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ROOT ROT OF TABLE BEETS IN NEW YORK STATE

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Figure 1. Patch of severely affected table beet plants in a field showing reduced stand and very uneven growth.
Figure 2. Table beet plants throughout a field exhibiting root rot symptoms.
Figure 3. Close-up of infected plants showing the reddish discoloration of the foliage.
Figure 4. Postemergence damping-off symptom exhibited by the seedling on the left.
Figure 5. Seedlings with wire-stem symptom.
Figure 6. Group of plants on the right with wire-stem symptoms, whereas the group on the left are healthy (protected by a drench application of dexon).
Figure 7. Older plants with an advance stage of wire-stem symptom resulting in constriction or severed root systems.
Figure 8. Fleshy beet roots with constriction (one on extreme left) or healed-wound symptom (small surface openings).

Root rot is the most important disease of table beets *(Beta vulgaris* L.) in New York. The disease was first reported by Natti (17) in 1953 as "Dry Rot of Table Beets." He stated that the disease does not occur every year, but sporadic outbreaks in some years can cause severe losses. In recent years, however, root rot has occurred more frequently and is becoming a limiting factor in table beet production. Root rot reduces both yield and quality of beets, causing serious processing problems and increased costs. Field observations have suggested that the initiation of root rot in table beets is associated closely with the cool, wet soil conditions that prevail in early to late spring in New York when considerable acreage of beets is often planted.

Damage and losses due to this disease are expressed as reduced stands, abnormally shaped roots of undesirable size and roots with external or internal rot. Detection and removal of affected roots prior to processing is too expensive and often not very effective. Thus, processors are reluctant to accept beet deliveries from fields with beets exhibiting symptoms of root rot even at low percentages. As a result, growers are often faced with the possibility of losing the entire crop from the suspect field.

Pathogenic fungi known to cause root rot of both table beet and sugar beet include *Pythium* spp., *Rhizoctonia solani, Aphanomyces cochlioides,* and *Phoma betae* (15,21). I n New York, *Pythium ultimum* is the primary causal agent of this disease (5,14,18) and causes severe economic losses during cool, wet soil conditions. Although *R. solani* is encountered less frequently, it is capable of causing seed and seedling diseases of table beets as well as infecting older plants later in the growing season during drier and warmer soil conditions.

The purpose of this bulletin is to illustrate, in detail, the symptomatology and diagnosis of root rot of table beet in New York; to describe the principal pathogens involved and their biology; and to summarize the strategies available for the management of this disease.

SYMPTOMATOLOGY AND DIAGNOSIS

Above-ground disease symptoms in heavily infested fields generally appear in patches of different sizes (Fig. 1) and often in low spots. However, when conditions are very favorable for disease incidence, the plants throughout the field may exhibit disease symptoms (Fig. 2). General symptoms are poor emergence, very uneven growth, dead seedlings and reddish discoloration of above-ground plant parts (Fig. 3). Specific disease symptoms include seed decay and preemergence damping-off, postemergence damping-off, wire-stem, misshapen fleshy roots, and fleshy roots with external or internal rots. The development of one or a combination of the above symptoms in any location will depend on the age and vigor of plants at time of infection, activities of other soil microorganisms, and weather conditions. **Seed decay and damping-off disease symptoms—**
Seedballs of table beets may become infected and decayed priorto germination (seed decay). Very young seedlings may also become infected and die before they can emerge above the soil surface (preemergence damping-off). Emerged healthy seedlings may become infected and exhibit a water-soaked and necrotic area just below or at the soil line (Fig. 4). The latter type of infection may result in wilting, collapse, and death of severely infected seedlings (postemergence damping-off).

Wire-stem symptoms—The stem and main root regions of 2- to 4-week-old infected seedlings that survive the postemergence damping-off stage usually become partially or completely shrivelled, giving them a thread-like appearance (wire-stem symptom; Figs. 5, 6). The infected regions are brown to black. Seedlings with wire-stem symptoms may have normal branching fibrous root systems, or roots that are brown and at different stages of rotting. Severely infected plants are stunted and reddish-purple. If plants are stressed and the infection progresses, infected roots may rot off just below the soil surface (Figs. 6, 7), and result in plant death and a reduced stand.

