

Diseases Limiting Production of Jerusalem Artichokes in Georgia

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ABSTRACT

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Rust caused by *Puccinia helianthi*, powdery mildew caused by *Erysiphe cichoracearum*, southern blight caused by *Sclerotium rolfsii*, and tuber rots caused by either *S. rolfsii* during the growing season or by *Fusarium* and *Pseudomonas* spp. during refrigerated or field storage were the most important diseases of Jerusalem artichokes in Georgia during 1980-1982. Rust caused a severe foliage blight, and tuber yields from unsprayed plots were 29% lower than those from plots sprayed with mancozeb. *S. rolfsii* caused a 60% plant loss in plots in which Jerusalem artichokes had grown the two previous years, and yields from these plots were only 40% of those treated with methyl bromide. Metam-sodium and PCNB were only moderately effective in reducing losses to *S. rolfsii* and in increasing yields. Incorporation of tubers into soil increased disease severity, indicating that residual tubers from a previous crop may serve as a food base for the pathogen. Rot organisms caused serious deterioration of tubers held in sandy soils during the fall and winter. Results indicate that diseases may limit the potential of Jerusalem artichoke as a fuel alcohol crop in the southeastern United States unless a suitable crop rotation is followed.

Jerusalem artichoke (*Helianthus tuberosus* L.), a plant native to North America, was being cultivated for its edible tubers by the Indians when the European explorers arrived in America (13,14). It was soon introduced into Europe and became a significant crop produced for both animal feed and human consumption. In the United States, it has remained a minor food crop and is sometimes considered a weed pest (14). In recent years, however, Jerusalem artichoke has received increased attention as a potential crop for fructose (2) and fuel alcohol (7,12) production. Some studies have indicated alcohol yields as high as 5,610 L/ha from tubers of the crop (12). Production research initiated in Georgia in 1980 indicated that rust (*Puccinia helianthi* Schw.), powdery mildew (*Erysiphe cichoracearum* DC.), southern blight (*Sclerotium rolfsii* Sacc.), and tuber rots of then unknown causes may limit commercial production of Jerusalem artichoke in the southeastern United States. This paper reports results of research conducted during 1981-1982 to determine yield losses caused by selected foliar and soilborne diseases, to determine the organisms responsible for tuber rots, and to evaluate certain control measures.

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MATERIALS AND METHODS

Research plots were located at the University of Georgia Horticulture Farm near Athens. Routine land preparation and cultural practices were used. Tuber seed pieces of the Mammoth French White cultivar were planted 30 cm apart in bedded rows spaced 0.96 m apart. Irrigation was applied as needed with overhead sprinklers.

Rust test. Rust was severe in production research plots in 1981. A test was conducted in the same general area in 1982 to study rust development and to determine tuber losses caused by the disease. Yield losses were determined by controlling rust chemically (mancozeb [Manzate 200], 2.24 kg/ha). Three-row plots 6.2 m long and separated by two unsprayed border rows were established on 17 May. Six replicates of sprayed and unsprayed plots were arranged in a randomized complete block design. The fungicide was applied to runoff with a back-mounted Solo mistblower beginning when plants were about 40 cm tall and continuing at 7- to 10-day intervals until 2 wk before harvest on 15-16 November. Rust development was recorded in each plot, beginning when the first uredia appeared. At harvest, some plants had many of their tubers destroyed by rot organisms. Plants were harvested individually by hand, and only those with minor tuber rot were harvested for yield.

Greenhouse tests were conducted to determine whether the rust strain prevalent on Jerusalem artichoke in Georgia could also infect sunflower (*Helianthus annuus* L.). Greenhouse-grown Teddy Bear (Yellow Pigmy) and Giant Gray Stripe sunflower plants 10-15 cm tall were atomized with uredospores collected from Jerusalem artichoke

plants in the field, held in a dew chamber at 25 C overnight, and placed on a greenhouse bench to allow disease development.

Southern blight test. *S. rolfsii* caused high plant and yield losses in 1980 and especially in 1981, when plants were grown on land previously planted to Jerusalem artichoke. In 1982, a field plot was established to quantify losses and evaluate selected chemical and cultural control measures. The plot area had been planted to Jerusalem artichoke the two previous years and had a high natural infestation of *S. rolfsii*. Some tubers and tuber pieces remained in the soil from the previous year's crop. Four-row plots 4.6 m long were established in early May. Six treatments with four replicates were arranged in a randomized complete block design: methyl bromide (Dowfume MC-2), 487 kg/ha applied under a polyethylene cover; metam-sodium (Vapam), 468 L/ha applied as a drench in 20,000 L of water; PCNB (Terraclor 75W), 17 kg/ha applied as a drench; tubers (remaining from the 1981 crop) removed from the soil; tubers added to the soil; and a check. All treatments were applied within a 3-wk period before planting on 17 May. The tuber removal and added treatments were included to test the hypothesis that residual tubers were contributing to southern blight severity with repeated culture on the same land. Tubers were removed by repeated tilling, raking, and hand collection and were incorporated at the rate of 3.2 t/ha with a rotary tiller into plots that were to receive extra tubers. No attempt was made to control rust or other foliar diseases. Weekly counts were made of dying plants to follow the progress of southern blight. Final stand counts were taken and tubers harvested from the two center rows of each plot for yield on 10-11 November.

