Synchytrium endobioticum

Scientific Name
Synchytrium endobioticum (Schilbersky) Percival

Synonyms:
Chrysophlyctis endobiotica Schilbersky
Synchytrium solani Massee

Common Name
Potato wart, potato wart disease, wart disease of potato, black wart of potato, cauliflower disease, potato tumor, potato cancer, potato canker, warty disease

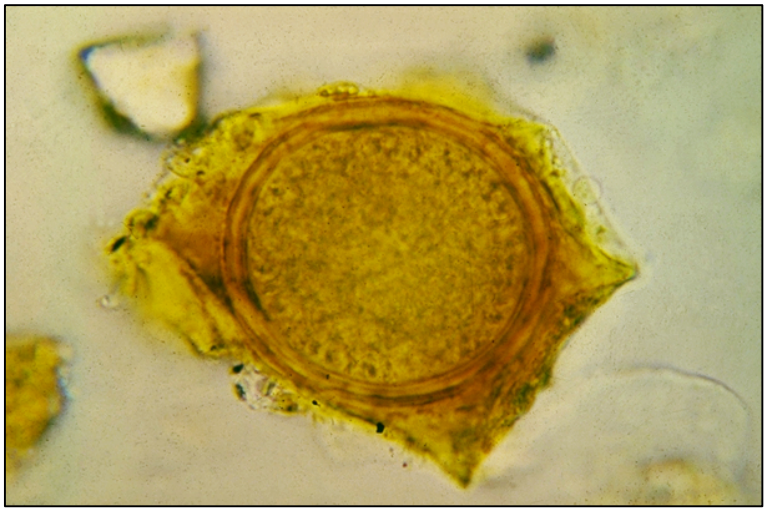


Figure 1. Live resting (winter) sporangium *of Synchytrium endobioticum*. Image courtesy of Central Science Laboratory, York (GB) British Crown.

Type of Pest
Fungal pathogen

Taxonomic Position
Kingdom: Fungi, **Class:** Chytridiomycetes, **Order:** Chytridiales, **Family:** Synchytriaceae

Reason for Inclusion in Manual
Previous CAPS Target: AHP Prioritized Pest List - 2005 through 2009
Additional Pest of Concern List (2010 to 2013); Solanaceous Hosts survey; Select Agent

Pest Description
A pathotype is a subdivision of a pathogen species characterized by its pattern of virulence or avirulence to a series of differential host varieties or cultivars. Ballvora et al. (2011) state there are 38 pathotypes of *Synchytrium endobioticum* occurring in Europe alone. Franc (2007), in contrast, states that there are approximately 43 pathotypes described from Europe, but that many presumably persist in small garden potato plots, not in commercial potato plots. The true number of pathotypes is unknown as researchers from different countries have used different sets of cultivars to identify and characterize pathotypes (Franc, 2007). Ballvora et al. (2011) state that pathotypes 1, 2, 6, and 18 are the most important occurring in Europe.

Hyphae: This species does not produce hyphae (EPPO, n.d.).

Sporangia: *Synchytrium endobioticum* produces sporangia, which contain 200 to 300 mobile zoospores (EPPO, n.d.; Franc, 2007).

There are two different sporangia, the winter sporangia (long-lived stage) and the summer sporangia (short-lived, quickly reproducing stage) (EPPO, n.d.).

> Winter sporangia (also called resting sporangia): "Winter sporangia (Fig. 1) are mostly spherical, thick-walled, about 50 µm in diameter (25-75 µm); tend to be integral components of small aggregates or crumbs of soil, 0.1-2.0 mm in diameter" (EPPO, n.d.; 1982). These can be irregular in shape (spherical to ovoid) with brown walls (Cakir, 2000). "As the host galls decay the host cell wall disintegrates, slightly changing the appearance of the outer surface of the sporangia" leaving it with a characteristic angular appearance (Byrne, 2008).
>
> Summer sporangia: "Summer sporangia are thin-walled and transparent. Zoospores may be visible within the summer sporangia" (Byrne, 2008).

Zoospores: "The swimming zoospore of S. *endobioticum* is approximately 3 µm in diameter, spherical to elongate in shape, and normally has a single lipid body protruding anteriorly or at one side. The zoospore has a single whiplash flagellum that is about 17 µm long with a whiplash portion that is ca. 2.5 µm in length as measured from negatively stained EM preparations. In the light microscope, the lipid body is the only structural detail which may be observed. The zoospores encyst readily in water; even in the absence of the host…the ribosomes are evenly dispersed in the cytoplasm" (Lange and Olson, 1977).

