9
US 75	McFarlane and Price (1952)	Selected from US 22	11,100	13.1	28	5.2
Virus yellows selected starting 1957						
C37	Lewellen et al. (1985)	VY selected from US 75	17,200	16.1	7	2.7
C31/6	Lewellen (PI 590799)	VY selected from US × European VY selections	16,200	15.4	7	2.9
C76-89-5	Lewellen (1998)	Full-sib family from C31/6	17,900	16.3	1	2.0
C69/2	Lewellen (2004a, b, c, d)	VY selected composite of all VY selections	19,000	17.0	6	3.5
Lines with Germplasm from *Beta maritima*						
C67/2	Lewellen (2004a, b, c, d)	10% *Beta maritima* through R22 (C51)	18,000	16.5	6	3.5
C26 × C27	Lewellen (2000b)	50% *Beta maritima* C37 × Atlantic *Beta maritima*	17,000	16.2	2	3.1
$LSD_{(0.05)}$			1700	0.9		0.4

[1]SY is gross sugar yield (root yield × % sugar). Field trial area fumigated with methyl bromide in 2000 to reduce the effects of soilborne diseases and pests

[2]Relative % loss due to BChV calculated from variety means from adjacent companion tests planted on February 27, 2002, BChV inoculated on May 9, 2002, and harvested on October 15, 2002

[3]Virus yellows foliar symptoms scored every 3 weeks during chronic infection from late June to mid-August on a scale of 1–9, where 9 = 100% yellowed canopy. $r = 0.81^{**}$ for % loss × VY scores

[4]VY = BYV, BWYV, and BChV in the USA

8.1.2 Beet Mosaic Virus

Infection by *Beet mosaic virus* (BtMV) is one of the most common diseases of sugar beet and other cultivated beets (Lewellen and Biancardi 2005). In California, it is almost always found in weed and wild beets of various origins growing near the Pacific coast in a perennial manner. The virus is transmitted nonpersistently by aphids including the green peach aphid (*Myzus persicae* Sulzer), often in association with VYs and is easily mechanically transmitted (Dusi and Peters 1999). It is common where cultivated beet is grown as a winter crop or overwintered for seed production (Shepherd et al. 1964). The damage caused by BtMV is small compared to that caused by VYs (Shepherd et al. 1964).

Because damage from most BtMV infections is modest, it has received low priority or no interest from breeders and seed companies. Major gene resistance was not known in sugar beet. However, in a self-fertile (*Sf*), annual (*BB*) line of sugar beet developed by Owen (1942) from Munerati germplasm (Abegg 1936), Lewellen (1973) identified an incompletely dominant gene that conditions resistance. He named this gene *Bm*. In both classical linkage and molecular marker research, this gene was found to be linked to the locus for genetic male sterility (*A1*) on Chromosome 1 (Friesen et al. 2006). The *Bm* allele was also backcrossed into biennial (*bb*) sugar beet backgrounds and evaluated under artificially inoculated conditions in replicated field trials (Lewellen et al. 1982). When all plants were inoculated in the four- to six-leaf stage, *BmBm/Bmbm* plants expressed high resistance, whereas the susceptible *bmbm* recurrent parents showed sugar yield losses that ranged from 8 to 22%. In singly and dually inoculated treatments with components of VYs, the damage caused by BtMV was additive as previously shown by Shepherd et al. (1964). BtMV-resistant breeding lines were released as C32 (PI 590675), C43 (PI 590680), and C719 (PI 590761) (Lewellen et al. 1982).

The *Bm* factor for resistance to BtMV was not found in *Beta maritima* directly, but in a sugar beet annual that likely had a *Beta maritima* source from Munerati's annual (Owen 1942). This suggests that even when not done intentionally, over time useful genes and traits from *Beta maritima* have probably enriched sugar beet germplasm.

8.1.3 Rhizomania

Rhizomania, caused by *Beet necrotic yellow vein virus* (BNYVV), is one of the most destructive diseases of sugar beet (Biancardi et al. 2002; Tamada and Baba 1973). BNYVV is transmitted by the obligate root parasite *Polymyxa betae* Keskin (Fujisawa 1976). Rhizomania was initially found in Italy (Fig. 8.3), then Japan, and it gradually spread over most sugar beet-growing areas worldwide (Biancardi et al. 2002; Brunt and Richards 1989; Scholten and Lange 2000). *Polymyxa betae* is distributed more widely than the BNYVV (Brunt and Richards 1989). Rhizomania is a disease, but its control is well reviewed by Biancardi and Tamada (2016).

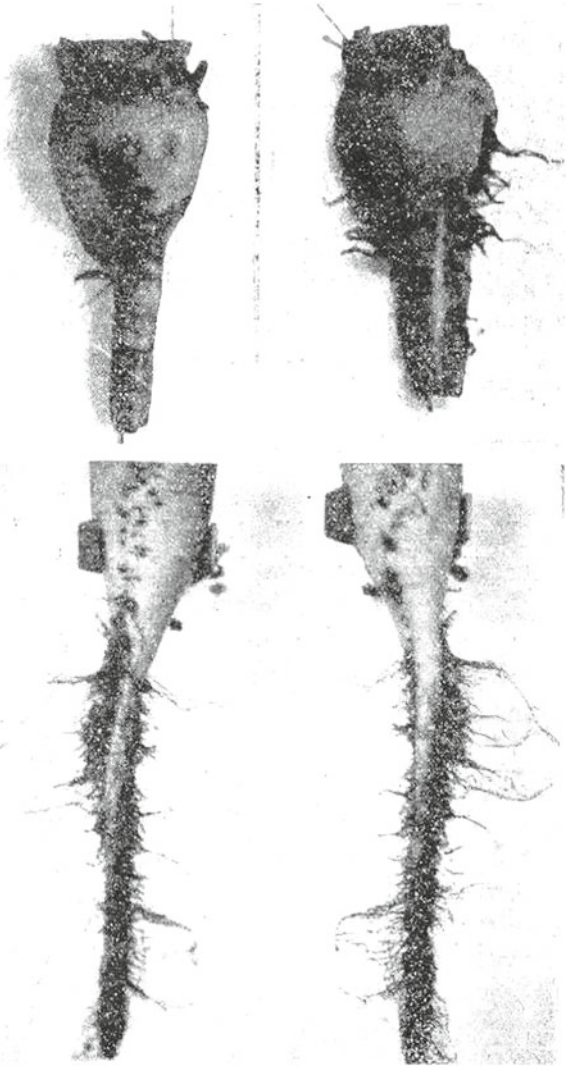


Fig. 8.3 Roots severely diseased by rhizomania (above) and by cyst nematodes (below). (Donà dalle Rose 1951)

The first assessments of commercial varieties in rhizomania-infested fields began in 1958 (Bongiovanni 1964), i.e., before the discovery of the disease's causal agent, attributed to Canova (1966).[1] Results from early field tests (Fig. 8.4), along with data from trials of seed companies from 1966 onward (Gentili and Poggi 1986), showed clearly that Alba P and some other similar multigerm diploid varieties of Italian

[1]Canova used the Italian term "rizomania" for the disease, which had been introduced around 50 years earlier by Munerati (Munerati and Zapparoli 1915). According to Biancardi et al. (2010), this term and not "rhizomania" should be employed for the disease.

Fig. 8.4 Susceptible variety sown between "Alba"-resistant multigerm families (San Pietro in Casale, Italy, 1979)

origin were the most productive varieties in rhizomania-infested soils (Biancardi et al. 2002).

The varieties in question also possessed good Cercospora Leaf Spot (CLS) resistance as a consequence of their parentage from Munerati's genotypes, from which the CLS resistance was obtained (Sect. 8.1.7). It is likely that these old genotypes also provided the genes conditioning the quantitative resistance to rhizomania carried by the variety Alba P (Biancardi et al. 2002; Lewellen and Biancardi 1990). It has been ascertained that the resistance of "Alba type" is governed by genes with additive effects (Biancardi et al. 2002; Frese 2010; Lewellen and Biancardi 1990). In the period from 1980 to 1985, the variety Rizor was bred at the SES-Italy breeding station, carrying a gene for qualitative rhizomania resistance (Fig. 8.5). The variety was much more productive than the varieties with quantitative resistance cultivated at the time (de Biaggi 1987). Additional information regarding the Alba and Rizor resistances is given in step 11, Sect. 1.7.

In 1983, rhizomania was first found in North America in a field located in California on the USDA-ARS station, Salinas, CA by R. T. Lewellen and confirmed

to be BNYVV (Duffus et al. 1984). Individual beets, exhibiting symptoms of both necrotic yellow veins and root bearding, were found in a field where beet cyst nematode (*Heterodera schachtii* Schmidt) trials had been conducted. In order to enrich the nematode inoculum, soil had been incorporated from several commercial sugar beet fields reported to be infested with beet cyst nematode (McFarlane et al. 1982). It may be that the root damage on nematode-resistant genotypes, owing to the *Patellifolia procumbens* resistance, was not due to sensitivity to cyst nematode infection, as reported by McFarlane et al. (1982), but instead was due to BNYVV.

Following the initial reports on rhizomania to the sugar beet industry in 1983, suspicious fields were further reported in several locations. One of these was the variety trial field of Holly Sugar's breeding program at Tracy, CA, where severe damage was observed by Erichsen on all entries except for one series of experimental three-way hybrids. The researchers at Salinas were asked by Erichsen to visit the trial (Fig. 8.6). It was determined that BNYVV rather than cyst nematode likely caused this differential reaction (Biancardi et al. 2002) (Fig. 8.7).

