Seed Rot and Damping-off of *Chenopodium quinoa* Caused by *Sclerotium rolfsii*

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ABSTRACT

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Sclerotium rolfsii was isolated from damped-off seedlings of *Chenopodium quinoa* in southern California. Susceptibility of *C. quinoa* to *S. rolfsii* was demonstrated in vitro and in the greenhouse. Soil organic matter appears to be important in disease development only when *S. rolfsii* sclerotia become aged and dry but not when inoculum consists of young undried sclerotia. This is the first report of this disease.

Chenopodium quinoa Wild., commonly referred to as quinoa, is a high amino acid and protein-yielding crop grown as a staple food in western South America. The completeness of quinoa protein, highly complemented in lysine and methionine, is associated with increased test weights and maturation rates of livestock, compared with other plant protein sources (2). Its attributes as food or feed, hardiness, low water requirement, and ease of cultivation promoted a crop improvement project at California Polytechnic State University, San Luis Obispo. Preemergent and postemergent damping-off of seedlings occurred in a fall cultivar trial. Diseased seedlings exhibiting stem girdling and collapse appeared in patchy areas in the field. *Sclerotium rolfsii* Sacc. was consistently isolated from affected seedlings. *C. quinoa* had not been previously reported as a host for *S. rolfsii*. Koch's postulates were then performed on three cultivars with different saponin contents.

Saponin content in the seed coat of quinoa is historically important in cultivar selection, because saponins naturally deter insects and birds. Saponins have a membranolytic action that results in reduced growth of some fungi (3). It was therefore important to determine if saponins in these seeds would contribute in resistance to *S. rolfsii*. The importance of an organic base for the establishment of pathogenicity by *S. rolfsii* has been stressed (1), but under some conditions, *S. rolfsii* grown from sclerotia can establish parasitism in the absence of organic matter (R. A. Grogan, *personal communication*).

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This paper describes four experiments with *C. quinoa*, which attempt to establish the role of *S. rolfsii* in damping-off, determine if saponins in *C. quinoa* seed coats impart resistance to *S. rolfsii*, and determine the role of organic matter in the virulence of *S. rolfsii* as a pathogen.

METHODS AND RESULTS

Isolation of fungus. Diseased *C. quinoa* seedlings with stem lesions just above the ground level were collected, sectioned, and surface-treated in 0.6% sodium hypochlorite for 2–3 min. The sections were then placed on potato-dextrose agar (PDA, Difco) or water agar and incubated at 25 C for 24 hr. Hyphal tips from the resulting cultures were transferred to nutrient agar or PDA augmented with a bacterial inhibitor (0.0033% rose bengal). After 4–7 days of growth at 20 C, second hyphal tip transfers were made to culture tubes containing PDA.

Only one fungus, *S. rolfsii*, was isolated from all diseased seedlings collected from field plots. The fungus was grown on PDA and vegetable agar (200 ml of V-8 juice, 2% agar, clarified with 3% $CaCO_3$), and characteristically white sterile mycelium was produced, followed by abundant small spherical sclerotia typical of the fungus.

In vitro pathogenicity. Tests evaluating pathogenicity of *S. rolfsii* to seeds and seedlings were made in vitro on PDA, vegetable agar, and water agar. Seeds of several cultivars of *C. quinoa* were obtained from the Universidad

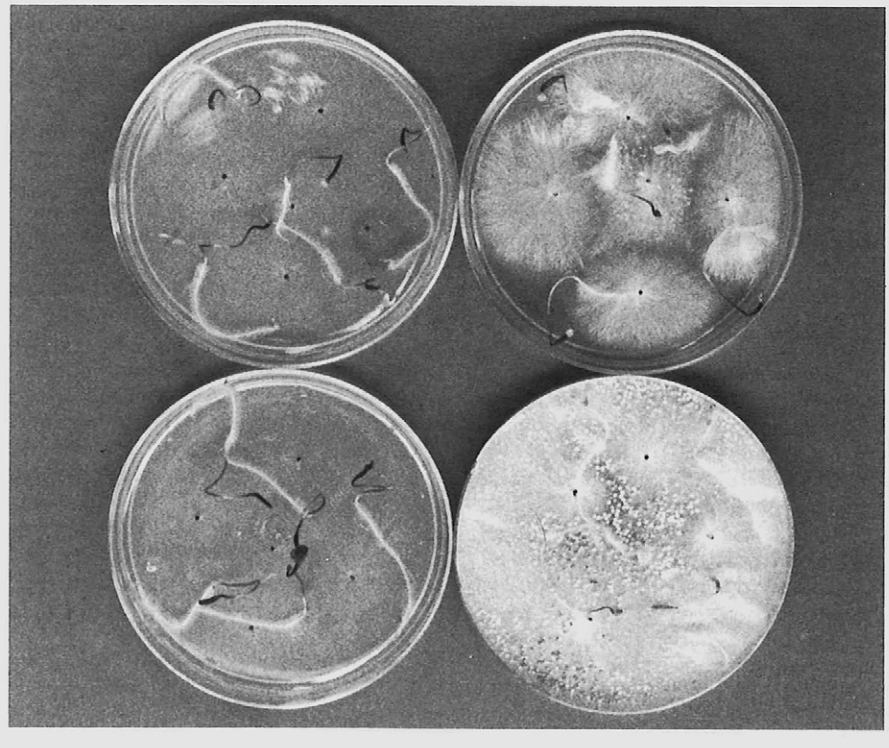

Fig. 1. *Chenopodium quinoa* seeds and seedlings growing on various agars inoculated with *Sclerotium rolfsii*. **Left:** (top and bottom) water agar. **Right:** (top) potato-dextrose agar and (bottom) V-8 vegetable juice agar.