Zoospores swell into prosori and then develop into sori (AU-DAFF, 2011), which are groupings of sporangia.

Prosori: "Oval, aseptate, smooth, thick walled, light golden brown, 40-50 µm in diameter, and usually lie at the bottom of the infected plant cell. There may be up to 4 prosori per infected plant cell" (reviewed in AU-DAFF, 2011). Curtis (1921) states that the prosorus "is applied to all stages between the completion of the rounding off of the zoospores…and the beginning of segmentation into sporangia."

Sori: "Contents escape the prosorus through the prosorus outer wall to form an ovoid, flattened or spherical haploid sorus of sporangia with each sorus containing 1-9 hyaline, thin-walled summer sporangia, which quickly release zoospores to initiate new infection sites" (reviewed in AU-DAFF, 2011).

An in depth description of the prosori and sori can be found in Curtis (1921).

Biology and Ecology

This pathogen is an obligate parasite, only feeding on the living tissues of the host plant. Once temperatures rise above 8°C (46.4°F) in the spring and moisture levels are

sufficient (Fig. 2), the winter sporangia found in decaying warts germinate, releasing 200 to 300 mobile zoospores (EPPO, n.d.; CFIA, 2012). The zoospores move through water in the soil using their flagellum until they find a suitable host (EPPO, n.d.). Zoospores penetrate the epidermal cells of meristematic tissues (specifically growing points, buds, stolon tips, and young leaf primordial of the tuber and lower stem). Both invaded and surrounding cells enlarge. After infection, rapid cell division causes an increase in meristematic tissue, providing additional infection courts (AU-DAFF, 2011).