Abnormal and infected fleshy root disease symptoms—Later in the growing season, infected plants that survive the wire-stem phase develop abnormal fleshy roots (Figs. 8,11,14,19, 20). Infected tissues of the root and stem enlarge more slowly than the surrounding healthy tissues, leading to the formation of constrictions of various shapes and sizes. At harvest, infected fleshy roots may exhibit several dry rot symptoms. The rotted tissues are generally firm and dry, brown to black and sharply delimited by healthy tissue (Figs. 9,10,12,13). The rotted areas range in size from a small lesion to the whole root. Fleshy roots may have small surface openings that often are difficult to detect (Fig. 8). These openings are connected to limited rotted areas (hence, the term "healed wound") or to rather extensive internal rotted areas (Figs. 9, 10). Fleshy roots at advanced stages of rot have large openings, with extensive portions of the roots discolored and decayed (Figs. 11, 12). Infection of the fleshy roots may also occur through the petioles in the crown area resulting in a downwardly progressing rot (Figs. 13, 18).

Infected fleshy beets at harvest time may also have superficial lesions that are only a few cells deep (Figs. 16, 17) and thus are of no economic significance. However, darker and deeper lesions also have been observed on fleshy roots in midseason or at harvest time (Fig. 13). Infections seem to occur through the sides of the fleshy roots and also the crown area. These lesions may progress rather rapidly, covering the entire fleshy root (Fig. 14). Infected tissue is black and somewhat soft. Plant-to-plant spread often occurs and

results in bare spots within the row as infected plants die (Fig. 15). These symptoms are typical of infection by *Rhizoctonia solani* which generally infects beets later in the growing season, when soils are warmer and drier. Various growth cracks (Fig. 21) also are observed but they do not appear to relate to root pathogen activities, but rather to physiological factors.

IDENTITY OF CAUSAL ORGANISMS

To determine the major fungal pathogen(s) associated with beet root rot, extensive isolations were made from infected stem and root tissues obtained from field- or greenhouse-grown beets. Isolations were usually made at weekly intervals utilizing several general agar media (acidified water agar, acidified potato-dextrose agar, and cornmeal agar), the selective medium of Ko and Hora (16) and a modification of the Tsao and Ocana medium (20). Isolation data obtained from field-collected plants during several growing seasons showed that *Pythium ultimum, Rhizoctonia solani* and several *Fusarium* spp. (including *F. roseum, F. solani, F. oxysporum,* and *F. moniliforme)* were the fungi most often associated with beets exhibiting root rot symptoms (5,14). These data also showed that *P. ultimum* was often the first fungus to be isolated from young seedlings, whereas *Fusaria* were predominantly isolated from infected tissues later in the growing season and especially at harvest time. Representative isolation data obtained from a beet field near Bellona, New York, are presented in Table 1.

Table beets were also grown in the greenhouse in field soils collected from locations with histories of severe root rot outbreaks. All disease symptoms were reproduced in greenhouse conditions including the healed wound and rot disease stages (Figs. 14, 16). The pattern and frequency of fungal isolation dat obtained from the greenhouse-grown beets were identical to those obtained from field-collected samples.

The pathogenicity of representative isolates of *Pythium, Rhizoctonia* and *Fusarium* species to table beets was evaluated alone and in all combinations in the greenhouse using field or sterilized soils. Results of numerous tests demonstrated that all the isolates of *Fusarium* species tested were not pathogenic, on their own, to table beets (Table 2). Thus, these species of *Fusarium* occur only as invaders of previously diseased or injured tissues. All isolates of *Pythium* and *Rhizoctonia* species evaluated were highly pathogenic to beet seedballs and seedlings up to three weeks old (Table 3). These fungi caused seed decay, pre- and postemergencedamping-off and wire-stem symptoms, and were not distinguishable from each other by symptoms alone. Isolates of *Rhizoctonia* also were capable of causing surface lesions on older plants.