Plants from Holly experimental hybrids were crossed to susceptible sugar beet, and the F1 plants were selfed. In a field test at Salinas under rhizomania conditions, 13-week-old individual S_1 families were either homozygous susceptible or segregated approximately 3 resistant:1 susceptible, thus supporting the hypothesis that resistance was controlled by a single dominant gene (Lewellen et al. 1987) (Fig. 8.8). Individually and collectively, the segregating S_1 families fitted the expected 3:1 (resistant:susceptible) ratio (Fig. 8.9).

Fig. 8.5 Rhizomania diseased field at Phitiviers, France, showing the resistant plot (1983)

Fig. 8.6 Rhizomania diseased field at Tracy, CA (1983)

Fig. 8.7 Susceptible variety USH11. Non-fumigated (left) and fumigated soil

The gene for resistance, unofficially called the "Holly" gene, initially was named *Rz* (subsequently referred to as *Rz1*) (Lewellen 1988). The source of *Rz1* could not be determined by pedigree and breeding records (Erichsen, personal communication, 1987), but it is thought that it likely arose from unknown or unintended outcrosses to *Beta maritima*, as no other similar gene could be found within cultivated beets (Biancardi et al. 2002). This gene provided high-level resistance to BNYVV. The resistance found in the commercial cultivar "Rizor" (developed by SES in Italy) (Biancardi et al. 2002; de Biaggi 1987; de Biaggi et al. 2003) and *Rz1* are the only major resistance genes found in the commercial sugar beet gene pool (Biancardi et al. 2002; Scholten and Lange 2000). The origin of the quantitative resistance to

Fig. 8.8 S1 families under rhizomania at 10 weeks, Salinas CA, 1986

Fig. 8.9 Roots showing segregation within S1 family at 13 weeks, Salinas CA, 1986

rhizomania "type Alba" and qualitative (type "Rizor" and "Holly") is attributable to materials derived from crosses with *Beta maritima* and obtained from Munerati (Biancardi et al. 2002). More recently, using molecular tools, it was confirmed that the resistance found in Rizor and the Holly material did not come from separate genetic sources (Stevanato et al. 2015). This evidence is indicative of the fact that the SES pollinator used most likely originated from the Ro 281 family (from Munerati's work) or a similar germplasm, which had been probably bred in public and private programs and then found its way to Holly Sugar through typical exchanges of germplasm (Panella and Biancardi 2016).

Once rhizomania was recognized in California, an extensive program to find host resistance by screening *Beta* genetic resources (cultivated and wild) was initiated by the USDA-ARS at Salinas. The identified resistance sources were incorporated into elite sugar beet germplasm (Biancardi et al. 2002). The *Rz1* allele proved to be handled easily in breeding programs. Resistance breeding to rhizomania has deployed the *Rz1* gene in elite germplasm worldwide (Amiri et al. 2009; Azorova and Subikova 1996; Barzen et al. 1997; Lewellen et al. 1987; Nouhi et al. 2008; Thomas et al. 1993; Whitney 1989b). However, as single dominant resistance genes often are eventually overcome by mutations in a variable pathogen gene pool, additional sources of resistance were sought by breeding programs worldwide. Since no additional resistant sources were found in the cultivated sugar beet gene pool, various genetic resources, especially *Beta maritima* accessions, were screened for rhizomania resistance (Francis and Luterbacher 2003; Geyl et al. 1995; Panella and Lewellen 2007).

The USDA-ARS germplasm improvement program used two different breeding approaches. The first breeding method focused on major gene resistance. When discovered, genes were backcrossed into elite sugar beet germplasm. Lewellen and coworkers identified several BNYVV-resistant *Beta maritima* accessions (Lewellen 1995a, 1997a), using field resistance and levels of virus titer (by ELISA) as preliminary evaluation assays (Whitney 1989b). A resistant accession from Denmark, WB42, was crossed with sugar beet parental line C37 (Lewellen et al. 1985) and was released as germplasm C48 and C79-3 (Lewellen 1997a; Lewellen and Whitney 1993). This resistance was shown to be different from *Rz1*. In growth chamber tests, it conferred higher resistance than *Rz1* and was designated as *Rz2* (Scholten et al. 1996, 1999). Thus far, there are five sources of resistance conditioned by a single gene from *Beta maritima*, although most sources have been shown to be either *Rz1* or *Rz2* (Biancardi et al. 2002; Panella and Lewellen 2007). *Rz3*, which maps to chromosome III, has been shown to be linked to *Rz1* and *Rz2* (Gidner et al. 2005). The source of *Rz3* is a *Beta maritima* accession, WB41 (Denmark). There is a variable BNYVV-resistant expression in the heterozygote in the genetic background in which it has been evaluated.

Nonetheless, sugar beets with the combination of *Rz1* and *Rz2* or *Rz3* (in the heterozygous state) showed a lower virus titer than *Rz1* alone (Gidner et al. 2005). Using R36 (Lewellen and Whitney 1993), a composite population of many *Beta maritima* accessions, Grimmer et al. (2007) identified a major QTL, named *Rz4*, that appeared to be different from *Rz1*, *Rz2,* or *Rz3* and also located on chromosome III. Using a mapping population, based on C79-11 as the resistance donor, another potential resistance gene, referred to as *Rz5*, was identified (Grimmer et al. 2008c). The resistance in C79-11 (Lewellen and Whitney 1993) was from *Beta maritima* accession, WB258 (step 12, Sect. 1.7). *Rz4* and *Rz5* map close to *Rz1* and each other, thus raising the possibility of belonging to an allelic series.

In the Imperial Valley (IV) of California (near the border with Mexico) in 2003, resistant hybrids, winter beet cultivars carrying the *Rz1* gene, showed rhizomania symptoms in a few fields. Over the next couple of years, laboratory, greenhouse, and field tests at Salinas confirmed that *Rz1* resistance gene had been overcome (Liu et al. 2005; Rush et al. 2006). Since then, resistance-breaking strains have been

found in major growing regions, including Colorado, Idaho, Minnesota, Nebraska, and Oregon (Liu and Lewellen 2007). Only partial resistance to these strains of BNYVV is conferred by *Rz2* and *Rz3* from *Beta maritima*, although combinations of *Rz1* and *Rz2* appear to condition more resistance than either alone. Encouragingly, progeny families of C79-9 (resistance from *Beta maritima* accession WB 151–PI 546397) appeared to have higher levels of resistance to resistance-breaking strains of BNYVV (Lewellen 1997a; Panella and Lewellen 2007).

The emergence of resistance-breaking strains of BNYVV rekindled the interest in the C79 populations with multiple, different sources of rhizomania resistance backcrossed to C37, created by Lewellen at Salinas (Lewellen et al. 1985; Lewellen 1997a, b). The 11 germplasms in the C79 series were from different genetic sources of resistance to BNYVV. They had been backcrossed 1 to 6 times with C37 (Lewellen 1997a, b). The seed from these sources had been poly-crossed in the field at Salinas and, following selection, was designated as R740 and placed in storage (Panella et al. 2018). With the renewed interest in other sources of genetic resistance, this seed was sent to the USDA breeding program at Fort Collins, Colorado. SNP markers, which were linked to *Rz1* and *Rz2* (Stevanato et al. 2012, 2014a; Panella et al. 2015a, b), were used to select individual plants. Two germplasms were released from this project: FC1740 was selected as homozygous resistant to SNP markers linked to both *Rz1* and *Rz2* resistance genes (inferred genotype—*Rz1Rz1Rz2Rz2*), and FC1741 was selected as homozygous to the marker linked to the *Rz2* gene for resistance and homozygous susceptible for *rz1* (inferred genotype—*rz1rz1Rz2Rz2*) (Panella et al. 2018). There is a possibility that other resistance genes may also be present in these germplasms but there were no SNP markers publicly available to ascertain this at the time of their release.

The second breeding method involved individual screening of *Beta maritima* populations and pooling the selected resistant plants—a composite approach (Doney 1993). The pooled plants were increased in mass, and there was no effort to classify the resistance sources as *Rz1*, *Rz2*, etc., or other factors. Several breeding populations were developed using this method and have been released as C26, C27, C51, R21, C67, R23, R23B, and R20 (Lewellen 2000b, 2004b). Although there are most likely major genes in these populations, the existence of additional minor resistance genes may eventually lead to a more durable resistance.

In an attempt to discover novel sources of quantitative multigene resistance, Richardson et al. (2019) conducted a thorough screening of available *Beta maritima* germplasm collection under field and greenhouse conditions using both resistance-breaking and nonresistant-breaking strains of BNYVV. Overall findings from field and greenhouse assays pointed to the superiority of accessions from Denmark in combating BNYVV as well as resistant breaking strains of BNYVV, thus providing evidence for their possible exploitation as pre-breeding donor material in future efforts aiming at the development of rhizomania-resistant varieties.