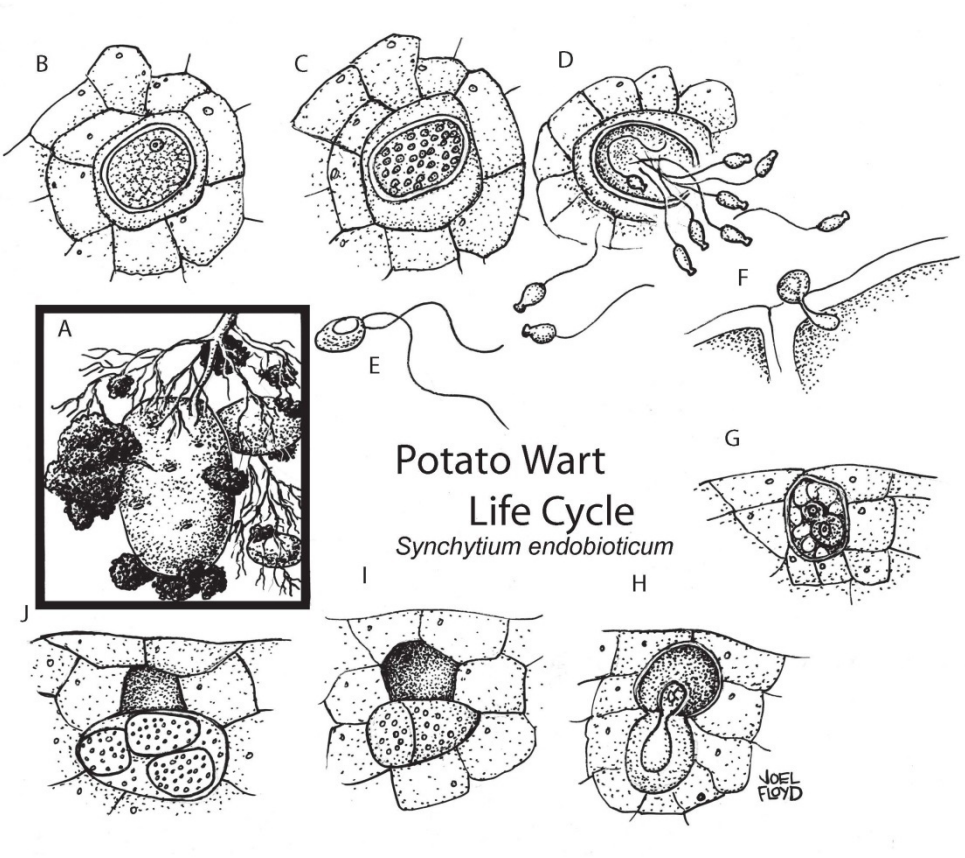


Figure 2. Life cycle diagram of *Synchytrium endobioticum*. **A:** infected tubers, stem and stolons with wart symptoms; **B:** resting sporangium; **C:** resting sporangium with maturing zoospores; **D:** discharged motile zoospores; **E:** two zoospores form a (diploid) sporangium to later form a zygote; **F:** zoospore entering a host cell by direct penetration; **G:** young prosorus in host cell; **H:** contents of prosorus passing into host cell; **I:** cross section of sorus with two (haploid) sporangia and remains of empty prosorus; and **J:** three mature (haploid) sporangia in sorus with zoospores beginning to be released. The haploid sporangia are summer sporangia and the diploid sporangia are resting winter sporangia (Stevenson et al., 2001; illustrations after Walker, 1957).

Infection is favored when temperatures are between 12 and 24°C (54 to 75°F) (Franc, 2007). Zoospores can infect host material quickly, approximately two hours after formation (AU-DAFF, 2011). If zoospores do not infect susceptible host material within

this time, they will not survive (Franc, 2007). Once the zoospores penetrate a host, they lose their flagellum and greatly enlarge to form the summer sporangium (EPPO, n.d.). This is a short lived and quickly reproducing stage. The summer sporangium rapidly releases large numbers of zoospores to re-infect surrounding cells. As long as conditions are favorable, the zoospores will continue to produce summer sporangia (EPPO, n.d.)

When conditions become stressed (*e.g.*, water become scarce), zoospores can fuse together forming a zygote. At this point, the host cell containing the zygote does not swell, but divides, forming an outer layer to the winter sporangia (EPPO, 1982). The winter sporangia can remain viable for up to 30 years (Abdullahi et al. (2005) reports that spores can be viable for up to 70 years) and can survive depths of up to 50 cm (approx. 19 $^{11}/_{16}$ in) in soil (APHIS, 2012). The longevity of the resting spore could be due in part to its chitin-protein complex and similarity of the cell wall to an insect cuticle. This can help render the sporangium resistant to the physical and chemical makeup of the soil environment (Arya et al., 1981).

Work has been completed to determine which potato varieties are resistant to the different pathotypes (Dimitrova et al., 2011).

Figure 3. Newly harvested, warted potato tubers of cv. Duke of York; note that some warts are already starting to rot. Image courtesy of Central Science Laboratory, York (GB) British Crown.

Symptoms and Signs

This disease does not usually present symptoms aboveground; however stems, leaves, and flowers can sometimes develop galls (Byrne, 2008; EPPO, 1982; EPPO, 2007). Attacked plants may show reduced vigor. Small greenish warts may form in the place of aerial buds at stem bases (EPPO, n.d.; EPPO, 1982).

This fungus affects the stolons (underground stems) and tubers (APHIS, 2012; EPPO, n.d.) targeting the meristematic tissue (AU-DAFF, 2011). Young potato warts are white, soft, and pulpy in texture (APHIS, 2012); the surface is rough and corrugated (AU-DAFF, 2011). The warts occur as infected plant cells swell, divide, and surround the zoospores (APHIS, 2012).

Figure 4. Warts formed on the tubers in the soil may surface during the growing season. Image courtesy of EPPO Gallery.

Warts (Fig. 3) can vary in size, from small and mild to large and severe (usually 1 to 8 cm; approx. $^{3}/_{8}$ to 3 $^{1}/_{8}$ in); they can also vary in color (Hampson, 1993; Cakir, 2000; Byrne, 2008). Warts are initially whitish (green if exposed to light), but darken gradually (EPPO, n.d.; EPPO, 1982; AU-DAFF, 2011). Sometimes developing warts can become exposed at or above the soil line (Fig. 4, 5) (CFIA, 2012).

The warts become a sink for nutrients and rapidly increase in size. Potatoes can sometimes be completely covered by the masses (Cakir, 2000). When harvested, warts may be extremely small and overlooked; however, they can continue to develop in storage (DEFRA, n.d.). Warts may be similar in color to the tuber when they develop in storage (Johnson, 2000). When plants are harvested, the gall can either dry up or reduce to a brownish-black mass that rots (Hampson, 1993).