Many greenhouse and field trials have been conducted to determine the efficacy of registered and

Table 1. Frequency of isolation of fungi from infected beet plants collected from a commercial field near Bellona, New York.

Sampling date (days after planting)	No. of fungi recovered/No. of plants examined[a]	Identity of fungi isolated[b]		
		Pythium ultimum	*Rhizoctonia solani*	*Fusarium* spp.
5	0/11	0	0	0
14	4/10	4	0	0
20	6/10	6	0	0
26	7/10	6	0	1
32	8/10	2	1	5
36	8/10	3	2	6
41	7/10	5	0	4
43	10/10	1	1	9
47	8/10	3	2	6
51	9/10	0	2	6
58	10/10	5	3	4
104[c]	44/50	2	2	35
104[d]	37/50	2	0	32
104[e]	36/50	1	3	28
104[f]	13/30	0	2	13

[a]Samples were washed for 1 hour in running **tap** water prior to planting on the agar media.

Other fungi isolated occasionally included species of *Phoma, Altemaria, Mucor, Cephalosporium,* and *Mycelia sterilia.*

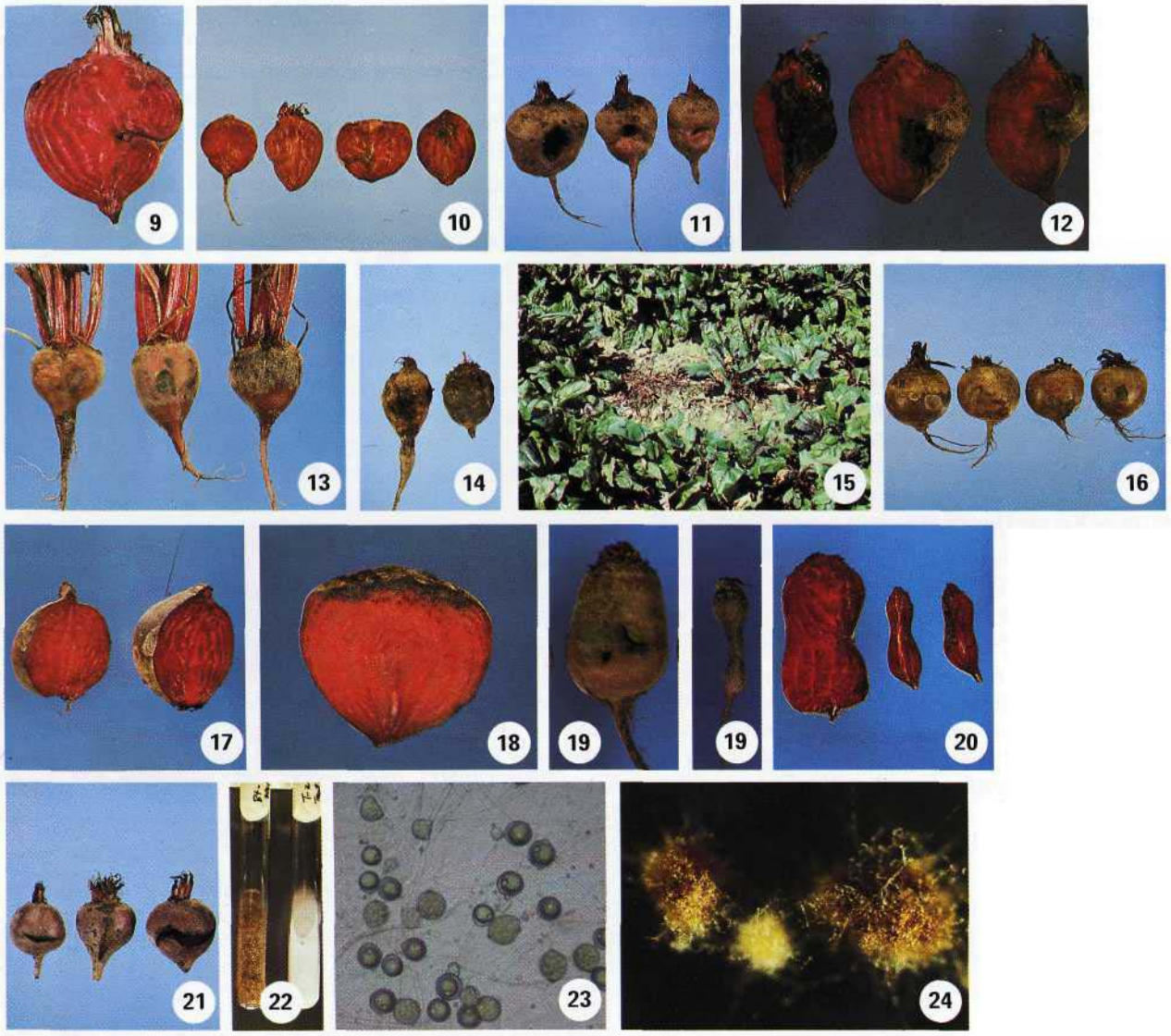
^'Samples were collected at harvest time with fleshy roots exhibiting rot, healed wound, superficial **surface** lesions, or growth cracks, respectively.

