# THE DISCOVERY OF RHIZOMANIA RESISTANCE TRAITS IN SUGAR BEET

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## ABSTRACT

Previously recognised as soil sickness or confused with other sugar beet diseases, the symptoms of rhizomania were known in several European countries well before the Second World War. Its rapid spreading was noticed in Italy after 1946, and few years later sporadic symptoms of the disease were observed in areas of intense sugar beet cultivation. Later became evident that the best entries carried the quantitative resistance named "Alba type". Around 1965, the pathologists involved in such researches could establish that the rhizomania was caused by an atypical fungus-virus symbiosis. With this discovery, the disease was correctly explained, and the word rhizomania became used over many important sugar beet production countries. In the 1970's, both the rapid diffusion of the disease and the worsening of the damages on sugar yield pushed many research institutes and seed companies to find more efficient control measures. After years of searching, two monogenetic traits now known as "Rizor type" and "Holly type" were identified and commercially exploited in Italy (1983) and in U.S.A. (1986), respectively. The background of the discovery of the different rhizomania resistances is given by the breeders involved.

## ABRÉGÉ - LA DÉCOUVERTE DES CARACTÈRES DE RÉSISTANCE À LA RHIZO-MANIE CHEZ LA BETTERAVE À SUCRE

Tout d'abord considérés comme un problème de sol ou bien confondus avec d'autres maladies, les symptômes de la rhizomanie, furent reconnus dans plusieurs pays d'Europe bien avant la seconde guerre mondiale. Son développement rapide fut remarqué en Italie à partir de 1946, et quelques années plus tard on observait sporadiquement les symptômes de la maladie dans des régions de cultures intensives. Plus tard il devint évident que ces meilleures variétés portaient la résistance quantitative appelée "Alba type". Vers 1965, les pathologistes impliqués dans ces recherches purent établir que la rhizomanie était causée par une symbiose atypique virus-champignon. Avec cette découverte, la maladie fut correctement expliquée et le mot rhizomanie

s'imposa dans la plupart des grands pays betteraviers. Dans les années 1970, la dissémination rapide de la maladie et l'aggravation des dommages sur les rendements poussèrent beaucoup d'instituts de recherches et de sociétés de semences à trouver des mesures de contrôle plus efficaces de la maladie. Après des années de recherches, deux caractères monogéniques, désormais connus sous les noms de "type Rizor" et "type Holly", furent identifiés et respectivement exploités commercialement en Italie (1983) et au U.S.A. (1986). La découverte de ces résistances sont décrites par les sélectionneurs impliqués.

## KURFASSUNG - DIE ENTDECKUNG DER RIZOMANIA-RESISTENZ BEI ZUCKER-RÜBEN

Die Symptome der Rizomania, wenn gleich anfänglich als Bodenkrankheit bezeichnet oder mit anderen Zuckerrübenkrankheiten verwechselt, waren in verschiedenen europäischen Ländern bereits lange vor dem zweiten Weltkrieg bekannt. Ihre rasche Ausbreitung wurde in Italien nach 1946 beobachtet und bereits wenige Jahre später waren Symptome auf intensive Zuckerrüben-anbaus zu sehen. Später wurde ersichtlich, dass die besten Sorten eine quantitative Resistenz, genannt "Alba-Typ", enthielten. Um 1965 konnten Pathologen, feststellen dass die Rhizomania durch eine ungewöhnliche Pilz-Virus-Symbiose hervorgerufen wurde. Durch diese Entdeckung, die die Krankheit erstmals richtig erklärte, wurde der Begriff Rhizomania in vielen bedeutenden Rübenanbaugebieten geläufig. Die rasche Verbreitung der Kranheit und der stärker werdende Schaden im Zuckerertrag in den frühen Siebzigern bewegte Forschungsinstitute und Saatgut-unternehmen nach wirkungsvolleren Kontrollmaßnahmen zu suchen. Nach jahrelanger Suche wurden zwei monogene Resistenzen – bezeichnet als "Rizor-Typ" und "Holly-Typ" - identifiziert und kommerziell in Italien (1983) beziehungsweise den USA (1986) genutzt. Der Hintergrund der Entdeckung dieser Resistenzen wird durch die beteiligten Züchter beschrieben.