Recently, the University of Padua, Italy, through a sponsored research project, has collected seeds of 35 populations of *Beta maritima* along the Italian and Croatian coasts of Adriatic Sea. Representative seed samples from each population were planted the year after collection both in the field and glasshouse. Molecular analyses

were performed in order to examine the presence of the *Rz1* source of resistance. Preliminary results showed that the frequency of the *Rz1* allele was significantly higher in sea beet populations collected on the Italian Adriatic coast. This would provide additional genetic proof about the speculated origin of *Rz1* from the Italian sea beet gene pool (Stevanato, personal communication). In a collaborative project between the University of Padua and the USDA Fort Collins program, 24 individuals from 64 populations were screened with markers for *Rz1* and *Rz2*. Many populations contained the *Rz1* SNP marker, while there were areas where the *Rz2* marker was present (unpublished data). A big future challenge is to determine the allelic diversity within these populations and to gain insight into its effect in relation to the level of resistance.

8.1.4 Beet Curly Top Virus

Curly top in beets is caused by a mixture of at least three closely related Curtoviruses in the family *Geminiviridae*: *Beet curly top virus* (BCTV), *Beet mild curly top virus* (BMCTV), and *Beet severe curly top virus* (BSCTV) (Strausbaugh et al. 2008). They are all transmitted by the beet leafhopper, *Circulifer tenellus* Baker (Fig. 8.10), which attacks sugar beet and many other crops cultivated in semi-arid areas (Western USA, Mexico, Turkey, and Iran) (Bennett 1971; Bennett and Tanrisever 1958; Briddon et al. 1998; Duffus and Ruppel 1993; Panella 2005b). Similar viruses occur in Argentina, Uruguay, and Bolivia (Bennett 1971).

Almost as soon as the sugar beet industry was established in the Western United States, BCTV severely impacted yields (Bennett 1971; Carsner 1933; Murphy 1946). Production in California was begun in 1870, and shortly thereafter BCTV symptoms were observed on beets grown there, and by the 1920s, it was clear the sugar beet industry required varieties with resistance to BCTV to survive (Bennett 1971; Bennett and Leach 1971; Carsner 1933; Coons 1953; Murphy 1946) (Fig. 8.11). The early breeding efforts resulted in the release of US 1, a curly top-resistant open-pollinated variety that was a huge step forward (Carsner 1933). At the time of its release, researchers already were looking at *Beta maritima* as a potential source of resistance to BCTV (Coons et al. 1931), which probably is why Coons was commissioned in 1925 to collect *Beta maritima* in Europe (Coons et al. 1955). Further increases in resistance to BCTV were achieved with US 33 and US 34 selected from heavily curly top infested fields of US 1, and eventually they were superseded by US 12 and US 22, which were further improved in US 22/2 and US 22/3 (Coons et al. 1955). However, as stated by Coons et al. (1955): "Hybridizations [of *Beta maritima*] with sugar beets were made and the segregating generations were selected for both leaf spot resistance and curly top resistance. The outlook of obtaining resistant strains in this way was promising but not more so than from the selections made from the sugar beet itself. Since breeding work with the sugar beet did not present the problems of ridding the progenies of multicrowns and rootiness, the emphasis on wild hybrids gradually dwindled."

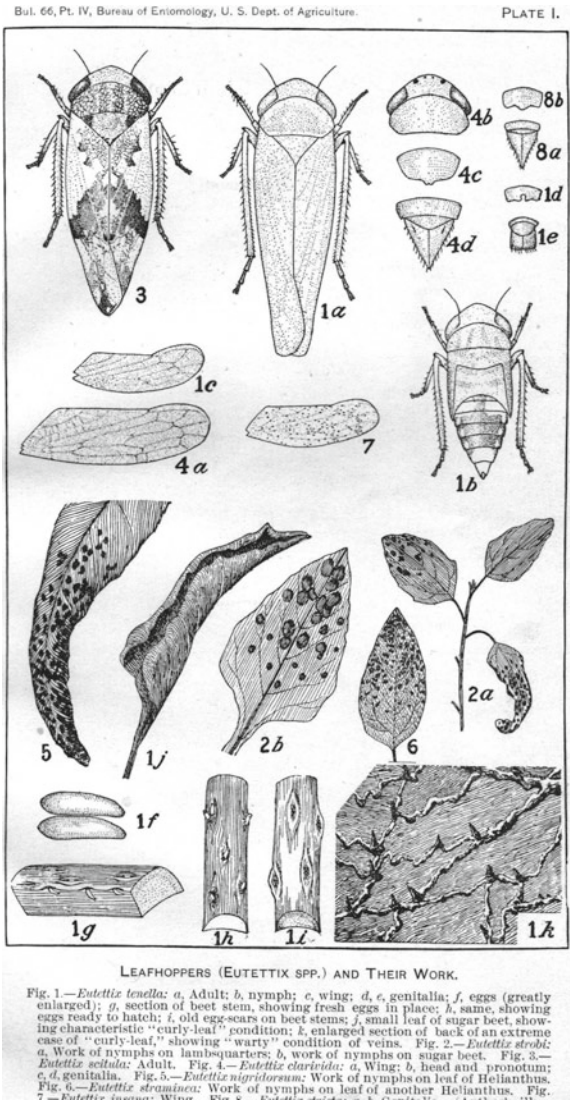


Fig. 8.10 Leafhopper (*Circulifer tenellus*)

Despite what Coons states, Owen speculated that his source of extreme resistance to BCTV, which he called "strain 286", was most likely a chance hybridization with a "wild beet" in California (Owen et al. 1939). We know that wild beets in California encompass introductions of *Beta macrocarpa* and *Beta maritima* from Europe, and may include feral domestic beets (chard, table beet, sugar beet) (Bartsch et al. 1999; Carsner 1928; McFarlane 1975). Owen also declared "However, some accidental hybridization of parental strains of US 1 and progenies comparable in origin with 286 is now suspected." Certainly, the spangled roots of early 286 progeny in the

Fig. 8.11 Beets diseased by BCTV (left)

photograph in the 1946 Proceedings of the ASSBT (Owen et al. 1946) resemble progeny of sugar beet crossed with a sea beet. It is during the development of US 1 that Carsner comments on the wild beets in southern California (Carsner 1928), which lends credence to Owen's remarks. The performance of 286 showed extreme resistance to curly top (Carsner 1926; Owen et al. 1946). CT9 and later, C569, which were widely used in the Western USA as components of curly top-resistant hybrids, were derived from this line (McFarlane et al. 1971; Owen et al. 1946). This example of *Beta maritima* being a largely unrecognized source of resistance and yet being characterized by Coons as difficult to work with when other sources were present in the sugar beet germplasm typifies the attitude of many of the commercial breeders who made little use of sea beet germplasm during the first 60 years of the last century (Lewellen 1992). Most of the beet curly top-resistant material in use today stems from this gene pool, which was widely used by USDA-ARS plant breeders and provided sources of strong resistance to curly top and may have been a source of resistance to other diseases. Nonetheless, there is continued screening of sea beet for resistance to all of the curly top viruses in a cooperative curly top nursery managed by the Beet Sugar Development Foundation and USDA-ARS planted in Kimberly, Idaho (Doney 1998; Hanson and Panella 2002b, 2003b, 2004a; Panella 1998b, 1999a, 2000b; Panella and Hanson 2001b; Panella and Strausbaugh 2011a, b, 2013; Strausbaugh and Panella 2014, 2015, 2016, 2017). In a recent search of the USDA-ARS National Plant Germplasm System's (NPGS) Germplasm Resources Information Network (GRIN) Database, there are two *Beta maritima* accessions that

had better resistance than intermediate (rating of <5; 0 to 9 scale; immune to dead) to beet curly top (PI 518338 and PI 504185) (USDA-ARS 2011a).

8.1.5 Powdery Mildew

Damage from powdery mildew caused by *Erysiphe polygoni* DC (syn. *E. betae* Weltzien) is common almost everywhere sugar beet is grown. Major gene resistance has not been found in sugar beet germplasm; however, quantitatively conditioned tolerance is known and widely used in commercial varieties (Lewellen 1995b; Whitney et al. 1983). In an initial screen of *Beta maritima* accessions at Salinas in field plots in the late 1970s and early 1980s, resistance to powdery mildew was identified in several accessions. In greenhouse tests on seedlings plants, Whitney (1989a) confirmed that high resistance segregated among these accessions.

Two accessions (WB97 and WB242) that showed high resistance were chosen as sources of resistance in a program to determine the inheritance of resistance and transfer this resistance to sugar beet (Lewellen 2000a). WB97 (PI 546394) was in the Salinas collection assembled and evaluated by McFarlane. WB97 was sent to Salinas from the Japan Sugar Beet Improvement Foundation in 1968 and identified as *Beta patula* WB46 from the Wageningen collection. If WB97 (WB46) is *Beta patula*, then it would have been collected from dos Embarcaderos near Madeira (Lange et al. 1999). McFarlane noted that WB97 was variable and did not have typical *Beta patula* characteristics and was more likely *Beta maritima* or crosses between *Beta patula* and *Beta vulgaris/Beta maritima*. Resistance to powdery mildew was transferred from WB97 to sugar beet, and a series of germplasm releases identified as CP01, CP03, CP05, and CP07 were made (Lewellen 2000a, 2004a, b). Resistance is conditioned by one dominant gene (Lewellen and Schrandt 2001) (Figs. 8.12 and 8.13).

