

# *Synchytrium endobioticum* (Schilb.) Percival

## Pest Risk Assessment for Oregon

This pest risk assessment follows the format used by the Exotic Forest Pest Information System for North America. For a description of the evaluation process used, see <http://spfnic.fs.fed.us/exfor/download.cfm>.

### IDENTITY

**Name:** *Synchytrium endobioticum* (Schilb.) Percival

**Taxonomic Position:** Chytridiales: Synchytriaceae

**Common Name:** Potato wart disease

### RISK RATING SUMMARY

**Numerical Score:** 6

**Relative Risk Rating:** HIGH

**Uncertainty:** Very Certain

**Uncertainty in this assessment results from:** Potato wart has been extensively studied in the countries in which it is established.

### RISK RATING DETAILS

#### Establishment potential is HIGH

**Justification:** Potato wart is apparently native to the Andes Mountains and has subsequently been spread throughout the world through the movement of infected or contaminated tubers. It has become successfully established in several countries in Europe, Asia, Africa, North America, South America, and Oceania. Previous detections in Maryland, Pennsylvania, and West Virginia had reportedly been eradicated by 1974, although surveys conducted in Maryland revealed the presence of resting spores of the pathogen were still present in one home garden. The spores were reportedly non-viable.

#### Spread potential is MODERATE

**Justification:** Potato wart has been spread throughout the world through the movement of infested tubers. Local spread is primarily through the movement of contaminated soil on equipment, vehicle tires, tubers, and plants. Spores may also be spread by wind. Symptoms in the field may not manifest until after repeated cultivation of susceptible hosts within a field or garden. Infected tubers may not manifest symptoms until in storage; however, meristematic tissue (sprouts) may be so severely affected plants will not emerge from infected seed tubers.

#### Economic impact potential is HIGH

**Justification:** Oregon is ranked 7<sup>th</sup> amongst all states for commercial potato production and 6<sup>th</sup> amongst all Oregon agricultural commodities, generating over \$164 million in value in 2008. Currently, this disease is not known to occur in the United States or has been eradicated from states in which it has been reported. Quarantines have been in place throughout the world for more than 65-years in an effort to contain the disease. Within

severely infested fields, crops losses may be 100%. Infected tubers develop galls (warts) that reduce their market value and are prone to desiccation or decay by other, secondary organisms (Figure 1). The disease is controlled primarily through the use of resistant cultivars, although potato wart has demonstrated the ability to develop new pathotypes that can overcome this resistance. Applications of chitinous material to infested fields have shown some ability to suppress disease.



**Figure 1.** Potato tubers infected with *Synchytrium endobioticum* (photo courtesy of Central Services Laboratory, United Kingdom).

### **Environmental impact potential is MODERATE**

**Justification:** Potato wart has demonstrated the ability to develop new pathotypes that can overcome the resistance bred into commercial potato cultivars.

### **HOSTS**

Major: *Solanum tuberosum*

Wild: *Solanum* (nightshade)

Experimental: Can also infect under artificial conditions *Petunia*, *Nicotiana*, *Lycopersicon esculentum*, *Physalis*, and *Capsicastrum*. Roots are infected but galls do not form.

### **GEOGRAPHIC DISTRIBUTION**

**Africa:** Algeria, South Africa, and Tunisia

**Asia:** Armenia, Bhutan, India, Nepal, and Turkey

**Europe:** Austria, Belarus, Belgium, Czech Republic, Faroe Islands, Finland, Germany, Ireland, Italy, Latvia, Luxembourg, Netherlands, Norway, Poland, Romania, Russian

Federation, Serbia and Montenegro, Slovakia, Slovenia, Sweden, Switzerland, Ukraine, and United Kingdom

**North America:** Canada (Newfoundland, Prince Edward Island) and Mexico (unconfirmed on wild *Solanum* spp.)

**Oceania:** New Zealand

**South America:** Bolivia, Ecuador, Falkland Islands, and Peru

## **BIOLOGY**

*Synchytrium endobioticum* produces resting or “winter” sporangia that germinate to release motile zoospores. The zoospores migrate in soil water to infect epidermal cells of meristematic tissues of young potato plants. The zoospores are short-lived and must encyst and infect susceptible host tissue within 1- to 2-hr of their formation. After infection, the zoospores form sori within infected cells. Each sorus contains one to nine “summer” sporangia, which germinate to give rise to more zoospores. These zoospores continue the disease cycle. Eventually, some of the zoospores conjugate to form motile zygotes that then infect the host to form winter sporangia again. After infection by zoospores or zygotes, infected and surrounding cells become enlarged or undergo rapid division. These cellular changes increase susceptible host tissue and thus infection courts for the fungus. Young galls develop rapidly (e.g., >1,800 fold in 16-days) at the expense of other plant tissues. Winter sporangia contaminate the soil as galls decay.