## INTRODUCTION

Rhizomania is considered one of the most serious diseases affecting sugar beet (*Beta vulgaris* subsp. *vulgaris* Sugar Beet Group). The damaged plants display the following symptoms: i) excessive rootlet proliferation; ii) altered root shape; iii) vascular root tissues with necrotic areas; iv) widespread yellowing on the leaves and necrosis of the veins; v) reduced root and leaf growth (Scholten & Lange, 2000). Typical chlorotic spots or vein yellowing may also appear on the leaves (Tamada, 1975). The disease modifies the metabolism of the plant causing reductions in sugar yield due in part to the increased concentration of non-sugars in the roots. In the most severe cases sugar yield can drop to as low as 10% of normal levels (Biancardi, unpublished). After the first systematic tests performed in Italy (Piolanti *et al.*, 1957) and the identification of the true pathogenic agents (Canova, 1966), the new disease was discovered in Japan (1965), Yugoslavia (1971), Greece (1972), France (1973), Germany (1974), Czechoslovakia (1978), China (1978), Austria (1979), Rumania (1979), USSR (1979), Hungary (1982), USA (1983) and subsequently in most countries where

the plant is cultivated (Richard-Molard, 1985; Asher, 1993). The heritability, the physiology and the molecular traits of the main sources of resistance were recently reviewed by Biancardi *et al.*, 2002.

The survival of sugar beet cultivation in the more diseased countries is possible only with the employment of resistant varieties. Therefore, the discovery of the different types of rhizomania resistance used in the practice is considered among the most significant successes of plants selection. The breeders directly involved in the early identification of such traits edited this paper.

Symptoms of atypical rootlets proliferation, low sugar content, and poor leaf and root growth, especially after rainy periods, had long been observed in Italy as in many European countries (Asher, 1993; Biancardi *et al.*, 2002). The spread of these symptoms, frequently confused with those caused by cyst nematodes (*Heterodera schachtii* Schm.), was first observed around 1946 in the north-eastern part of the Po valley (Anon., 1953; Piolanti *et al.*, 1957; Bongiovanni, 1960). Some years later, similar anomalies were observed in many areas across north-eastern Italy (Piolanti *et al.*, 1957). Bongiovanni & Lanzoni, 1964, reported the presence of infected fields in other regions of northern and central Italy.

Research institutes took an interest in the spread of the unknown syndrome beginning the investigations into its causes or the pathogenic agents. In 1957, Donà dalle Rose, Munerati's successor at the Stazione Sperimentale di Bieticoltura in Rovigo, described as follows the symptoms in beet fields around Venice: " . . shortened and globular roots with abnormal shape and abundant rootlet proliferation especially in correspondence with root grooves; browning of the vascular rings.". The author used the term "*rizomania*" to describe the combination of these root symptoms in sugar beet (Donà dalle Rose, 1957).

In the following year, tests carried out in various parts of Italy enabled Piolanti *et al.*, 1957, to advise farmers of the earliest preventative measures, which, even today, are still substantially valid (Biancardi *et al.*, 2002). In the year 1963, Bongiovanni demonstrated empirically the biotic origin of the pathology by infecting a healthy plot using soil taken from a field displaying the aforementioned symptoms (Bongiovanni & Lanzoni, 1964). Ghillini *et al.*, 1965, confirmed this hypothesis when they identified the fungus even involved in the infection processes, classified as *Polymyxa betae* by Keskin *et al.*, 1962, and Keskin, 1964.

The true causes of the disease were explained with the isolation of the virus, which is transmitted and inoculated by *Polymyxa betae* and responsible for the appearance of the symptoms (Canova, 1966). The virus was later classified and denominated beet necrotic yellow vein virus (BNYVV) by Tamada & Baba, 1973. Thanks to these discoveries and to progresses in the ELISA technique for isolating and identifying the virus (Clark & Adams, 1977), the disease was recognized in many cultivation areas with temperate climate (Asher, 1993). The fungus has spread everywhere, and no definitive conclusion has been drawn as to whether on its own (i.e. without the presence of BNYVV) it is able to cause pathological manifestations (Winner & Schäufele, 1977; Brunt & Richard, 1989).