Table 2. Pathogenicity of several isolates of *Fuaarium* to nongerminated Ruby Queen beet seedballs and 1-, 2-, or 3-week-old seedlings.

Fusarium isolates	No. seedlings killed/ No. tested				Avg. weight of surviving plants (grams)			
	Seedballs	1-wk	2-wk	3-wk	Seedballs	1-wk	2-wk	3-wk
None	1/37	0/10	0/10	0/10	21.6	22.3	19.5	16.0
Fusarium #1	0/16	0/5	0/5	0/5	15.6	19.1	17.1	21.9
Fusarium #2	0/19	0/5	0/5	0/5	16.3	15.6	15.4	18.2
Fusarium #5	0/14	0/5	0/5	0/5	15.9	16.2	15.9	17.2
Fusarium #7	0/20	0/5	0/5	0/5	19.0	16.6	19.8	22.7
Fusarium #8	0/33	0/10	0/10	0/10	15.5	16.8	14.8	14.9
Fusarium #9	0/13	0/5	0/5	0/5	20.4	16.4	18.5	22.1
Fusarium #10	0/19	0/5	0/5	0/5	20.8	16.0	20.3	22.1
LSD 0.05					5.1	5.1	5.4	5.9

Table **3. Pathogenicity of one isolate each of *Pythium ultimum, Rhizoctonia solani* and *Fusarium oxysporum* isolated from field-grown beet plants to 1-, 3-, or 5-week-old Ruby Queen seedlings.**

Pathogen(s)	No.seedlings killed/No.tested			Avg.plant wt.(gm)/age group		
	1-wk	3-wk	5-wk	1-wk	3-wk	5-wk
P. ultimum	9/10	3/10	0/10	4.3	25.2	37.4
R. solani	3/10	5/10	4/10	29.5	26.5	16.0
F. oxysporum	0/10	0/10	0/10	36.5	40.7	44.1
P. ultimum + *F. oxysporum*	9/10	0/10	0/10	0.6	34.8	41.8
R. solani + *F. oxysporum*	3/10	8/10	5/10	25.2	7.6	21.0
P. ultimum + *R. solani*	10/10	9/10	9/10	0.0	6.8	4.9
P. ultimum + *R. solani* + *F. oxysporum*	9/10	5/10	4/10	2.8	22.4	17.1
None	0/10	0/10	0/10	36.2	36.7	47.4
LSD 0.05				10.4	18.5	13.9