WB242 (PI 546413) was obtained for the Salinas collection from Rietberg, Bergen op Zoom, the Netherlands in May 1974. It was reported to have been collected from the Loire River Estuary, France, and to have reduced nematode cyst counts in tests

Fig. 8.12 Segregation for reaction to *Erysiphe polygoni* within plot of CP04 with WB242 source

Fig. 8.13 Adjacent 5-monts-old plants segregating for reaction to powdery mildew

at IRS, Bergen op Zoom. It is probably similar to other accessions obtained from the Netherlands including one called Le Pouliguen Group 2 (PI 198758–59) received from Boss in 1987. Germplasm developed from the introgression of powdery mildew resistance into sugar beet from WB242 has been more extensively studied than that from WB97. Sequential backcrosses and improvements were released as germplasm lines CP02, CP04, CP06, CP08, and CP09CT (Lewellen 2000a, 2004a, b).

Resistance to powdery mildew from WB242 is conditioned by one major gene named *Pm* (Lewellen and Schrandt 2001). Molecular markers to this resistance factor were identified (Janssen et al. 2003; Weiland and Lewellen 1999). WB242 is susceptible to rhizomania and backcrosses to introgress *Pm* into sugar beet utilized recurrent sugar beet lines that had resistance to rhizomania (*Rz1*). During field tests under both rhizomania and powdery mildew conditions, it was observed that derivatives from line CP02 also carried resistance/tolerance to sugar beet cyst nematode. Population CN12 was released as a source for resistance genes for powdery mildew (*Pm*), rhizomania (*Rz1*), and sugar beet cyst nematode in a background with adaptation for the Western USA (Lewellen 2006b). Other releases have included CN12-446, CN12-751, CN12-770, CN12-8-407, CN07-410, CN07-413, and CN18-438 (Lewellen, unpublished). Although resistance to downy mildew caused by *Peronospora farinosa* (Fr.) Fr. f.sp. *betae Byford* (syn. *Peronospora schachtii* Fckl.) has been reported (Dale et al. 1985), we are not aware of any breeding programs using this source for commercial varieties.

8.1.6 Root Rots

Rhizoctonia crown and root rot of sugar beet (caused by *Rhizoctonia solani* Kühn) affects or threatens sugar beet-growing areas worldwide (Ahmadinejad and Okhovat 1976; Büttner et al. 2003; Herr 1996; Ogata et al. 2000; Panella 2005c; Windels et al. 2009). In the USA, where it is registered for use, Quadris™ (an azoxystrobin fungicide) effectively controls this disease; however, the timing of application is critical (Stump et al. 2004). As crop rotations are shortened in the USA, Europe, and worldwide, this disease is becoming an increasing problem. Rhizoctonia root rot is best managed through an integrated program, based on resistant germplasm using good cultural practices and timely fungicide application (Herr 1996).

In the 1950s, Gaskill (USDA-ARS at Fort Collins, Colorado) began a Rhizoctonia crown and root rot resistance breeding program primarily based on the Great Western Sugar Co. (GWS) sugar beet germplasm (Lewellen 1992; Panella 1998a). Schneider and Gaskill (1962) also were looking at introduced germplasm at that time. Although in their report most everything is described as *Beta vulgaris* (Schneider and Gaskill 1962), they comment that much of the material is annual, which suggests that if it is not *Beta maritima*, it had most likely hybridized with it at some point. Some of this *Beta maritima* germplasm made its way into SP5831, released for resistance to Aphanomyces black root (Doney 1995). This source, as well as other sources of *Beta maritima*, was incorporated into some of the early Rhizoctonia-resistant releases. These included FC706 (Hecker and Ruppel 1979), FC708 (Hecker and Ruppel 1981), and FC710 (Hecker and Ruppel 1991; Panella 1998a, 2005c). Although commercial sugar beet breeding companies used and exchanged this germplasm, much of this activity was informal and it is not easy to document the use of *Beta maritima* (Lewellen 1992).

Since the 1980s, efforts to screen *Beta maritima* for new sources of resistance to *R. solani* have increased (Asher et al. 2001b; Burenin 2001; Luterbacher et al. 2000, 2005; Panella and Frese 2003; Panella and Lewellen 2007). Most of the Rhizoctonia-resistant germplasm (commercial and public) can trace its parentage to the USDA-ARS program at Fort Collins, Colorado, started by Gaskill (Panella 2005c). This program continues to screen *Beta maritima* for resistance to *Rhizoctonia solani* and to incorporate resistant accessions into enhanced germplasm for release (Hanson and Panella 2002c, 2003c, 2004b, 2005, 2006, 2007; Panella 1999b, 2000c; Panella et al. 2008, 2010, 2011b, 2012, 2013, 2014, 2015a, 2016; Panella and Hanson 2001c; Panella and Ruppel 1998).

Fusarium yellows is an important soilborne disease found in sugar beet (*Beta vulgaris* L.) production areas throughout sugar beet-growing areas worldwide (Christ and Varrelmann 2010; Panella and Lewellen 2005; Hanson et al. 2018). Many *Fusarium* species have been reported to cause Fusarium yellows (Hanson 2006; Hanson and Hill 2004; Hanson and Lewellen 2007; Ruppel 1991; Windels et al. 2009); however, the primary causal agent in sugar beet is *Fusarium oxysporum* Schlechtend. Fr. f. sp. *betae* (Stewart) Snyd & Hans. (Stewart 1931). The severity of *Fusarium* yellows is influenced by temperature, inoculum dose, and presence of sugar beet

cyst nematode (*Heterodera schachtii* Schm.) (Gao et al. 2008; Hanson et al. 2009a, b; Landa et al. 2001). When conditions favor its occurrence, yield losses can be devastating (Hanson et al. 2009a, b).

Unfortunately, *F. oxysporum* f. sp. *betae* is highly variable in its morphology, pathogenicity, and genetic structure (Harveson and Rush 1997; Hanson et al. 2018; Hill et al. 2011; Ruppel 1991). Other species of *Fusarium* also have been shown to cause yellowing-like symptoms on sugar beet (Burlakoti et al. 2012; Hanson and Hill 2004). Research to date has identified resistant commercial cultivars and a high degree of variability in virulence (Hanson et al. 2009a, b). Management of this disease is heavily dependent on the use of resistant hybrid cultivars (Franc et al. 2002; Hill et al. 2011). In sugar beet, *F. oxysporum*-resistant lines are known, but the genetic system that controls *Fusarium* diseases is still unclear (de Lucchi et al. 2017). Some public breeding has been done, and *Beta maritima* accessions do have resistance (Panella et al. 2015b). Currently, germplasms containing *Beta maritima* germplasm are being screened by the USDA sugar beet breeding program in Fort Collins, Colorado, and field resistance is correlated to molecular markers (unpublished data) linked with resistance to *F. oxysporum* f. sp. *betae* (de Lucchi et al. 2017).

8.1.7 Cercospora Leaf Spot

Cercospora leaf spot (CLS) caused by the fungus *Cercospora beticola* Sacc. is the main fungal disease of beet-growing areas in temperate and humid environments (Fig. 8.14) and affects approximately one-quarter of the cultivated acreage (Holtschulte 2000; Jacobsen and Franc 2009). Pioneering studies on genetic resistance to CLS began in the late 1800s, but only in the early 1900s did the efforts in hybridization and selection made by Munerati achieve the first results. No other source of resistance has been isolated against this disease and incorporated into sugar beet cultivars, except for the "C2 form", which was active only against rarely distributed strains (Lewellen and Whitney 1976). Therefore, the CLS-resistant varieties currently used are derived from crosses with *Beta maritima* obtained by Munerati (de Bock 1986). Mass selections on sea beet began on plants sown in cultivated soil, followed by inbreeding, with the main objective being to fix enough bienniality (Munerati et al. 1913b). Crosses with the sea beet were begun, first using predominantly biennial lines, followed by a number of backcrosses to eliminate the negative traits of the wild parents (fangy and fibrous roots, tendency toward bolting, etc.). Further selections improved bolting resistance and, after 10 years, led to the release of the line RO581, which was considered the first substantially improved CLS-resistant line (Coons et al. 1955). The line was distributed to public and private breeding stations. The American variety US201 is cited as one of the oldest derived lines, together with the Italian Cesena R and Mezzano 71, the Polish Buszczynski CLR, the French Desprez RC2, and the Dutch Vanderhaven AC (Bongiovanni et al. 1958). The increased effort of the breeding companies has produced an improvement in sugar

Fig. 8.14 Drawing of beet moderately diseased by CLS (KWS Cercospora Tafel)

yield and bolting resistance, which had been the main negative traits of the CLS-resistant varieties. With the recent breeding progress, sugar yield is today at similar levels to that of the susceptible varieties (Panella and Lewellen 2007) (Fig. 8.15).

It has been estimated that a severe epidemic in the USA can cause up to a 42% loss of gross sugar (Smith and Ruppel 1973), or up to a 43% relative dollar loss (Shane and Teng 1992). In the USA, initial breeding efforts were based on inbred germplasm developed from Pritchard's (1916) lines and other European lines (Coons 1936) along with germplasm selected by American Crystal in the Arkansas Valley of Colorado (Skuderna 1925). However, as this breeding effort was getting underway, there was another source of Cercospora resistance brought into the USA from Europe (Coons et al. 1955). This material had been seen by Coons in 1925 when it still had many of the undesirable traits from *Beta maritima.* It had been further developed by Italian breeders, and by the time Coons saw it again in 1935, it had been greatly improved (Coons et al. 1955).