# IDENTIFICATION OF THE FIRST GENETIC RESISTANCE

The earliest observers of the syndrome's spread across Italy did not overlook the fact that some varieties of Italian origin appeared less damaged (Piolanti *et al.*, 1957). In 1958, three field trials were set up by Bongiovani & Lanzoni, 1964, to check these observations in Vighizzolo (Padua), Cona and Cavarzere (Venice). The fields had been heavily infected the previous year and consequently the sugar yields were very low (table 1). As a result of the severe damages caused to commercial fields, farmers were advised not to grow sugar beet in fields, which had previously shown symptoms of the disease (Bongiovanni, 1960, 1965). Nevertheless, the trials confirmed that commercial varieties reacted differently from one another and this implied that there was possibility for selection and genetic improvement. This was the only course of action left open following the results of ineffectiveness of the usual agronomic and chemical systems to limit the damages of the unknown disease (Bongiovanni, 1965).

As far as we know, the earliest planned selections began in 1966 at the Maribo seed company (Gentili & Poggi, 1986) on an infected field near Verona. The genotypes used were prevalently of Italian origin (Johansson, 1985) and one of most employed materials of that time was the diploid multigerm variety Alba P. Breeders at the Alba seed company, involved in the selection of the variety, carried out individual selections on mother beets grown in soil that was later found to be infected by rhizomania (Usai M., personal communication). It is likely therefore that for many years selection for resistance to the disease was carried out unconsciously. Other European seed companies presumably used these genotypes initially.

Breeding selection work at the Rovigo section of the Istituto Sperimentale per le Colture Industriali-ISCI (formerly Stazione Sperimentale di Bieticoltura) began in 1977 with the sowing of 22 diploid families in an infected field near San Pietro in Casale (Bologna). Individual selection was carried out on the roots that survived the winter, since they were less sensitive to frost than those damaged by the disease (Biancardi & Biondani, 1991). This system was found to be effective but relatively risky. In fact, intense frosts could cause the loss of even the more resistant beets. Refractometric degree was also used as selection parameter to complement the visual evaluation of individual beets.

Winter conservation and reproduction of mother beets selected in September encountered difficulties as well due to low sugar content and the development of rots inside the root (Biancardi & Biondani, 1991). It has only been possible on rare occasions to carry out brei sampling to determine the content of sugar and non-sugars. In fact, the procedure can cause severe damage to roots and reduce their survival chances.

The following year the family RO236 was identified which produced a satisfactory amount of sugar in infected soil. The family derived from Munerati's selections and showed several similarities to Alba P, as good cercospora leaf spot (CLS) resistance, sensitivity to bolting, low root yield. Segregation analysis carried out on the two genotypes provided similar results and demonstrated the quantitative character of this rhizomania resistance, referred to from now as "Alba-type" (Lewellen & Biancardi, 1990; Biancardi, 1999).

Mass selections were performed on male sterile lines (CMS) and on the relative maintainers (O-Type). The CMS line MS2-R, obtained in 1983 after two cycles of individual selection in soil with moderate rhizomania infection, was crossed with the RO236 pollinator, labelled RO401 in 1981. Given the susceptibility of the monogerm lines, medium levels of infection were required for this selection, or alternatively, early harvesting in June or July so as to prevent the excessive weakening of the selected beets (Biancardi, unpublished).

In 1985, the hybrid RO401 x MS2-R was tested together with other experimental and commercial genotypes in healthy soil in Rovigo and in severe rhizomania conditions in San Pietro in Casale. The results were satisfactory (table 2) and demonstrated the quantitative and additive character of this type of resistance (Biancardi *et al.*, 1988; Lewellen & Biancardi, 1990).

Beginning to 1985 a selection method was used based on mechanically inoculated leaves of seedlings in controlled conditions (Grassi *et al.*, 1989). The appearance and the number of chlorotic spots on the leaves, appearing about 15 days after inoculation, was considered an index of rhizomania susceptibility. The system provided good results when integrated with field evaluations of the yield traits of the same materials (Biancardi & Biondani, 1991).

There is evidence that Alba-type resistance was derived from materials selected by Munerati in Rovigo: i) the Alba company, founded in 1933, initially worked only with materials of Rovigo origin; ii) characteristics and models of segregation which are similar to Alba P have been identified in the germplasm of Rovigo; iii) the Alba P displayed a high level of cercospora CLS resistance; as Skaracis & Biancardi, 2000, stated, this resistance derives from crosses made by Munerati between sugar beet and sea beet (*Beta vulgaris* L. subsp. *maritima* L. Arcang.). The seed of the wild plants used for the first hybridisation was collected by Munerati himself in the summer of 1909, near Porto Levante in the delta of the river Po, Italy (Munerati, 1946; Biancardi, 1999).