The disease does not kill the plant; however sprouts can be damaged which can limit

Figure 5. A heavily infected plant showing yellowish warts on subsoil tubers and greenish warts at soil level. Image courtesy of HLB B.V., Wijster (NL).

emergence from seed tubers (Byrne, 2008). Early infection of young tubers leads to distorted and spongy tubers, while older tubers are only infected on the eyes. Warts eventually rot and disintegrate (EPPO, n.d.; EPPO, 1982). As zoospores infest cells, they will begin to swell while tissues multiply. This will lead to a characteristic cauliflower appearance (EPPO, n.d.).

This fungus does not affect the roots (EPPO, n.d.).

In partially resistant cultivars, the warts are superficial and scab-like. In highly resistant cultivars, "the zoospore dies soon after invasion by necrotic abortion (hypersensitive reaction) of the infected tissue" (reviewed in AU-DAFF, 2011).

Pest Importance

This species was once considered the most serious disease of potato but has been controlled fairly well through phytosanitary measures and resistant potato varieties. This species is still considered an important pest of potato due to the long lasting resting sporangia. Also, new strains of the fungus are capable of attacking previously resistant potato varieties (DEFRA, n.d.).

Synchytrium endobioticum is still of great economic importance in Poland, Romania, Russia, and Switzerland and of some economic importance to most other EPPO countries (EPPO, 1982). The severity of outbreaks is dependent on soil conditions as tubers develop and the variety of potato (DEFRA, n.d.) as well as pathovar and level of infection (Wale et al., 2011). In badly infested soils, it is possible to have 100% loss of potential tuber yield (AU-DAFF, 2011); losses may also occur in storage if galls develop after harvest (Hampson, 1993). The detection of potato wart on Prince Edward Island during the 2000 growing season resulted in an estimated $30 million loss to the island's economy in the first year (Franc, 2007).

Only a few sporangia are needed for infection to occur. Infection has been reported at levels below 1 sporangium or even $^{1}/_{25}$ sporangium per gram of soil (reviewed in Baayen et al., 2005). This disease can have a negative effect on any scale of potato production from small gardens and subsistence farming to commercial farming for consumption or production of seed potatoes (Franc, 2007). Potato wart is most severe in cool, wet mountainous regions.

This disease has been regulated through quarantines and domestic legislation for the past several decades to prevent its spread. The stringent quarantine and sanitation measures put into place for *S. endobioticum* have helped contain the disease and minimize losses (EPPO, 1982). Although the fungus is found throughout most potato growing countries, it is absent from a majority of host fields due to these phytosanitary measures (EPPO, 2007).

One such measure is the 'Wart Control Directive' by the European Union which "prohibits cultivation of potatoes on infected plots and cultivation of susceptible potato cultivars, or any plants for planting, in the adjacent buffer zone (EU, 1969)" (Baayen et

al., 2005). If introduced into a field, the entire crop may be deemed unmarketable (although it does not make the tuber inedible). Once infection occurs, infected plants are destroyed and infested fields are banned from having potatoes planted in them for at least 20 years (EPPO, 1982; Ballvora et al., 2011). Safety zone can only be planted with cultivars that are resistant to the detected pathotype (Ballvora et al., 2011). Plots can only be released after a minimum of 20 years has passed since the last infection. Plots have to be sampled, tested, and found free of any viable sporangia or any evidence of infection (EPPO, 2003b).

According to the United States Potato Board, total U.S. potato and potato product exports reached record levels in fiscal year 2013. The value of these exports is estimated at $1.6 billion and consists of 17% of U.S. potato production (USPB, 2013).

S. endobioticum is listed as a harmful organism in 123 countries on six different continents (USDA-PCIT, 2013). There may be trade implications with these countries if this fungus becomes established in the United States.

Chemical soil treatments are not considered a viable eradicative option for sporangia found in infested fields (Byrne, 2008).

Known Hosts

The only known cultivated host species is potato (*Solanum tuberosum*); however wild *Solanum* spp. have been found in Mexico (EPPO, n.d.). Potato is the only plant believed to be important in the disease cycle (Franc, 2001).