The Italian germplasm was incorporated into Great Western Sugar Company varieties GW 304 and GW 359 (source Cesena) and the USDA-ARS researchers also used "Mezzano 71" (Coons et al. 1955; Lewellen 1992). Brewbaker et al. (1950) also referred to breeding lines from some other crosses with European *Beta maritima*, as well as wild beet (most likely *Beta maritima*) out of California. Although it is not known if US 201 (PI 590678) developed from Mezzano 71 was ever used in a commercial hybrid (Lewellen 1992), it found its way into many of the ARS breeding programs (Panella 1998a). It is these early CLS-resistant germplasm pools

Fig. 8.15 Performance of CN12 progenies under severe nematode conditions, Imperial Valley, May 2007. Individual plants from CN12 were selfed and the S1 progeny evaluated under severe nematode conditions in overwintered Imperial Valley. This picture contrasts the differences in reaction to SBCN under these conditions among sets of S3 lines that had been selected for NR (foreground) and nematode susceptibility (background)

that formed the basis of Cercospora resistance breeding in the USA, and much of that resistance came from the *Beta maritima* sources out of Munerati's program and, later, from the curly top germplasm that was added to the Cercospora breeding pools to incorporate resistance to these two important diseases. Further efforts at breeding for resistance in ARS to CLS were focused on combining CLS resistance with other disease resistances, mainly through inbreeding (Panella 1998a). These early breeding efforts have been reviewed in several publications (Coons 1975; Coons et al. 1955; Lewellen 1992; Panella and McGrath 2010; Skaracis and Biancardi 2000). In the last 40 years, because of the renewed interest in using *Beta maritima* as a genetic resource in sugar beet breeding, developing new sources of resistance to CLS has become an important goal. Efforts in the 1980s by the USDA-ARS Sugar Beet Crop Advisory Committee (now Crop Germplasm Committee—CGC) focused on evaluations of sea beet for resistance to CLS as one of the most important goals (Doney 1998). In Europe, innovative methods to introgress genes from sea beet into sugar beet were developed by Bosemark (1969, 1971, 1989), which lead to the efforts of the Genetics and Breeding Work Group of the IIRB to develop "buffer populations" for CLS resistance, as described by Frese et al. (2001) in an example for rhizomania. Efforts in evaluating *Beta maritima* in Europe and the United States were intensified, and some of this germplasm with CLS resistance was discovered (Panella and Frese 2000). In the USA, sea beet germplasm has been screened by the Sugar Beet CGC since 1986 (Hanson et al. 2009a, b, 2010, 2011; Hanson and Panella 2002a, 2003a; Panella 1999c, 2000a; Panella and Hanson 2001a; Panella et al. 1998), and

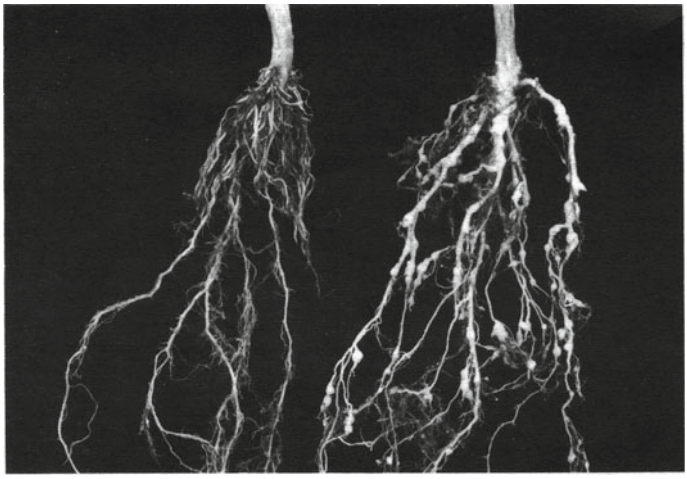


Fig. 8.16 Nematode resistance in commercial hybrids derived from *Beta maritima*. In this picture, two commercial hybrids (SBCN susceptible on left, partially resistant on right) are shown in an Idaho, USA field under SBCN conditions (courtesy Betaseed, Inc). Hybrids with partial resistance to SBCN are now being commercially grown across the northern growing areas of USA. Unlike the *Beta procumbens* resistance, yield drag does not occur in the absence of *Heterodera schachtii*

there are now 123 accessions in GRIN of *Beta maritima* that have been screened for resistance to CLS. Of these, 13 were rated as very resistant (3 < on a scale of 1 = no disease to 9 = dead) (USDA-ARS 2011b) (Figs. 8.16). The GENRES CT95 42 project in Europe evaluated 82 *Beta maritima* accessions, 10 of which were scored very resistant (3<; same scale) (Frese 2004a). Many of these accessions have been incorporated into breeding programs, which are being released to increase the genetic base of the CLS-resistant commercial varieties (Panella and Lewellen 2007; Panella et al. 2015b).

8.1.8 Polymyxa Betae

Polymyxa betae (Fig. 8.17) is the vector of numerous soilborne viruses of sugar beet (Abe and Tamada 1986; Kaufmann et al. 1992; Liu and Lewellen 2008; Wisler et al. 1994), including *Beet necrotic yellow vein virus* (BNYVV), the cause of rhizomania (Tamada and Baba 1973). BNYVV is transmitted by viruliferous zoospores of this plasmodiophorid protozoan. *Polymyxa betae* is an obligate parasite and is found in almost every soil in which sugar beet is grown (Liu and Lewellen 2007). Beet is infected by anterior bi-flagellate zoospores. *Polymyxa betae* forms long-living resting spores clustered together to form cystosori. Viruliferous cystosori can survive many decades in the field. The life cycle, ecology, and infection process have been well documented (Keskin 1964; Tamada and Asher 2016a, b). As a parasite per se,

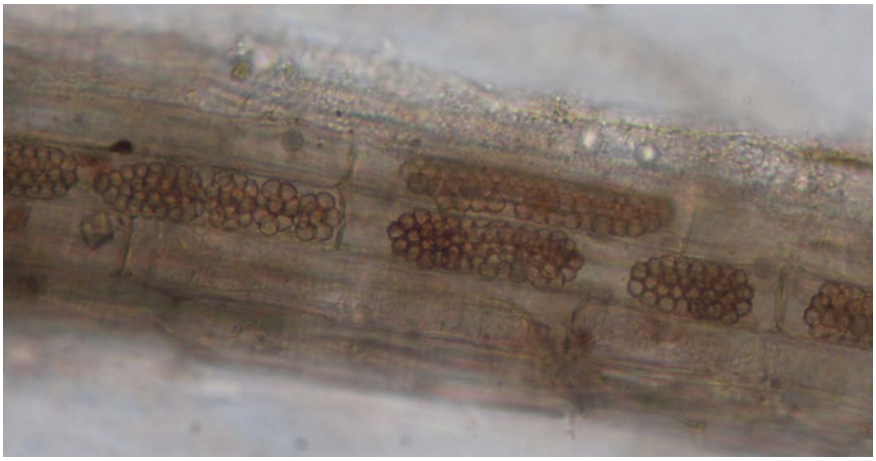


Fig. 8.17 *Polymixa betae* is the vector of BNYVV. Shown here are *Patellifolia betae* cystosori in sugar beet root cells (courtesy John Sears)

Polymyxa betae is usually not considered to cause measurable damage. However, in well-designed and controlled tests, it has been shown to cause reductions in yield (Liu and Lewellen 2008; Wisler et al. 2003).

To quantify the level of *Polymyxa betae* in sugar beet roots, in addition to microscopic techniques, end-point PCR methods were developed (Mutasa et al. 1993, 1995, 1996). However, these methods only indicate *Polymyxa betae's* presence or absence at one specific time. Moreover, the presence of DNA from non-infecting or dead zoospores attached to roots can give misleading results. Kingsnorth et al. (2003) developed protocols for both sequence-independent and hybridization probe real-time PCR for the detection of *Polymyxa betae* glutathione-S-transferase (GST) in infected sugar beet roots. They also demonstrated that real-time PCR analyses of both serially diluted zoospore suspensions and infected root material provided a close relationship between the threshold cycle and the amount of *Polymyxa betae*.

One strategy for breeding more durable resistance to BNYVV is to combine virus resistance genes (e.g., *Rz1*, *Rz2*) (Sect. 8.1.3) with resistance to the vector, *Polymyxa betae* (Asher et al. 2009; Barr et al. 1995; Pavli et al. 2011). A two-gene system (*Pb1*/*Pb2*) conferring resistance against *Polymyxa betae* has been identified and mapped (Asher et al. 2009). The resistance to the vector is simply inherited and acts additively to the *Rz1* resistance against BNYVV, while it also confers protection comparable to *Rz1* in individuals lacking this gene.