More than 80 years later, Lewellen, 1995a, found some rhizomania resistance traits in the C79-10 and C79-11 accessions of sea beet: the first one was collected by Coons in 1971 (Coons, 1975), the second was provided by Biancardi and De Biaggi in the year 1979 (Lewellen, 1995a). The seeds of both sea beet accessions were harvested in the same seashore around Porto Levante.

## THE MONOGENIC RESISTANCE

The rapid spread of CLS and rhizomania throughout many parts of Italy prompted the Società Europea del Seme (SES) based in Massalombarda (Ravenna) to begin, in the spring of 1980, a program aimed at improving the genetic resistance to both diseases. The SES was an Italian-Belgian company founded in 1971, when it incorporated the old breeding station Mezzano.

Screening of CLS resistant genotypes began in fields infected by rhizomania located in San Martino in Argine (Bologna) and Villa Serraglio (Ravenna). The decision to use this type of material and the testing procedure was made in collaboration with De Biaggi, who was at that time breeder at the ISCI-Rovigo before being hired by the SES.

All genotypes carrying CLS resistance owned by the SES were originated from old families selected by Munerati or from derivations of these genotypes. The latter mainly came from public and private American breeding stations through the usual germplasm exchanges.

In 1980, only the mother beets sown in San Martino in Argine were selected as that field was severely and evenly infected. Among the genotypes that were sown, only multigerm 2n families 2100, 2120, 2130, 2242, and 2281 revealed interesting levels of resistance. The remaining entries were almost completely destroyed by the disease. About 20 mother beets per family were selected the following February. This system was already in use at the ISCI-Rovigo. The beets of each family were employed as pollinators for 2 CMS-susceptible lines (MS80NF and MS80DP) in 5 crossing plots. Line MS80NF was endowed with high combining ability. In August 1981, the seed of the 10 mongerm 2n hybrids was harvested.

In 1982, an agreement with the Institut Tecnique de la Betterave (ITB) in Paris allowed similar tests in infected fields around Pithiviers (Loiret). The visual evaluations of the symptoms and the yield analysis were very satisfactory for the hybrid 2281R1 x MS80NF, which produced about two times more than the multigerm variety Alba P, considered at that time the best resistant check. The production traits of the remaining 4 hybrids were similar to the resistant checks.

The pedigree records of the 5 families used for selection in 1980 were not detailed. Family 2281 was believed to have been derived from Munerati's genetic pool because of its CLS resistance. There was also the suggestion that it may have originated from the family US313, which was supposed of American origin and resistant to CLS (therefore of Italian derivation) and had subsequently readapted to Italian conditions in the SES breeding programs. Investigations aimed at retracing that American family and locating its origins have not proved fruitful (Lewellen, unpublished).

In 1982 an increased amount of the seed of the 2281R1 x MS80NF hybrid was obtained, provisionally coded SES IR1 and released as 'Rizor'. The seed of the pollinator was harvested separately. In 1983 the hybrid was sown in six field trials, three in France and three in Italy (Richard-Molard, 1984; De Biaggi, 1987). The 2281R1 family was sown in the same year in San Martino in Argine for a second cycle of mass selection in severely diseased soil.

Data from the French tests for the SES IR1 (Rizor) gave the following sugar yields in relation to the susceptible checks (table 3): i) much higher in the heavily infected field of Erstein (Bas Rhin); ii) higher in the moderately diseased field in Rougement (Loiret); iii) lower in healthy conditions of Leouville (Eure et Loir). The results of the Italian tests also confirmed that Rizor was superior to both Monodoro (resistant check) and Monofort (susceptible check) in infected soil. In the healthy fields, Rizor yielded on average the same as Monofort.

The mother beets of the 2281R1 family sown in San Martino displayed a fair degree of variability in leaf colouring. As well as those with normal colour, there were approximately 10% that were almost entirely yellowed, while about 40% were an in-between pale green colour. The correspondence between leaf yellowing and symptoms of rhizomania on the roots was remarkable. This relationship, which is useful for visual scoring of the degree of resistance, was far more striking on the French fields.