Figures 9 and 10. Vertically cut fleshy roots with healed-wound symptom exhibiting different degrees of internal rot.
Figure 11. Fleshy roots with rot symptoms (large openings).
Figure 12. Vertically cut fleshy roots with rot symptoms showing the extensive, but sharply delimited, rotted areas.
Figure 13. Fleshy roots with dark and deep lesions. These roots also exhibit infected (dark and shrivelled) petioles.
Figure 14. Severely infected fleshy roots showing symptoms of attack by Rhizoctonia solani (originating from deep surface lesions).
Figure 15. Symptoms of plant-to-plant spread of disease resulting in a bare spot.
Figure 16. Fleshy roots with superficial lesions.
Figure 17. Vertically cut fleshy roots with superficial lesions.
Figure 18. Fleshy root infected from the top through the crown area and via the petioles attachment.
Figure 19. Infected fleshy roots exhibiting surface opening and constriction produced in the greenhouse in naturally infested soil.
Figure 20. Vertically cut fleshy roots with healed-wound and rot symptoms produced in the greenhouse.
Figure 21. Fleshy roots with growth cracks symptoms.
Figure 22. Pythium ultimum (right) and Rhizoctonia solani (left) growing on potato-dextrose agar in glass tubes.
Figure 23. Sporangia (thin-walled) and oospores (thick-walled) of Pythium ultimum.
Figure 24. Close-up of sclerotia of Rhizoctonia solani.

experimental fungicides for the control of beet root rot in New York. Excellent control of the disease has often been obtained with the fungicides dexon (Lesan), metalaxyl (Ridomil, Apron), or previcur (2-4, 7, 8, 18). All these fungicides are known for their high and specific activities against the water mold fungi, including *P. ultimum.* In contrast, the use of a fungicide like terraclor (PCNB), benomyl (Benlate) or demosan (Chloroneb) as seed or soil treatment has often failed to control the disease in natural field soils, especially during the early part of the growing season. Terraclor, benomyl, and demosan are known to be very effective for the control of *R. solani* and its diseases on many crops. The combined use of both types of fungicide as seed or soil treatments has been most effective in controlling the disease, especially in warmer and drier soils (1,18).

The information presented above and extensive field observations strongly suggest that *P. ultimum* is the most important pathogen causing root rot of table beets in New York. This conclusion is especially true during cool and wet soil conditions that often prevail during the early part of the growing season when a large part of the beet acreage is planted. *Rhizoctonia solani* is capable of causing serious damage, probably during unusually warm periods early in the growing season but more likely causing lesions and rots later in the season when soil conditions are generally warmer and drier.

BIOLOGY OF *P. ULTIMUM AND R. SOLANI*

Pythium ultimum is widely distributed in New York (6, 20) and in many other agricultural areas (10, 13). Results of a recent survey showed that the population of this fungus in New York soils ranged from 37 to 2,426 growth-generating units per gram soil with an overall average of 599. It has a very wide host range including many of the major vegetables grown in New York such as beans, cabbage, cucumber, lettuce, melons, and peas; many agronomic crops; and weeds. The fungus grows quickly, producing abundant fluffy white masses of strands (hyphae) on host tissues or agar media (Fig. 22). The fungus also produces thin-walled vegetative reproductive structures (sporangia) and thick-walled sexual spores (oospores) (Fig. 23). Sporangia survive in soil only for several months, but oospores can remain viable for several years even in the absence of a host crop. This fungus can also survive in infected tissues, or by attacking hosts and colonizing crop residues in soil, especially in moist soil. *Pythium ultimum* is considered a low temperature species and most damaging to table beets and other hosts during cool and wet soil conditions. However, the fungus has been shown to grow well at higher temperatures on agar media on which the optimum temperature is between 20 C to 25 C. In laboratory tests, P. *ultimum* infected table beets at soil moistures considerably below the field capacity level. Thus, the severity of damage in cool and wet conditions is probably due to the indirect effect of these factors in reducing competition of other microorganisms in soil and also the lower growth rate of the host plants in such conditions (10, 12).