In research at Salinas by Liu and Sears, Kingsnorth's methods were modified to screen *Beta* germplasm for possible resistance to *Polymyxa betae* (Liu, personal communication 2010). In a screen of germplasm, 38 materials were tested including accessions of *Patellifolia procumbens*, *Patellifolia webbiana*, and *Patellifolia patellaris*. Four commercial hybrids received from KWS and Betaseed, Inc. ("Roberta" (*rzrz*), "Beta4430R" (*Rz1*), "Angelina" (*Rz1Rz2*), and "BetaG017R" (*Rz2*)), which

have been extensively used in rhizomania research at Salinas (Liu and Lewellen 2007, 2008; Liu et al. 2005), were used as checks. The remaining 31 entries represented a broad germplasm base from the breeding program at Salinas and included rhizomania-resistant and rhizomania-susceptible sugar beet inbreds, populations, and open-pollinated lines. Many of the Salinas entries had germplasm from *Beta maritima* in their background. Based on the GST copy number, where lower values indicated more resistance or lower incidence of *Patellifolia betae*, there was a range from 9 to 881,000 copies. *Patellifolia patellaris*, *Patellifolia procumbens*, and *Patellifolia webbiana* were highly resistant to *Polymyxa betae* with an average of 52 copies. This agrees with previous findings (Paul et al. 1992, 1994). The four commercial hybrids ranged from 48,000 to 881,000 copies with "Angelina" being most susceptible. This result was supported by microscopic examinations, in which "Angelina" had the most cystosori. Except for three entries, the sugar beet lines fit in the same range of susceptibility. The exceptions were monogerm C790-15 (PI 564758) (Lewellen 1994), CP04 (PI 632285) (Lewellen 2004a), and monogerm C812-41 (PI 651522). C790-15 and CP04 were identical to *Patellifolia* accessions for copy number suggesting high resistance. C812-41 had ten times more copies and although partially inbred would likely segregate at most loci. These results need to be confirmed but suggest that high resistance may occur within sugar beet. C790-15 does not have known *Beta maritima* germplasm and is susceptible to rhizomania although in the field at Salinas showed tolerance (Lewellen, unpublished). C790-15 was selected in an S_1 progeny, recurrent selection program that may have favored selection for resistance to *Polymyxa betae*, if genetic variability occurred. CP04 and C812-41 have germplasm from *Beta maritima* and resistance to rhizomania, *Rz1* and *Rz2* or *Rz3*, respectively. WB242 was the *Beta maritima* line used to breed CP04 (Sects. 8.1.5 and 8.1.11.1). C812-41 has WB41 and WB42 *Beta maritima* germplasm through C48 (PI 538251) (Lewellen and Whitney 1993) collected from Denmark and the source of the *Rz2* and *Rz3* resistance to BNYVV (Sect. 8.1.3). It is not known if this putative *Polymyxa betae* resistance came from *sea beet* or not. For C812-41, C790-15-type germplasm was used as the final sugar beet recurrent parent.

8.1.9 Black Root

Aphanomyces root rot or black root and Aphanomyces damping-off are caused by the oomycete, *Aphanomyces cochlioides* Drechs (Buchholtz and Meredith 1944; Drechsler 1929). Black root is a chronic rot of the mature root, which can be a component of a root rotting complex, often including Fusarium yellows and Rhizoctonia crown and root rot (Harveson and Rush 2002). Aphanomyces root rot has been reported in Canada, Chile, Eastern Europe, France, Germany, Hungary, Japan, Russia (and the former Soviet Union), the UK, and the USA (Asher and Hanson 2006; Panella 2005a; Windels and Harveson 2009).

Early *Aphanomyces* resistance breeding programs were centered in the Red River Valley (Minnesota and North Dakota, USA) and with the USDA-ARS stations at

Beltsville, MD and East Lansing, MI. Progress was slow until a greenhouse screening method was developed by Coe and Schneider (Coe and Schneider 1966; Doxtator and Downie 1948; Doxtator and Finkner 1954; Schneider 1954). In the early generations of testing, curly top and leaf spot-resistant material found its way into this program, some of which contained a significant contribution from *Beta maritima* germplasm (this chapter). Schneider and Gaskill (1962) tested a number of foreign accessions (including some *Beta maritima*) for resistance. It is unknown how much of a contribution was made by resistance genes from sea beet.

More recently, evaluations by the USDA-ARS Sugar Beet Crop Germplasm Committee (CGC) and the European GENRES project ("*Evaluation and enhancement of Beta collections for the extensification of agricultural production*"— GENRES-CT95-42) have screened sea beet germplasm for resistance to *Aphanomyces* (Asher et al. 2001a; Doney 1998; Panella and Frese 2003). In the European evaluations of 159 accessions of *Beta maritima*, 5 had high resistance to *Aphanomyces cochlioides* (Luterbacher et al. 2005), and of the 87 screened by the USDA-ARS, 11 had high resistance to this disease (USDA-ARS 2011c). The USDA-ARS breeding program at East Lansing, MI, continues developing *Aphanomyces*-resistant germplasm and studying its inheritance (McGrath 2006; Yu 2004).

8.1.10 Minor Fungal Diseases

High resistance to blackleg disease caused by *Pleospora bjoerlingii* Byford (*Phoma betae* Frank) was observed on fodder beets and on hybrids with *Beta maritima* (Burenin and Timoshenko 1985; Kazantseva 1975). Under severe attack of rust (*Uromyces betae*), Coons (1975) identified some *Beta maritima* population free from infection.

8.1.11 Nematodes

8.1.11.1 Cyst Nematodes

Sugar beet cyst nematode (SBCN) (*Heterodera schachtii* Schm.) is among the most damaging pests known on sugar beet worldwide. Major gene resistance has not been found in sugar beet germplasm (Doney and Whitney 1969). However, high resistance is well known in the Genus *Patellifolia* (formerly Section *Procumbentes* of Genus *Beta*) Ulbrich (Schneider 1937). Resistance from *Patellifolia procumbens* was transferred by Helen Savitsky to sugar beet as a 19-chromosome alien addition line reduced to 18 chromosomes containing a translocated fragment (Savitsky 1975, 1978) (Fig. 1.41). Similar interspecific hybrids have been made and advanced many times since (Jung et al. 1994). This nematode resistance was named *Hs1pro-1* and has been cloned (Cai et al. 1997). The literature on nematode resistance from *Patellifolia*

procumbens has been reviewed (Jung et al. 1994; Panella and Lewellen 2007; Yu 2005). Commercial varieties using *Hs1pro-1* have been developed by commercial seed companies but show a yield penalty under most cultural conditions (Lewellen and Pakish 2005). Resistance to nematode, in which there is no yield drag, remains needed.

Among the *Beta maritima* accessions assembled at Salinas by McFarlane were several that had been reported to be partially resistant to SBCN or have reduced numbers of cysts (although we will refer to this as a "partial resistance" to SBCN, it is often referred to as tolerance rather than resistance) (Heijbroek et al. 1977). Among these was accession WB242 (PI 546413) (Sect. 8.1.5) that had been provided by Rietberg, IRS, Bergen op Zoom, the Netherlands in May 1974 and stated to be an accession collected from Loire River Estuary in France. The accessions with partial resistance were crossed with about 60 other individual sea beet accessions to sugar beet (Lewellen and Whitney 1993). The bulked F_2s were placed in the USDA-ARS NPGS *Beta* collection (NSSL serial no. 206290). The F_2s also were mass selected at Salinas under rhizomania conditions to produce a broadly based sugar beet × sea beet population called R22. R22 was released as C50 (PI 564243) (Lewellen and Whitney 1993). After five cycles of recurrent phenotypic selection, an improved R22 line released as C51 (PI 593694) was produced (Lewellen 2000b). The primary emphasis was selection for resistance to rhizomania and Virus Yellows.

In 1995, an experimental hybrid with R22 was grown in an Imperial Valley of California test under rhizomania conditions in comparison to "Rhizosen" (*Rz1* Holly Hybrids cultivar) and a rhizomania-susceptible commercial cultivar "HH41" that had been grown widely in Imperial Valley (Lewellen and Wrona 1997). As had been observed previously for R22 and R22 hybrids at Salinas, R22 and R22 hybrids seemed to express greater resistance to rhizomania than that conditioned solely by *Rz1*. It was unclear whether this greater resistance was due to improved resistance to rhizomania or resistance to some other pest or disease present in the field. Resistance to beet cyst nematode was suspected by JR Stander and RT Lewellen because most of the rhizomania trial areas also were infested with cyst nematode. Despite its 12.5% *Beta maritima* germplasm, the R22 hybrid had significantly higher sugar yield than Rhizosen (Lewellen and Wrona 1997). A field trial area was established on the Brawley Station, Imperial Valley of California (IV) for evaluation of reaction to rhizomania. Later, it became evident that the cyst nematode population also had increased and had become the predominant disease factor in this trial area (Becker et al. 1996). Since 1995, an expanded area has been successfully used to screen and select *Beta* germplasm resources and breeding lines for resistance to SBCN.

During the later stages of development of C51, R22 was being backcrossed into self-sterile sugar beet breeding lines such as C78 (Lewellen 1997b). In the same 1995 trial with R22, some of these backcross-derived lines also were superior to lines with only *Rz1*, suggesting that the factor from R22 for enhanced performance or disease resistance had been further introgressed into sugar beet and was highly heritable and efficacious. Line C67/2 (PI 628750) (about 6% *Beta maritima*) (Lewellen 2004c) and C72 (PI 599342) (about 3% *Beta maritima*) were as resistant as R22. Based upon subsequent greenhouse tests, it was shown that cyst counts were highly correlated

Table 8.3 Performance of a C927-4 experimental hybrid under non-diseased and severe sugar beet cyst nematode (SBCN) conditions in the Imperial Valley of California in comparison to commercial hybrids

Variety	*Rz1, Rz1*, R22 (*Bvm*)	Severe SBCN		Non-SBCN
		SY[a] (kg/ha)	Appearance[b]	SY (kg/ha)
US H11		3800	3.3	
Beta 4430R	*Rz1*	7800	3.1	15,200
Phoenix	*Rz1*	6300	3.8	14,300
C927-4H5	*Rz1*, R22 (*Bvm*)	11,200	1.8	13,200
$LSD_{(0.05)}$		1900	0.7	1800

[a]SY is refined white sugar yield
[b]Appearance is scored from 1 (healthy) to 5 (dead)

with canopy appearance scores in the IV (higher scores for greater canopy loss); sugar yield was significantly, inversely correlated with canopy scores and cyst counts (Lewellen and Pakish 2005). From these tests, it was determined that the superior performance of R22 and populations extracted from it was due to partial resistance to *Heterodera schachtii* and that this differential canopy response gave a reliable way to identify and discriminate SBCN resistance from susceptibility.