At harvest, 30 mother beets were selected out of an initial 520. The genetic structure of the 2281R1 family, originating from beets reproduced through open pollination, did not allow precise segregation pattern. It was evident nevertheless that resistance was not a quantitative trait. It was confirmed that the family had preserved a fairly good CLS resistance.

In 1984, about 15 hectares were farmed for seed production of hybrid 2281R1 x MS82NF to be distributed to the growers. In the same year, the 30 selected beets were crossed once more with the MS82NF line to produce the following materials: i) seed of hybrid 2281R2 x MS82NF; ii) seed by open pollination with bulk harvesting of the improved 2281R2 family; iii) a series of full-sib progenies by isolating under paper bags pairs of branches of pollinator plants. Furthermore, all of the pollinator plants were cloned using their respective flower meristematic tissues. The clones were used in the further improvement of Rizor. The new 2281R2 x MS82NF hybrid, initially coded SES IR2, constituted the second version of Rizor (De Biaggi, 1987).

The series of full-sibs sown in greenhouse in September 1984 underwent the ELISA test the following January (Giunchedi *et al.*, 1987). One of these families, displaying a near-zero virus concentration, was coded 2281R3, In the spring of the same year it was crossed with the line MS80/82NF with better combining ability. In this way, a further improved version of Rizor, coded SES IR3, was released (De Biaggi, 1987).

## THE AMERICAN SOURCE OF RESISTANCE

Rhizomania was first identified in California in 1983 (Duffus *et al.*, 1984). Typical symptoms of the disease were observed for several years, but, like in Italy, they were confused with manifestations of cyst nematodes. The symptoms were presumed to be caused by rhizomania based on the experiences of Europe and Japan. Yellow vein symptoms of BNYVV were observed on the leaves in a 1983 field trial in Salinas, California, intended to select resistance to cyst nematodes. Leaves and roots were analysed using an antibody supplied by Tamada & Baba (1973).

Samples of leaves and soils from different growing areas underwent analysis, which revealed that rhizomania was already widespread in several parts of California. Some evidence suggested that BNYVV had been present for several years. It subsequently emerged that the BNYVV isolated in USA is identical to the A type identified in many European countries (Koenig *et al.*, 1995).

As had happened unconsciously in Italy, the breeding program of the USDA in Salinas had probably produced some resistant lines. This may have occurred as a result of resistance identified in several breeding lines of Salinas such as Y439 (Y39, C39R), Y547 (Y47, C47R) (Lewellen & Biancardi, 1990; Lewellen, 1995a).

In the summer of 1983, Erichsen, breeder at Holly Hybrids in Tracy, California, observed that both growth and yield of beet in a variety trial was very low with the exception of three experimental hybrids. These hybrids had been obtained by different pollinators crossed with the same monogerm CMS. It was evident that the behaviour of the three hybrids was due to the female parent. The ELISA tests carried out on the roots confirmed the presence of BNYVV. The hybrids

yielded on average 70 t/ha of roots, whereas the susceptible hybrid USH11 yielded 12 t/ha (Lewellen *et al.*, 1987).

During 1985 and 1986, similar experimental hybrids were evaluated in Salinas in rhizomania conditions (table 4). Compared with varieties of European and Japanese origin the hybrids confirmed the high level of resistance of the Holly monogerm CMS line. As in the trial grown in Tracy in 1983, the Holly hybrids segregated according to a typical scheme of a single dominant gene. It was subsequently established that resistance was transmitted by a single dominant allele called *Rz* (Lewellen, 1988).

In 1986, reselections and increases of the resistant CMS line and of its O-Type were supplied to seed producers in USA and in Europe. The *Rz* allele is now widely used in many commercial hybrids throughout the world. In terms of importance, distribution and use, this trait is only surpassed by genetic monogermity (Savitsky, 1950). The resistant CMS/O-Type counterparts initially presented a particular flower type and several problems of seed quality. Moreover, due to inbreeding depression, it displayed poor seed production and low frost tolerance. Fortunately, these negative characteristics are not related to rhizomania resistance.