Rhizoctonia solani is widespread throughout the vegetable areas of New York (6,9) and other locations (19). Like P. *ultimum,* it attacks many weeds and crops including beans, cabbage, lettuce, peas and potatoes. The population of *R. solani* among vegetable crop fields in New York varied from 1 to 9 (average: 5) growth-generating units per 100 grams of soil. The fungus generally produces light to dark brown masses of strands and compacted, seed-like structures, called sclerotia (Figs. 22, 24). The fungus, in the form of hyphae, survives in soil in colonized host tissues or as sclerotia free in soil. The fungus exists in soil in many forms, which differ in their ability to attack table beets and other hosts. The most severe damage to beets occurs in relatively warm, dry soils. The optimum temperature for the growth of most forms of the fungus is 24-29 C.

CONTROL STRATEGIES

As a result of the major involvement of .P. *ultimum* in inciting beet root rot in New York, the application of single control measures, such as seed and soil treatments with selective fungicides, can be very effective in controlling the disease. However, it is more appropriate and practical to practice an integrated control approach for the long-term management of this disease. All effective and practical control measures that are known to reduce the soil population of the pathogens and their damage to table beets should be used. Control measures should be practiced in infested fields and also in clean fields to prevent buildup of the problem.

Chemical Control Measures:

1. Fungicide-treated seed—Extensive greenhouse and field trials were conducted during the past several years to identify the most effective fungicide seed treatments. Results have demonstrated the need to use a highly effective fungicide against P. *ultimum.* Dexon (Lesan) has been an effective fungicide, but once current supplies are exhausted this material will no longer be available. Several experimental fungicides have recently been shown to be very effective against P. *ultimum.* Apron (seed treatment formulation of metalaxyl) plus thiram or captan as slurry treatments have consistently performed well and better than other treatments. (Apron has just received EPA approval for use as a beet seed treatment.) Thiram or captan alone does not control P. *ultimum* satisfactorily under severe

disease pressure. However, thiram is effective against many other fungi including *Phoma betae.*

2. Fungicide soil treatment—This is an effective short-term control measure for beet root rot. Ridomil (flowable formulation of metalaxyl) appears very effective and currently is being evaluated as a in-furrow or over-the-row spray application. It is also available in granular formulation in combination with terraclor. Current plans are to work through the IR-4 program for registration of Ridomil for use on table beets as a soil treatment at planting time. Preplant soil treatment with broad-spectrum, soil fumigants such as methyl bromide, chloropicrin, vorlex and telone C-17 are effective against the beet root rot fungi but their use is not practical or economically feasible.

Cultural Control Measures:

1. Crop rotation and cover crops—Whenever possible, crop rotations that include grain crops such as corn, barley, wheat and oat should be followed. Continuous table beet production will increase populations of the beet root rot fungi and increase disease severity. Also, crops susceptible to the beet root rot fungi such as beans, cabbage, peas or potatoes should not be considered as rotational crops as they too will increase populations of the pathogens. Plowing cover crops under may reduce root rot severity if enough time is allowed for residue decomposition prior to planting. The beneficial effect may be due to improved soil structure or the increased activity of beneficial soil microorganisms.

2. Plowing and seedbed preparation—Root rot pathogens are most abundant in the top 15-20 centimeters ofthesoil. Deep plowing and turning under of infected debris will reduce the population of root rot fungi. Reducing soil compaction by subsoiling or chiselling below the plowed layer will increase drainage, pro mote deeper and greater root formation and increase crop tolerance to root rot damage. Growing table beets on ridges will reduce damage by *P. ultimum.* Ridging will increase soil temperature and reduce soil mois ture, and thus provide conditions less favorable for infection and damage to beets by *Pythium.*

3. Site selection and planting date—Sites that are well-drained with good soil structures are less conducive to damage by *P. ultimum.* Whenever possible, heavy-textured soil with a history of severe root rot incidenceshould be planted late when the soil has had adequate time to warm.