Inoculum is spread primarily through the movement of contaminated soil adhering to tubers, equipment, and other carriers. Winter sporangia survive passing through the stomachs of animals and thus can be spread through animal manure. Winter sporangia can also be spread by wind. Winter sporangia remain viable for 30- or more years in soil and can be found at depths of up to 50 cm. Under optimal conditions, the disease can develop from less than one winter sporangium per gram of soil. Motile zoospores can migrate  $\leq 50$  mm and thus rarely contribute to field-to-field spread.

Water is required for germination of winter and summer sporangia. The following environmental conditions favor disease development: cool summers with average temperatures  $<18^{\circ}\text{C}$ , winters of 160-days at  $\leq 5^{\circ}\text{C}$ , and annual precipitation of  $\geq 70$  cm. The disease rarely occurs outside of areas with these environmental conditions. Temperatures of  $12\text{-}24^{\circ}\text{C}$  favor infection.

Potato wart can serve as a vector for *Potato Virus X*.

## **PEST SIGNIFICANCE**

**Economic impact:** *S. endobioticum* has been reported as causing 100% yield loss in badly infested fields. Loss in storage also occurs because of tuber galls forming after harvest. Once a field becomes infested, reproductive structures of the pathogen can remain viable for 30- or more years. Control is provided primarily by the planting of resistant cultivars, although new pathotypes have developed that can overcome this resistance. The application of chitinous materials to fields can suppress the disease, providing growers with one other method of control. The adoption of quarantine

regulations in response to positive detections of potato wart can also contribute to economic losses for growers.

At present, potato wart does not occur in Oregon. In 2008, Oregon ranked 7<sup>th</sup> in the nation for potato production, generating more than \$208 million in gross sales receipts.

**Environmental impact:** Potato wart has been reported infecting wild *Solanum* species in Mexico. It has been reported infecting potatoes grown in home gardens in Canada and can spread from those gardens elsewhere through movement of contaminated soil.

**Control:** Worldwide, countries control disease spread and exclude the pathogen through the use of quarantine legislation. Resistant cultivars have been developed in Europe and North America. Although resistant plants become infected, symptom development is suppressed and galls remain superficial. Some resistant cultivars have a hypersensitive reaction that kills infecting zoospores. However, new pathotypes have been identified that may infect formerly resistant cultivars. Experiments in Canada identified crushed crab shell (23% chitin) as an effective means of suppressing the disease. No other chemical control is available.

#### **DETECTION AND IDENTIFICATION**

**Symptoms:** Aerial symptoms are not readily apparent, although decreased plant vigor may occur. Occasionally, galls will form on upper stem, leaf, or flower tissue. Symptoms on belowground plant parts are more readily apparent. Infected tissues include stem bases, stolon tips, and tuber eyes. Galls vary in shape, but are mostly spherical outgrowths 1- to 8-cm in diameter. Aboveground galls are typically green to brown, turning black at maturity and then decaying. Belowground galls are white to brown, turning black as they decay. Galls may not be evident until harvest. Those present at harvest may become desiccated or may decay. Tubers may be disfigured or completely replaced by galls. Galls may also form on tubers after harvest. Potato wart does not kill its host, although seed tubers may be so disfigured that no plants emerge.

**Morphology:** Potato wart is a long-cycled chytrid, producing summer sporangia and winter (or resting) sporangia. Both sporangia germinate to form zoospores; zoospores are pear-shaped, 1.5-2.2  $\mu\text{m}$  diameter, with a posterior flagellum. Resting sporangia are mostly golden-brown, spherical, thick-walled, and about 35-80  $\mu\text{m}$  diameter.

#### **Testing methods for identification:**

Within soil, potato wart can be identified through a variety of methods, including wet-sieving and dry-sieving. Recently, PCR-based detection methods have been employed for the detection of *S. endobioticum* in soil extracts and *in planta*.

#### **MEANS OF MOVEMENT AND DISPERSAL**

Inoculum is spread primarily through the movement of contaminated soil adhering to tubers, plants, equipment, and other carriers. Winter sporangia survive passing through the stomachs of animals and thus can be spread through animal manure. Winter sporangia can also be spread by wind. Motile zoospores can migrate  $\leq 50$  mm and thus rarely contribute to field-to-field spread.

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