Other *Solanum* that can be infected include: *Solanum chacoense*, *S. commersonii* (Commerson's nightshade), *S. curtilobum* (rucki), *S. demissum* (nightshade), *S. orchranthum*, *S. pimpinellifolium* (currant tomato), *S. pseudocarpsicum*, *S. sisymbriifolium* (sticky nightshade), and *S. stoloniferum* (Fendler's nightshade) (USDA, 1990; reviewed in Farr and Rossman, 2005).

Artificially inoculated hosts

Other solanaceous plants can be artificially inoculated, including tomato (*Solanum lycopersicum)* (Hampson, 1979; Hampson and Haard, 1980) but the pathogen does not induce gall formation in these species (Abdullahi et al., 2005).

The following genera have been artificially inoculated: *Capsicastrum, Datura, Duboisia, Hyoscyamus, Lycium, Nicandra, Nicotiana, Physalis,* and *Schizanthus* (reviewed in Hampson, 1993).

Pathogen or Associated Organisms Vectored

This pathogen was previously reported as a vector for potato virus X (Franc, 2001). However, this is now considered unlikely (Wale et al., 2011). The warts can serve as an entryway for decay organisms.

Known Distribution

This species most likely originated from the Andean zone of South America. It was introduced into Europe in the 1880s. Statutory measures have been put into place to limit its spread (EPPO, n.d.). Due to these measures, DEFRA (n.d.) states that this species is found only locally throughout most of the European countries where it is currently found.

Synchytrium endobioticum has many different pathotypes that are defined by their virulence on different potato cultivars. Pathotype 1 (European race 1) is one of the most common and is found through the EPPO region (CABI, 2013).

Africa: Algeria, South Africa, and Tunisia; **Asia:** Armenia, Bhutan, India, Nepal, Turkey, and Russia; **Europe:** Belarus, Bulgaria, Czech Republic, Denmark, Faroe Islands, Finland, Germany, Georgia, Ireland, Italy, Latvia, Luxembourg, Montenegro, the Netherlands, Norway, Poland, Romania, Russia, Slovakia, Slovenia, Sweden, Switzerland, Ukraine, and the United Kingdom; **North America:** Canada (Newfoundland and Prince Edward Island) **Oceania:** New Zealand; **South America:** Bolivia, Chile, Ecuador, Falkland Islands, and Peru (EPPO, n.d.; Cakir, 2000; De Boer, 2001; Basim et al., 2005; Dimitrova et al., 2011; CABI 2012; CFIA, 2012; Gorgiladze et al., 2014; IPPC, 2014).

Many countries have unconfirmed records, including: Belgium, China, Ecuador, Egypt, Hungary, Iran, Korea, Lebanon, Mexico, Uruguay, and Zimbabwe (EPPO, n.d.).

S. endobioticum is reported as an eradicated pest in Austria, Chile, France, Lithuania, Portugal, and the United States (EPPO, 2013; EPPO, 2014). It was considered eradicated in Denmark until it was detected there again in September, 2014 (IPPC, 2014).

Pathway

This species moves almost exclusively through human-mediated movement; the capacity for natural spread via zoospores is very limited, about 50 mm (approx. 1.96 in) or less from the host plant each cycle (CABI, 2013). Spread throughout Newfoundland, Canada has been linked to sea, road, and rail transportation routes (Hampson, 1993).

Spread can occur through infected tubers, soil, machinery and other implements used in infected fields, as well as footwear and manure from animals that have fed on infested material (APHIS, 2012). This species has also been recovered on vehicles moving between infested and uninfested areas (Hampson et al., 1996; Hampson and Wood, 1997; Jennings et al., 1997). Spread may also occur through wind or irrigation (reviewed in Hampson and Coombes, 1989; Hampson, 1996). Soil fauna could potentially play a role in limited spread; Hampson and Coombes (1989) found that earthworms could facilitate small scale dissemination of the disease. Composting of infected material is ineffective (Steinmöller et al., 2012) and could potentially lead to further spread of the pathogen if used.