Crosses and backcrosses from R22 to C931 (Lewellen 2006a) to produce a self-fertile Doggett-type population were made to transfer *Beta maritima*-derived rhizomania resistance to sugar beet. Large numbers of individual plants were selfed to produce selfed progeny lines for evaluation. One of the specific lines with enhanced performance was released as C927-4 (PI 640421) (Lewellen 2004d). Subsequent tests in Imperial Valley and at Salinas in the field and greenhouse showed that C927-4 performance has been due in part to resistance to SBCN (Table 8.3).

From C927-4, a series of selfed progeny lines were developed and tested for resistance to SBCN. Based on nematode tests under field and greenhouse conditions, CN927-202 (PI 640420) was selected from C927-4 and released (Lewellen 2007). From other backcrosses to sugar beet populations derived from R22, another selfed progeny line was found that had partial resistance to SBCN. This line was ultimately released as CN926-11-3-22 (PI 640421) (2% *Beta maritima*) after two additional cycles of selfing and reselection for resistance to SBCN (Fig. 1.44) (Lewellen 2007). From two different sugar beet × *Beta maritima* broadly based populations called C26 (PI 610488) and C27 (PI 610489) (Lewellen 2000b), a selfed progeny line from a backcross to C931 was identified that appeared to be resistant to SBCN. This nematode-tolerant line was the only one identified from this material and was released as CN921-306 (PI 640422) (25% *Beta maritima*) (Lewellen 2007).

The specific accession(s) among the Salinas collection of sea beet lines that contributed the resistance gene(s) for cyst nematode resistance to R22 was not known. One of the logical candidates was WB242, which was being used concurrently in the powdery mildew (Sect. 8.1.5) resistance genetics and breeding program (Lewellen 2000a; Lewellen and Schrandt 2001). For the powdery mildew research, WB242

and WB97 (PI 546394) were crossed and backcrossed to sugar beet to set up a Doggett population. When individual plants of this population were examined and selected, it was observed that in addition to segregation for reaction to powdery mildew (*Pm_:pmpm*), some root systems were heavily infested with SBCN cysts, whereas intermingled roots from some adjacent plants were completely free of visible cysts. As mother roots and stecklings were being advanced from sequential backcrosses to sugar beet for resistance to *Erysiphe polygoni* and rhizomania, the root system of each plant was also examined and, where possible, preference was given for seed production to ones without nematode cysts. Within the population that became P912, there appeared to be a low frequency of SBCN-resistant plants. Similar selections originating with WB242 lead to CP04, CP06, CP07, and CP08 (Lewellen 2004a, b). When evaluated under the Imperial Valley conditions, these progressions of backcross lines from WB242 germplasm showed similar performance for resistance to SBCN as R22- and R22-derived material (Lewellen and Pakish 2005). P912 was released as CN12 (Lewellen 2006a, b). From CN12, individual selfed progeny lines were evaluated and selected (Fig. 8.16). Some of these have been released as CN12-446 (PI 657939) and CN12-770 (PI 657940).

In an informal exchange of breeding lines for disease resistance, an accession of *Beta maritima* was received from IRS, the Netherlands in 1987. This accession was reported to be Le Pouliguen Group 2 PI 198758–59. Le Pouliguen Group 2 had been selected for low SBCN cyst counts from *Beta maritima* collected from Le Pouliguen, Brittany, France by Cleij and coworkers at IRS, Bergen op Zoom and SVP, Wageningen (Hijner 1951; Lange and de Bock 1994). These materials were shown to have partial resistance to SBCN but initially thought not to be useful in sugar beet breeding (Heijbroek 1977; Heijbroek et al. 1977). Repeated selection was carried out, and rather high levels of resistance were achieved (Mesken and Lekkerkerker 1988). In 1990, several of the selected stocks were released to the European breeding companies (Lange and de Bock 1994). In tests at Wageningen by Lange and de Bock (1994), it was found that the resistant selections from this *Beta maritima* reduced the number of cysts by about two-thirds. In addition, it was shown that the *Beta maritima* resistance resulted in many of the cysts being much smaller than those on the susceptible control varieties. These smaller cysts contained fewer eggs and reduce the multiplication rate of the nematodes even further. Greenhouse tests at Salinas showed Le Pouliguen Group 2 to have reduced cyst counts as compared to susceptible sugar beet. Although Le Pouliguen Group 2 did not enter the breeding program at Salinas, it was believed to be similar to WB242 and corroborated the value of partial resistance in *Beta maritima*. Eight years later, similar *Beta maritima* material called accession N499 (PI 599349) at Salinas was obtained from KWS seed company. After initial tests in the field at Salinas and Brawley, CA under SBCN conditions, this weedy appearing annual sea beet was backcrossed into sugar beet population C931. An improved population was released as CN72 (PI 636339) (Lewellen 2006b). From CN72, individual selfed progeny families were evaluated at Salinas and Brawley and one line was released as CN72-652 (PI 657938). The SBCN partial resistance from this *Beta maritima* source from Le Pouliguen, France progressed to commercial

Fig. 8.18 Field trials in Imperial Valley of California are used to select and evaluate reactions to cyst nematode

usage in hybrids developed by KWS and Betaseed, Inc. to ameliorate the damage caused by *Heterodera schachtii* (Fig. 8.18).

The genetic relationship for resistance to SBCN from *Beta maritima* among R22 populations, WB242, CN12, Le Pouliguen Group 2, and N499 (CN72) is now known (Stevanato et al. 2014b). Because most of these lines and sources have been derived from the Loire River Estuary in France, all seem have the same gene for SBCN resistance. Nonetheless, WB242 has high resistance to *Erysiphe polygoni* (*Pm*) and has a compact, dark green canopy with slow bolting tendency that distinguishes it from the SBCN resistance from the other sources, particularly N499. In Imperial Valley tests, it appears that partial resistance to *Empoasca* sp. also may occur in WB242-derived material. The SBCN resistance derived from *Beta maritima* is not immunity, but conditions lowered reproduction of cyst nematode (Lange and de Bock 1994; Lewellen and Pakish 2005) and greatly reduces the losses caused by *Heterodera schachtii* under field conditions (Lewellen and Pakish 2005). Similar resistance from *Beta maritima* has been advanced by the commercial seed companies into commercial hybrids and shows equally favorable resistance without sugar yield drag associated with the *Beta procumbens* source under commercial sugar beet production.

Many technologies have been developed to very quickly genotype large numbers of SNPs in DNA samples (Stevanato et al. 2014a). SNP markers linked to the nematode tolerance were developed using the WB242 source. A segregating F_2 population, developed from WB242 as pollinator was crossed to a male sterile line was used for bulked segregant analysis to develop an SNP marker linked to the gene for

sugar beet nematode tolerance, named HsBvm-1 (Pegadaraju et al. 2013; Stevanato et al. 2014b). This marker was able to select among a set of 13 tolerant (heterozygous for the marker) and 13 susceptible commercial (homozygous susceptible) as well as the homozygous-resistant F_2 plants (Stevanato et al. 2014b). These results have been confirmed in another segregating F_2 population with WB242 as the resistance donor parent (unpublished data).

8.1.11.2 Root-Knot Nematodes

Damage from root-knot nematode (RKN) caused by numerous species of *Meloidogyne* is common where sugar beet is grown in a subtropical or warm temperate climate. Resistance to RKN could not be found in cultivated *Beta vulgaris* in a screen of 190 accessions (Yu 1995) (Fig. 1.45). In an initial search of 113 *Beta maritima* accessions, resistance was identified in WB66 (PI 546387). The original source of WB66 is unknown but likely was found within a collection from Wageningen (WB37) in 1963 by way of the Japan Sugar Beet Improvement Foundation in 1968. Resistance from WB66 has been transferred to sugar beet (Yu 1996, 2001; Yu et al. 1999, 2001; Yu and Lewellen 2004). An isozyme marker was identified for RKN resistance (Yu et al. 2001).

Beet germplasm with resistance initially was released and registered as germplasm line M66 (Yu 1996). A molecular marker was identified, and the inheritance of resistance was shown to be conditioned by a single dominant gene named *R6m-1* (Weiland and Yu 2003). Subsequently, resistant beet germplasm from backcrosses to sugar beet was released as M6-1 (Yu 2001). An additional release was made following the fifth backcross to sugar beet after homozygous-resistant plants were selected (Yu and Lewellen 2004). The *R6m-1* gene in lines M66, M6-1, and M6-2 has been shown to condition resistance to at least six species of *Meloidogyne* (Yu et al. 1999; Yu and Roberts 2002).