Tuber rot studies. Tubers from the field were examined for rot each season from 1980 through 1982 and causes of the rots were determined. Isolations were also made from tubers held in refrigerated storage. Diseased tubers were washed free of soil, surface-disinfested for 2 min in 0.5% sodium hypochlorite, rinsed, and sections placed on water agar and potato-dextrose agar (PDA). Typical colonies were transferred and later identified. Bacterial isolations were made by macerating excised tissue in sterile distilled water, allowing the preparation to stand for 20 min, and streaking for isolation on plates of nutrient yeast-

dextrose agar (23 g nutrient agar, 5 g yeast extract, 10 g glucose, and 1 L water) and medium B of King et al (5). Bacteria were identified only if shown later to have rotting potential. Fluorescent pseudomonads, the predominant bacteria capable of causing rots, were identified using published determinative methods and keys (3,8).

Pathogenicity tests were conducted to determine rotting potential of the fungal and bacterial isolates. Disease-free tubers were washed and surface-disinfested as described. Inoculations with fungi were made by removing a cylinder from tubers with a sterile 8-mm cork borer and inserting a mycelial plug of the test fungus or by cutting tubers in half and placing a mycelial plug on the cut surface. Inoculated tubers were placed in 9-cm glass or 25-cm plastic containers with covers. For bacterial inoculations, tuber slices 7 mm thick were cut aseptically and placed in a petri dish. A loopful of a turbid suspension of bacteria was introduced into a 2-mm wound made in the center of each slice. Tuber sections inoculated with fungi and bacteria were kept moist during incubation by placing water or moist filter paper in the bottom of the dishes. Initial tests were conducted at 25 C. Later, selected pathogenic isolates of *Fusarium* and *Pseudomonas* spp. were tested for their rotting capacity

at 5, 15, 25, and 35 C. The effect of these temperatures on the in vitro growth of selected *Fusarium* isolates was also determined by placing mycelial plugs in the centers of plates containing PDA and measuring radial growth daily.

RESULTS

Rust tests. Observations made in production test plots in 1981 indicated that rust caused significant tuber yield reductions, but loss determinations were not possible because all plots were uniformly infected. Rust development was observed closely in 1982 and yield losses determined. The first uredia were observed on 3 August, when plants were about 1 m tall. The disease developed rapidly once introduced into unsprayed plots and border rows, and within 2 wk, numerous pustules had appeared on all foliage, especially on the abaxial surfaces (Fig. 1). A few pustules appeared on stems. Foliage often became infected in the early stages of development and never reached full size. Pustules were so abundant that the foliage became "blighted," with symptoms starting at the bottom of each plant and moving toward the growing point. Most foliage had been killed by heavy rust infection by the end of the growing season. In mid-October, the uredial stage was rapidly converted to the black telial stage. All plots sprayed with mancozeb were essentially free of rust and remained green throughout the growing season until frost. The rust significantly (*t* test, $P = 0.05$) reduced tuber yields. Mean yields of tubers from sprayed and unsprayed plots were 2.98 and 2.12 kg/plant, with a projected yield of 45.1 and 32.1 t/ha, respectively.

Rust was the only foliage disease serious enough to cause significant yield reduction. Powdery mildew appeared in the plots, but infection was sporadic and light.

In greenhouse tests, rust uredospores collected from field-grown Jerusalem artichoke plants caused infection and produced uredia on Giant Gray Stripe sunflower plants. Teddy Bear sunflower plants were infected (cleared leaves had rust mycelium), but no uredia developed.

Southern blight test. Significantly more plants emerged from tuber seed

pieces planted in soil treated with methyl bromide and metam-sodium than from seed pieces planted in soil without chemical treatment (Table 1). The cause of seed piece failure in the untreated soil could not be determined accurately; however, the presence of a profuse growth of typical mycelium of *S. rolfssii* on the rotting seed pieces indicated that this organism was responsible. Tuber removal and tuber addition treatments had no significant effect on plant emergence. Lower emergence from seed pieces planted in PCNB-treated soils indicated phytotoxicity at the rate used.