Attempts at reconstructing the genealogy of the Holly source of resistance have not proved successful. The Californian germplasm generally had a narrow genetic base as a result of intense selection for resistance to bolting and to other diseases. European materials were superior in terms of sugar content, processing quality, bolting resistance and root shape. As the European germplasm had been derived from long and intense breeding activity, it was useful for improving the American genetic pool. These European germplasm sources had been obtained before 1976; many were polyploid or triploid with male-sterile cytoplasm (Lewellen, 1992).

The material in which the *Rz* gene was identified contained fertile diploid and multigerm plants that were pair-crossed with O-Type monogerm lines. F2 plants were then selected for monogermity and the O-Type trait. The selection work was carried out in Tracy in conditions of pollen isolation. The progeny in which the *Rz* gene was found was self-sterile, but nevertheless produced a small quantity of selfed seed.

A second cycle of screening was carried out within the initial crossings and subsequent selections for monogermity and for the O-Type character. Every couple of monogerm O-Type and the relative CMS was multiplied separately. The plants obtained from each CMS, corresponding to an O-Type monogerm, were crossed with each of the three multigerm testers including the pollinator of the susceptible commercial hybrid HH37. The 1983 variety trial was grown in soil which was subsequently found to be infected with rhizomania. Therefore, the *Rz* gene must have derived from one of the two aforementioned sources.

The CMS line, selected in Sheridan (Wyoming) and Tracy, was selected for its high combining ability for sugar yield and for its resistance to curly top. Its genetic base was complex and its origin unknown. The breeding program of the Holly Sugar Company was based on evaluations of the combining ability of single plants and pairs of plants originating from a broad genetic base population (Helmerik *et al.*, 1965).

Even if the monogerm parents and the other sources were evaluated for their resistance to rhizomania without positive results, it is not entirely impossible that the *Rz* gene was present at a very low frequency level in the monogerm materials. Given the rarity of the *Rz* gene in American germplasm (Lewellen *et al.*, 1987; Lewellen & Biancardi, 1990), many geneticists believe that this gene might have derived from sea beet (Biancardi *et al.*, 2002).

## THE MORE RECENT SOURCES OF RESISTANCE

Soon after the discovery of rhizomania in California, the wild beet (WB) collection of about 60 accessions maintained at Salinas was screened for resistance to the disease (Lewellen & Whitney, 1993). Observations that WB42, an accession from Denmark (Lewellen, 1995b), was resistant in a field test (Lewellen & Whitney, 1993) were confirmed in greenhouse tests employing ELISA criteria (Whitney, 1989).

Individual resistant plants were selected and crossed to line C37. Following one backcross to C37, resistant $BC_1F_1$ plants were increased *inter se* and released as germplasm line C48 (Lewellen & Whitney, 1993). WB42 was also one of the sea beet components in releases C50, C51, and C58 (Lewellen & Whitney, 1993; Lewellen, 2000). Continued backcrosses were made to C37 and resistance from WB42 was also released as germplasm line C79-3 (Lewellen, 1995a; 1997). From WB42 materials provided by Lewellen, Scholten *et al.*, 1999, showed that WB42 resistance was closely linked to *Rz* but was located at a different locus. They named the WB42 allele *Rz2*. In greenhouse and growth chamber tests, WB42 germplasm was shown to condition lower virus concentration in roots than *Rz* (Scholten *et al.*, 1996; Paul *et al.*, 1993).

It is reported (Harju & Richard-Molard, 2002) that the variety Angelina carries both the Holly (*Rz*) and sea beet genes of resistance. Under the conditions of the trials at Pithiviers, France, with the P-type of BNYVV (Koenig *et al.*, 1995), the double resistance of Angelina gives higher resistance than *Rz* alone (Harju & Richard-Molard, 2002). Similar but not such dramatic results also were recently observed in field trials at Salinas under rhizomania and in greenhouse virus baiting evaluations (Lewellen & Liu, unpublished), where Angelina had higher sugar yield than *Rz* varieties (table 5) and the lowest ELISA values for BNYVV.

From the aforementioned sea beet collection, it was possible to isolate other apparently different resistance traits. For example, R22 was developed from a cross between a single sugar beet line (C37) and 60 sea beet accessions (Lewellen, 1992). The F3 generation of the composite cross was released in 1988 as C50 (Lewellen & Whitney, 1993). After several cycles of recurrent phenotypic selection for resistance, a synthetic version of R22 was produced. This synthetic line has shown a better level of protection against the combined damaging effects of rhizomania and root rots caused by high temperature in Imperial Valley, California (Lewellen, unpublished).