4. Herbicide effects—The role, if any, of herbicides used on table beets, on the incidence and severity of root rot, has been evaluated extensively in greenhouse and field tests. In greenhouse tests, both RoNeet and Pyramin had no effect on seedling emergence and only slightly reduced final counts when used at recommended rates (Cornell Recommendations for Commercial Production; Publication Office, Cornell University, Ithaca, New York 14853). The combined use of RoNeet and Pyramin at the full (but not at the three-quarters) recommended rate of each significantly increased root rot and decreased stand counts. The use of Solubor at the full or three-quarters (but not at the one-half or one-quarter) recommended rate applied in a drench treatment resulted in lower emergence and stand counts, and also reduced seedling growth. Preplant application of RoNeet or Pyramin at full rate or their combination at three-quarters rate increased the injury from Solubor at the recommended rate.

Results of field trials on the effects of the three herbicides on root rot severity and yield of table beets have been inconclusive. Nevertheless, the three herbicides should not be used together and a lower rate of Solubor should be applied. The application of Solubor as a fertilizer supplement instead of an over-the-row band spray application should be considered.

5. Fertilizer effect—A series of field trials were conducted in cooperation with N. H. Peck and M.T. Vittum (Department of Horticultural Sciences, Geneva) to determine the role of fertilizers used on table beets on the incidence and severity of root rot. Results showed that all fertilizers used in all placement patterns that were evaluated revealed no significant effect on rot incidence or severity (8). However, any program that results in increased plant vigor, especially during the seedling stage (first three weeks after planting), will increase plant tolerance to root rot.

Biological Control Measures:

1. Resistant germplasm—A broad range of beet germplasm was evaluated for resistance to *P. ultimum* in greenhouse or field tests. Greenhouse tests consist of transplanting 8- to 10-day-old seedlings into pasteurized soil infested with 200 growth-generating units (sporangia) of P. *ultimum* pergram of soil. Susceptible seedlings generally become infected and die within the first two-three weeks. Field tests are now conducted in a table beet root rot nursery. The field soil of this nursery has received pasteurized soil heavily infested with *P. ultimum* for three consecutive years.

Original screening of beet germplasm to root rot was conducted in 1962 by J. J. Natti (Department of Plant Pathology, Geneva). He evaluated a total of 153 Plant Introduction (P.I.) beet collections along with 10 cultivars for damping-off resistance. The test was conducted in a field with a long history of vegetable crop production. Thus, any of several pathogenic fungi such as *Pythium, Rhizoctonia,* or *Fusarium* may have affected emergence and seedling establishment. Damping-off in his test ranged from 6 to 100 percent. All the lines that showed 20 percent or less damping-off from Natti'stest, along with a large number of table beet cultivars, breeding lines, and other selected collections, have been evaluated first by the new greenhouse test, specifically against P. *ultimum.* None of the

commercial cultivars or advanced breeding lines were tolerant to *P. ultimum.*

Of the many P.I. accessions evaluated, only two selections (P.I. 164810 and P.I. 175046) were moderately tolerant as they consistently had the greatest number of surviving plants. Unfortunately, both of these accessions are wild-type beets (annual and without a fleshy root). G. A. Marx (Department of Horticultural Sciences, Geneva) initiated a project in which the main objective was to transfer this resistance into commercially acceptable cultivars. Progenies of advanced generations from crosses between the wild-type parents and commercial cultivars (principally Ruby Queen) have been obtained with good horticultural characteristics. To date, none of the progenies has proven to be as tolerant as the parent selections. However, selection and retesting of promising progenies are continuing.

2. Use of antagonistic beneficial microorganisms— This approach, utilizing the addition to soil of beneficial soil microorganisms that adversely affect the root rot pathogens, was initiated in cooperation with H. C. Hoch (Department of Plant Pathology, Geneva). To date, one fungus *(Laetisaria arvalis)* has given good control of the seed decay and damping-off diseases incited by *P. ultimum* (11). This beneficial fungus reduced disease incidence and also prevented the population buildup of the root rot fungus. It was effective as a seed treatment or when applied to soil in the form of colonized substrates such as wheat bran, beet pulp or cornmeal. However, the production and application of this fungus for the control of P. *ultimum* is not practical or economical at this time. Nevertheless, work with this beneficial fungus is continuing since it is also active against the other table beet pathogens, *R. solani* and *Phoma betae* (14).

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