S. rolfssii caused extensive plant losses during the growing season in plots that did not receive chemical treatments (Table 1). More plants were killed in plots where tubers were removed or added than in plots where no treatment was used. Plants died most rapidly and uniformly in plots where tubers were removed. In all plots, disease was most severe during July and August, when high temperatures and frequent rains were common. Mean temperatures and total rainfall were 26.3 C and 14.8 cm for July and 25.1 C and 12.9 cm for August. The disease was easily recognized by the presence of masses of white mycelium and sclerotia near the soil line that often extended 8 cm or more on each side of the plant stems (Fig. 2). Methyl bromide was highly effective in preventing losses to *S. rolfssii* during the early growing season, but some losses occurred late in the season when plots became infested from surrounding untreated plots. Metam-sodium and PCNB were only moderately effective in controlling the disease.

The high mortality rates were reflected in significantly reduced tuber yields (Table 1). Yields from plots where tubers were removed or added or where no treatment was used were 21, 28, and 40% of those where soil was treated with methyl bromide. Metam-sodium and PCNB significantly increased yield but were less effective than methyl bromide. Tuber rot caused by *S. rolfssii* was common in all plots, even those treated with methyl bromide.

Tuber rot studies. Observations made from 1980 through 1982 indicated that *S. rolfssii* was the most important cause of tuber rot during the summer growing

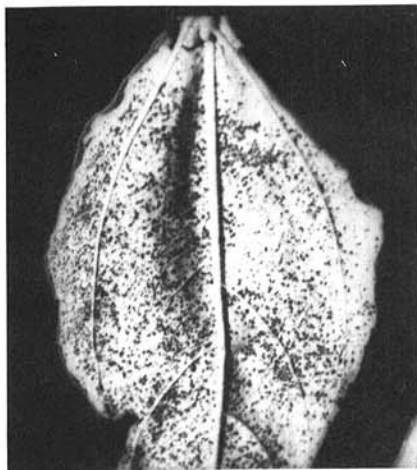


Fig. 1. Abaxial side of Jerusalem artichoke with numerous uredia of *Puccinia helianthi*.

Table 1. Effect of selected treatments on emergence, mortality, and tuber yield of Jerusalem artichoke grown in soil with a high natural infestation of *Sclerotium rolfssii*

Treatment ^a	Emergence (%)	Mortality ^b (%)	Yield (t/ha)
Methyl bromide	100.0	15.0	28.2
Metam-sodium	91.7	46.7	18.4
PCNB	73.3	49.0	18.7
Tubers removed	81.7	77.3	5.8
Tubers added	80.0	80.7	7.8
Check	83.3	60.0	11.4
FLSD ($P = 0.05$)	8.2	16.4	3.3

^aRates of methyl bromide, metam-sodium, and PCNB were 487 kg, 468 L, and 17 kg/ha, respectively—all applied before planting.

^bIncludes seed pieces rotted before emergence and plants killed by *S. rolfssii* during the growing season.



Fig. 2. Southern blight of Jerusalem artichoke caused by *Sclerotium rolfssii*. Note profuse growth of robust mycelium surrounding stem.



Fig. 3. Cluster of tubers of Jerusalem artichoke (A) completely destroyed by rot organisms compared with (B) a healthy cluster.

season. It was easily recognized by the presence of profuse growth of robust white mycelium that caused a white to light brown soft rot, sometimes involving the entire cluster of tubers on a plant. Other types of rots were observed when tubers were held in refrigerated storage or in the field during late fall and winter. The most common was a dark black rot that often started at the tuber bud tips and progressed slowly into the tubers. At times, all the tubers on a plant were destroyed (Fig. 3). This rot and similar rots were most serious when plants were grown on sandy soils. Isolations from rotted tubers collected from the field and taken from storage during 1980-1982 yielded primarily *Fusarium* spp., the most common being *F. roseum* (Lk.) Snyd. & Hans. and *F. oxysporum* Schlecht. (9). In one typical series of isolations, 49 fungal isolates obtained from rotted tubers were identified as follows: 43 were *Fusarium* spp., 4 were *Alternaria* sp., and 1 each was *Trichoderma* sp. and *Oedocephalum* sp. In laboratory pathogenicity tests with these fungal isolates, 13 of the *Fusarium* isolates caused rot of tubers, whereas none of the others was pathogenic. In laboratory temperature studies at 5, 15, 25, and 35 C in which six of the 13 *Fusarium* isolates were tested, rot was most severe at 25 and 30 C. No rot occurred at 5 C, and only two of the isolates caused rot at 15 C after 2 wk. Optimum temperature for vegetative growth of the isolates on PDA was 25 C. *Rhizoctonia solani* Kühn was also occasionally the cause of a brown rot of tubers.