## CONCLUSIONS

After about 50 years of observations and breeding activity, two types of rhizomania resistances were identified and exploited in commercial varieties: the multigenic (Alba) and the monogenic (Rizor, Holly, WB42 etc.). With varying combinations of such sources, is possible a satisfactory control of the disease in many countries in presence of different pathotypes of the BNYVV. The differences among the so-called "monogenic resistances" are still an open question because of the presence of not easily detectable minor or modifying genes influencing the segregation patterns. Even the use of molecular analysis did not completely clarify the structure of the chromosomal fragment where the alleles map. As it is confirmed by such analyses carried out by a number of researchers, the origin of all the mentioned resistances appears to be in more or less close relationship with sea beet.

*Tab. 1 - 1957 Variety trials in Italy on fields severely diseased the year before (Bongiovanni & Lanzoni, 1964 ).*

| Locality | Variety | Root yield (t / ha) | Sugar content (%) | Sugar yield (t / ha) |
|---|---|---|---|---|
| Cona (Venice) | Alba P [1] | 20.75 | 9.24 | 1.92 |
| | Buszczynski CLR [2] | 16.80 | 9.98 | 1.68 |
| Cavarzere (Venice) | Alba P [1] | 16.55 | 6.60 | 1.09 |
| | Saros H9N [3] | 7.43 | 5.06 | 0.38 |
| Vighizzolo (Padua) | Alba P [1] | 16.98 | 8.12 | 1.38 |
| | Mezzano NP [1] | 14.82 | 10.30 | 1.53 |

[1] Italian variety with good CLS resistance.
[2] Polish variety with good CLS resistance; derived from Munerati's germplasm.
[3] Hungarian variety with unknown traits.

*Tab. 2 - 1985 Variety trial in severely infected field at San Pietro in Casale (Italy).*

| Variety | Root yield (t / ha) | Sugar content (%) | Sugar yield (t / ha) |
|---|---|---|---|
| Monodoro [1] | 27.89 | 16.24 | 4.53 |
| Ritmo [1] | 35.06 | 16.23 | 5.68 |
| Rizor [1] | 35.31 | 16.89 | 5.96 |
| Buramo [2] | 17.04 | 16.19 | 2.75 |
| Monofort [2] | 11.22 | 16.59 | 1.82 |
| 401 / 81 x MS1 | 21.97 | 16.31 | 3.57 |
| 401 / 81 x MS2 | 26.69 | 17.13 | 4.56 |
| 401 / 82 x MS2-R | 36.81 | 17.07 | 6.27 |
| LSD (P<0.05) | 4.43 | 0.60 | 0.75 |

[1] Resistant variety.
[2] Susceptible check.

*Tab. 3 - 1983 yield evaluations in differently diseased fields.*

| Locality | Rhizomania | Variety | Roots (t / ha) | | Sugar content (%) | | Sugar yield (t / ha) | |
|---|---|---|---|---|---|---|---|---|
| Erstein, France | severe | Rizor [1] | 45.14 | a | 17.80 | a | 8.04 | a |
| | | Monodoro [1] | 20.15 | b | 16.90 | a | 3.41 | b |
| | | Mono 1167 [1] | 19.46 | b | 16.83 | ab | 3.28 | b |
| | | Mono 4086 [1] | 22.70 | b | 17.42 | a | 3.95 | b |
| | | Ritmo [1] | 21.43 | b | 17.35 | a | 3.72 | b |
| | | Dora [1] | 14.69 | d | 15.52 | bc | 2.28 | cd |
| | | Monosvolof [2] | 5.98 | e | 12.35 | d | 0.74 | e |
| Rougemont, France | moderate | Rizor [1] | 49.20 | a | 17.58 | a | 8.60 | a |
| | | Monodoro [1] | 38.20 | b | 16.03 | b | 6.30 | b |
| | | Mono 1167 [1] | 34.96 | b | 16.40 | ab | 5.90 | b |
| | | Mono 4086 [1] | 32.96 | b | 16.60 | a | 5.65 | b |
| | | Ritmo [1] | 39.20 | b | 17.80 | a | 6.86 | b |
| | | Dora [1] | 37.44 | b | 16.20 | b | 6.13 | b |
| | | Monosvolof [2] | 33.92 | b | 15.96 | b | 5.32 | b |
| Leouville, France | healty | Rizor [1] | 58.02 | bc | 17.48 | ab | 10.15 | c |
| | | Monodoro [1] | 60.40 | b | 17.23 | b | 10.41 | c |
| | | Mono 1167 [1] | 53.61 | c | 17.83 | a | 9.55 | c |
| | | Mono 4086 [1] | 54.45 | c | 17.38 | ab | 9.46 | c |
| | | Ritmo [1] | 46.96 | c | 17.75 | ab | 8.86 | c |
| | | Dora [1] | 59.77 | b | 17.70 | ab | 10.58 | b |
| | | Monosvolof [2] | 67.80 | a | 16.90 | b | 11.45 | a |
| S. Martino, Italy | severe | Rizor [1] | 49.71 | a | 15.93 | a | 7.92 | a |
| | | Ritmo [1] | 37.69 | b | 14.94 | a | 5.63 | b |
| | | Monodoro [1] | 41.11 | b | 13.46 | b | 5.53 | b |
| | | Monofort [2] | 22.90 | c | 13.43 | b | 3.07 | c |
| | | Monova [2] | 14.22 | d | 10.10 | c | 1.43 | d |