Fig. 2. Seedlings of *Chenopodium quinoa* growing in inoculated soil. **(1)** Sand and rolled oats, inoculated with 1-mo-old sclerotia. **(2 and 3)** Sand and rolled oats inoculated with 6-mo-old sclerotia. **(4)** Sand inoculated with 6-mo-old sclerotia. **(5)** Sand inoculated with 1-mo-old sclerotia. **(6)** Sand, uninoculated control.

Superior Polytechnica, Rio Bamba, Equador. Twenty seeds of two cultivars with seed coats differing in saponin content were surface-treated in 0.6% sodium hypocholorite for 2 min before being implanted on the inoculated agar. Seeds placed on dampened filter paper in petri dishes were used to determine germinability.

All seeds and seedlings germinating on PDA or vegetable agar in the presence of the fungus were diseased. Fungus grown on water agar did not successfully parasitize implanted viable seeds (Fig. 1).

Soil tests for pathogenicity. Three experiments were done in soil or growth media in the greenhouse: the first in Cropley clay, a mineral soil; the second in a fir bark and volcanic scoria mixture; and the third in an organic-free washed sand. Rolled oats (0.4:4 fir bark scoria, v/v) were incorporated into all but the control treatments.

In the first attempt to establish pathogenicity in soil, inoculum was grown in cornmeal/sand (3:1) for 12 days at 25 C. Seeds of cultivars 203 (containing no saponins) and 213 (high in saponins) were planted in Cropley clay soil amended with the cornmeal/sand inoculum containing sclerotia at 500, 100, and 0 g per 15-cm pot. The experiment was replicated six times. The plants were allowed to grow 6 mo to maturity. No damping-off occurred. When the roots were examined at harvest, no lesions were found even though sclerotia of *S. rolfsii* were among the rootlets, indicating that the fungus was present in the soil.

In the second experiment a fir bark/volcanic scoria (3:1) planting mix was used instead of soil. Seeds from cultivars 214 (high in saponins), 70 (moderate saponin content), and 202 (no saponins) were used (20 seeds per pot) to determine the effect of saponin contents on host susceptibility. Autoclaved 15-cm pots were filled with the soil treatments, placed on inverted pot saucers, watered, inoculated with 1-mo-old sclerotia, and left to incubate for 7 days before seeding. The experiment was replicated three times.

Observations and data records were made for 14 days until the seedlings reached the first true leaf stage. Isolations were made from preemergent colonized seeds and seedlings and from post-emergent damped-off seedling stems and rootlets.

In the control (fir bark/scoria medium alone), the fungus destroyed 50% of the seeds and seedlings; in the same medium supplemented with rolled oats, damping-off was 100%. *S. rolfsii* was isolated from all diseased seedlings. Saponin content of the seed coat did not influence the susceptibility of emerged seedlings, but seedling emergence was 95% in uninoculated soil with cv. 214 (high saponin content) and 62% with cv. 70 and cv. 202. The proportion of emerged seedlings that were diseased was constant in all cultivars.

In the third experiment, 20 quinoa seeds and 20 sclerotia of *S. rolfsii* were planted in washed river sand alone (free from organic matter) or in washed sand amended with 0.4 g of rolled oats per 10-cm pot. Half the pots were inoculated with 1-mo-old sclerotia freshly harvested from vegetable agar plates and half with sclerotia dried for 6 mo in the laboratory. The experiment was replicated three times. All seedlings in the sand amended with rolled oats were killed by the fungus regardless of sclerotial age. In the sand without oats that was inoculated with 1-mo-old sclerotia, 58% of the seedlings were killed; none were killed in sand inoculated with 6-mo-old sclerotia. No seedlings were lost in the uninoculated controls (Fig. 2).

DISCUSSION

The pathogenicity of *S. rolfsii* to the seeds and seedlings of *C. quinoa* is firmly established by the parasitism of the seeds in the agar plate and greenhouse soil experiments. This is the first report of this pathogen on quinoa.

Saponin content of the seed coat did not influence the development of disease in seedlings in these experiments, and saponin content would probably not be a factor in disease resistance in the field.

In the first greenhouse experiment using clay soil low in organic matter, no disease occurred. These results support Boyle's (1) results and observations that host survival is high in highly infested soil that is low in a saprophytic food source for the fungus.

Results from the second greenhouse experiment indicate that fir bark and scoria with or without rolled oats is a more suitable medium for promoting pathogenicity of *S. rolfsii* to quinoa. Addition of rolled oats increased pathogenicity 50%.

In the washed sand experiment, no seedlings were lost to disease in the unamended medium when dried 6-mo-old sclerotia were the inoculum, but 58% of the seedlings were killed (this in the absence of an organic food supply for the fungus) when fresh 1-mo-old sclerotia were used. These results indicate that sclerotia that have a chance to dry and age need an organic base to initiate parasitism, but sclerotia that remain damp and possibly nondormant do not.

LITERATURE CITED

1. BOYLE, L. W. 1961. The ecology of *Sclerotium rolfsii* with emphasis on the role of saprophytic media. Phytopathology 51:117-119.
2. MAHONEY, A. W., and L. HENDRIX. 1975. An evaluation of the protein quality of quinoa. J. Agric. Food Chem. 23(2):190-193.
3. SEGAL, R., and E. SCHLOSSER. 1975. Role of glycosidases in the membranlytic, antifungal action of saponins. Arch. Microbiol. 104(2):147-150.