Resistance to RKN was also discovered in WB258 (PI 546426) (Yu 1997, 2002a, b). WB258 was collected by de Biaggi and Biancardi in the Po Delta in 1979 and sent to McFarlane at Salinas (step 12, Sect. 1.7). WB258 was also shown to have resistance to rhizomania (Lewellen 1995a, 1997a; Whitney 1989c) (Sect. 8.1.3). Root-knot nematode resistance from WB258 is near immunity and conditions resistance to all *Meloidogyne* species tested (Yu et al. 1999). Resistance from WB258 and WB66 may or may not be the same, whereas resistance from WB66 is marked by an isozyme (Yu et al. 2001), which from WB258 is not (Yu 2002b). This difference suggests that WB66 and WB258 were collected from different locations and populations. Resistance to root-knot nematode may be essential in the development of sugar beet for subtropical areas, where *Meloidogyne* spp. cause severe losses.

8.1.12 Insects

In *Beta maritima*, some degree of resistance has been found to bean aphid (*Aphis fabae*) colonization (Dale et al. 1985) and to the multiplication rate of green peach aphid (*Myzus persicae*) (Lehmann et al. 1983). Lowe and Russell (1969) ascertained that the resistance to aphids is inherited in pattern suggesting a trait under polygenic control. These findings have not led to any practical application.

8.1.13 Multiple Resistances

The diseases of beet crops may appear alone or, more frequently, associated with one another. In this case, genotypes endowed with multiple resistances would be useful (McFarlane 1971), and, indeed many hybrids are multiple disease resistances. Many recent public germplasm releases, multigerm, monogerm, and O-type lines, have multiple disease resistances (e.g., Lewellen 2006b; Panella and Lewellen 2005; Panella et al. 2011a, 2015). These materials were crossed with genotypes bearing the monogenic resistances to rhizomania taken from *Beta maritima*. Luterbacher et al. (2005, 2004) published the results of a large survey including cultivated and wild germplasm belonging to the genus *Beta*. Between 580 and 700 accessions were evaluated in several European countries in the presence of three foliar diseases (VYs, powdery mildew, Cercospora leaf spot). The assessment of resistances was performed both in field and glasshouse conditions. In taxa within section *Beta*, there were some cases of multiple resistances identified in *Beta maritima*. The rate of entries displaying more than one resistance was higher in the genus *Patellifolia* and section *Corollinae.* Regarding the soilborne diseases caused by *Aphanomyces cochlioides*, *Pytium ultimum*, *Rhizoctonia solani*, and BNYVV, *Beta maritima* showed the highest number of accessions endowed with multiple resistances. By this term, Scholten et al. (1999) also mean the combination in the same genotype of different types of resistance to the single disease. The combination of diverse resistances increases the plant's ability to combat the effects of the disease with complementary reaction mechanisms (Lewellen and Biancardi 1990). This synergy is currently employed for contrasting the yield reduction in severe rhizomania diseased fields (Sect. 8.1.3).

8.2 Resistances to Abiotic Stresses

Surveys conducted on commercial varieties of sugar beet have shown the existence of a reduced genetic variability for tolerance to water stress. The physiological basis of salt resistance in *Beta maritima* has been explored by Koyro (2000) and Bor et al. (2003). The habitat of *Beta maritima* requires resistance to abiotic stresses caused by both salinity and drought (Shaw et al. 2002). These traits are ones that

have been sought in sugar beet for many years (reviewed by van Geyt et al. 1990), especially in climates where sugar beet cultivation is rain-fed. The effect of climatic and precipitation patterns on rain-fed sugar beet production areas in Europe has been studied (Pidgeon et al. 2001), and there is concern on the effect that global climate change will have on continued production (Jones et al. 2003; Pidgeon et al. 2004).

8.2.1 Drought and Heat Tolerance

Drought tolerance has long been of interest to sugar beet breeders (van Geyt et al. 1990) and is one of the often-mentioned rationales for conserving and using *Beta maritima* as a genetic resource of sugar beet (Doney and Whitney 1990; Frese 2003, 2004b; Stevanato et al. 2004). Because of the variability of rainfall in the UK, researchers there have long been interested in drought tolerance in sugar beet and *Beta maritima* germplasm, and in developing assays to determine drought tolerance (Thomas et al. 1993). The GENRES CT95 42 project in Europe evaluated 155. *Beta maritima* accessions (Frese 2004a). In this test, a standard was used, the cultivar "Saxon", and data from all accessions that were significantly different in weight than Saxon were normalized to Saxon and the deviation from the mean for individual accessions was divided into a 1 to 9 scale with 1 as the most tolerant (Frese 2004a). Five of the seven most drought-tolerant accessions (scored 1) were *Beta maritima* as were three with a drought stress score of 2 (Fig. 8.19). The drought screening was done at Broom's Barn Research Station in the UK and much of the subsequent investigations and reporting out of these results have been done by scientists located there (Ober et al. 2004a, b, 2005; Ober and Rajabi 2010; Ober and Luterbacher 2002).

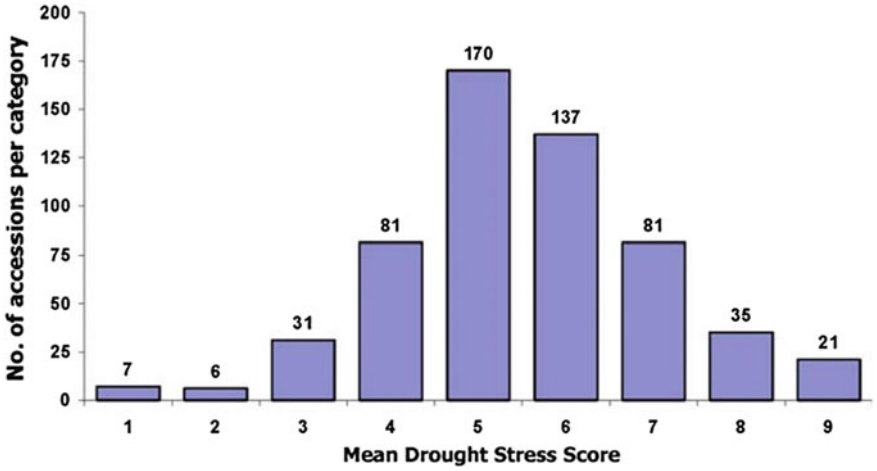


Fig. 8.19 Drought stress tolerance frequency distribution (Frese 2004a)

Some researchers working with *Beta maritima* are approaching the issue from examining the life history traits of the sea beet and how these traits, including resistance to drought, have evolved over time as important survival traits (Hautekèete et al. 2002, 2009; Wagmann et al. 2010). Although many of the countries, which grow winter beet in the Mediterranean and other heat and drought-stressed areas, are very interested in drought tolerance, only a few are working actively with sea beet (Srivastava et al. 2000).

8.2.2 Salinity Tolerance

The resistance of *Beta maritima* to salt stress is well known and in the early 1980s this trait was used as an indicator of *Beta maritima* gene flow into ruderal beet populations (Evans and Weir 1981). Research has examined betaine accumulation and its relation to salinity comparing sugar beet with *Beta maritima* (Hanson and Wyse 1982). More recent work has compared the effect of salinity on lipid peroxidation and antioxidants in the leaves of sea beet and sugar beet (Bor et al. 2003; Koyro 2000) and evaluated the osmotic adjustment response between the two taxa to try and understand the response to salinity (Bagatta et al. 2008; Koyro and Huchzermeyer 1999).

There is an increasing interest in halophytic crops because the world's supply of freshwater is shrinking and world population growing (Baydara 2008). If more saline water can be used to produce food, it will make available more freshwater for human consumption. There is an interest in using *Beta maritima* as a model system, a potential donor of salt tolerance genes, and even as a potential halophytic cash crop (Koyro et al. 2006; Koyro and Lieth 2008). Sugar beet is not the only crop that could benefit from the salt resistance in the sea beet genome; there is also interest in developing more salt-tolerant fodder beet cultivars (Niazi et al. 2000, 2005; Rozema et al. 1990). This response to saline soils is especially important to areas in the Mid-Eastern and North African areas where both heat and salinity of irrigation water are a problem. Recent work in Egypt looked at gene expression in relation to salt stress (El-Zohairy et al. 2009). Although sugar beet is well adapted to saline areas when compared to other crop plants, at germination it is equally sensitive to saline conditions. Research has looked at gene expression and phenotypic differences in sugar beet and sea beet during this critical time of crop establishment (McGrath et al. 2008; Panella and Lewellen 2007).

8.3 Other Traits

According to Krasochkin (1959) and many other authors, *Beta maritima* collected in the northern sites should be an important resource for increasing the sugar content in sugar beet. Campbell (1989) selected 30 sea beets with very high sugar content in good correlation with the root weight. Dale et al. (1985) ascertained that in sea beet

accessions there were plants developing male sterile flowers. These plants produced seed if individually crossed with normal pollen producers of the same accession, thus suggesting the presence of the O-type trait or CMS in *Beta maritima* populations (Sect. 3.10). What is important to remember is that we can never with certainty predict what traits will be of importance in the future. Populations of sea beet existing in situ, undergoing continual coevolution with pests, disease, and the environment, are our insurance policy that we will have the genetic resources to fill future needs (this chapter).

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