[1] Resistant variety or experimental hybrid.

[2] Susceptible check.

*Tab. 4 - Yield evaluations of experimental germplasm and commercial varieties in diseased fields at Salinas, CA.*

| Locality (Year) | Rhizomania | Variety | Root yield (t / ha) | Sugar content (%) | Sugar yield (t / ha) |
|---|---|---|---|---|---|
| Salinas, California (1985) | Moderate / severe | 84C39 - 031 [1] | 60.80 | 12.90 | 7.82 |
| | | Rizor [2] | 40.10 | 13.80 | 5.48 |
| | | Ritmo [2] | 31.50 | 12.80 | 4.54 |
| | | Monodoro [2] | 37.50 | 11.90 | 4.50 |
| | | Monohikari [2] | 25.50 | 12.20 | 3.20 |
| | | HH37 [3] | 24.10 | 9.90 | 2.41 |
| | | USH 11 [3] | 20.10 | 9.00 | 1.80 |
| | | LSD (P<0.05) | 8.30 | 1.10 | 1.08 |
| Salinas, California (1986) | Severe | 85C47 - 06 [1] | 39.80 | 14.60 | 5.88 |
| | | Rizor [2] | 25.80 | 14.30 | 3.72 |
| | | Ritmo [2] | 21.30 | 13.40 | 3.20 |
| | | Monodoro [2] | 19.90 | 13.30 | 2.68 |
| | | Monohikari [2] | 22.80 | 13.40 | 3.06 |
| | | HH37 [3] | 19.90 | 12.40 | 2.46 |
| | | USH 11 [3] | 15.40 | 11.60 | 1.79 |
| | | LSD (P<0.05) | 5.30 | 1.00 | 0.77 |

[1] Experimental hybrid with Holly (*Rz*) monogerm lines crossed by HH37 a multigerm diploid susceptible pollinator.

[2] Resistant variety.

[3] Susceptible check.

*Tab. 5 - Performance of hybrids with different resistances to rhizomania grown under moderate disease condition at Salinas, CA, in 2002. Mean of two tests.*

| Variety | Rhizomania Resistances | Root yield (t / ha) | Sugar content (%) | Sugar yield (t / ha) |
|---|---|---|---|---|
| Angelina | *Rz*, WB42 | 78.40 | 19.00 | 14.91 |
| C 927 - 4H5 | *Rz*, R22 * | 80.20 | 18.10 | 14.48 |
| Phoenix | *Rz* | 81.10 | 17.60 | 14.43 |
| Beta 4776 R | *Rz* | 78.00 | 18.30 | 14.28 |
| Rizor | *Rz* | 70.80 | 18.70 | 13.25 |
| USH 11[1] | *rz rz* | 53.30 | 15.60 | 8.36 |
| LSD (P<0.05) | | 7.80 | 0.60 | 1.46 |

* R22 resistance from *B. vulgaris* subsp. *maritima* though population C50 (Lewellen, 1995a).

[1] Susceptible check.

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