High populations of bacteria were consistently associated with rotted tubers in the field and in refrigerated storage, and some strains caused rot when tested in the laboratory. In one series of tests, 10 of 67 bacterial strains obtained from

diseased tubers caused rot of tuber slices. One of these was weakly virulent and was not identified. The other nine were oxidase and arginine dihydrolase positive fluorescent bacteria that were subsequently identified as *P. marginalis* (Brown) Stevens (five isolates), *P. fluorescens* Migula (three isolates), and *Pseudomonas* sp. (one isolate). All isolates produced a negative or variable hypersensitive reaction on tobacco (6). In temperature studies, the isolates of *P. marginalis* and the *Pseudomonas* sp. caused rot over the entire temperature range (5-35 C), with an optimum at 25 C. The isolates of *P. fluorescens* caused rot at 5-25 C but not at 35 C.

DISCUSSION

Results confirm initial observations that rust, southern blight, and tuber rots can be serious enough to limit commercial production of Jerusalem artichoke in the southeastern United States. Powdery mildew was moderately severe in some plots, but yield losses caused by the disease were not determined. The severity of diseases was unexpected because Jerusalem artichoke was reported to be relatively free of diseases when grown on a limited basis in other areas (7,12). Rust was not a problem in research and production trials conducted in recent years in Minnesota (L. Waters, *personal communication*) and Canada (F. Kiehn, *personal communication*). However, Shoemaker (13) reported that a rust outbreak reduced yields in research plots near Rosslyn, VA, in 1926 on land used for the same crop the previous year. Burning of stalks and change of location was recommended in the 1920s for control of rust in Europe (13). Sclerotinia wilt, the disease of greatest importance in other areas (7,13), has not been observed in Jerusalem artichoke in Georgia, probably because of high temperatures.

Rust reduced yields enough in our trials to warrant control measures. Although rust was controlled very effectively by chemical means, breeding and selection for resistance seems to be a more practical solution. We could find no studies in which cultivars or selections of Jerusalem artichoke were evaluated for resistance to *P. helianthi*, although *Helianthus* spp. have a multiplicity of rust-resistance genes (15). Russian workers (10) have used wild species of *Helianthus*, including *H. tuberosus*, as a gene source for breeding rust resistance into sunflower. In our tests, the population of *P. helianthi* present on Jerusalem artichoke in Georgia produced uredia on one sunflower cultivar tested but not the other. We have not observed rust on commercial plantings of sunflower in Georgia, which indicates that the hybrids being grown have resistance to the race that is prevalent on Jerusalem artichoke. Because interspecific crossing of *Helianthus* is possible (11,15), transfer

of resistance genes to *H. tuberosus* seems to be feasible. Use of host resistance may be complicated by the cross-infectivity of *P. helianthi* on different *Helianthus* spp. and the development of new virulent races by hybridization (15).

S. rolfssii was devastating to Jerusalem artichoke, especially when the crop was planted repeatedly on the same land. The residual tubers from a previous crop apparently serve as a food base for the organism the following year because addition of tubers resulted in increased disease severity. Tuber removal increased speed and uniformity of disease development, probably because our removal operation spread inoculum (sclerotia) uniformly over the plots and sufficient organic matter (stalk sections) was present as a food base (1). We do not consider any of the chemical treatments evaluated to be economically feasible. Based on control studies with other crops (1), cultural practices such as land selection, crop rotation, and deep turning of organic matter may be essential control measures for southern blight control on Jerusalem artichoke.

Tuber rots are of great concern because field storage may be the only practical way to hold tubers for extended periods. Jerusalem artichoke tubers do not have a corky protective layer, and they dry rapidly unless held in the soil or in refrigerated storage. Tubers even partially deteriorated by rot organisms have low alcohol yields compared with healthy tubers (S. J. Kays, *unpublished*).

The organisms associated with tuber rots in storage and in the field in Georgia are quite different from those reported previously (4). Johnson (4) found *Botrytis cinerea*, *Rhizopus nigricans*, and occasionally, *Fusarium* and *Penicillium* spp. were most frequently associated with tubers stored under refrigeration in Minnesota. *Sclerotinia sclerotiorum* (not isolated by Johnson) and *R. nigricans* were the only organisms capable of causing tuber rots near 0 C. *P. fluorescens* was also commonly isolated by Johnson (4) but did not rot tubers in inoculation tests. *Fusarium* spp. were isolated most commonly from tubers held in the field and in refrigerated storage in Georgia, and some isolates caused rot in pathogenicity tests. *P. marginalis* and *P. fluorescens* were frequently associated with decaying tubers held in the field and especially in refrigerated storage. Some isolates of these *Pseudomonas* spp. grew and caused slow rot development at 5 C, which indicated they may continue to cause deterioration at temperatures too low for growth of the *Fusarium* spp. We suspect that the *Fusarium* and *Pseudomonas* spp. interact in causing tuber deterioration, but additional work is needed to confirm this. There is no apparent practical control measure for the tuber rots except to avoid soil types where the rots appear to be most severe.

We could find no reports on differences in tuber rot susceptibility among cultivars and selections of Jerusalem artichoke.

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