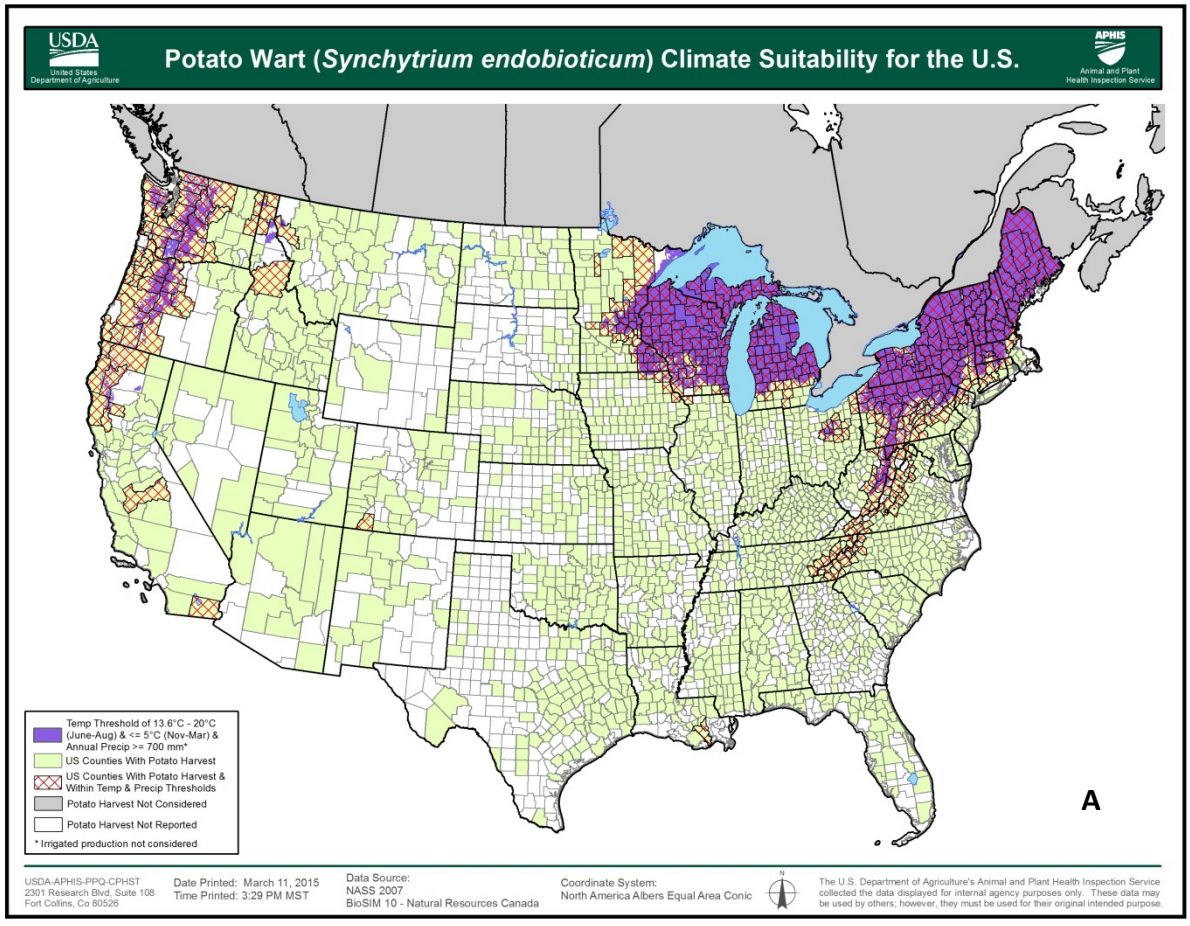
In international trade, this species may move on infected potato tubers or in soil from areas where the fungus is known to occur (EPPO, n.d.). It is thought that this species was first introduced into Europe on breeding material from the Andes after the potato blight event from 1840 to 1850. It continued to spread throughout potato-growing countries in Europe until phytosanitary measures were put into place, which helped prevent its further spread (DEFRA, n.d.).

Import of *Solanum* spp. propagules is currently prohibited from all countries under federal regulation 7 CFR 319.37-2a pending a pest risk analysis (USDA, 2013).

There have been no interceptions of *S. endobioticum* at U.S. ports of entry since January, 2003. However, there have been interceptions of *Solanum tuberosum* plant material intended for propagation from the following host countries: Brazil (1), Canada (12), Germany (1), Peru (1), and Ukraine (12) (AQAS, 2013).

Potential Distribution within the United States

This species has previously been found in the United States in Maryland, Pennsylvania, and West Virginia (Putnam and Hampson, 1989), but these populations are now considered eradicated (CABI, 2013).



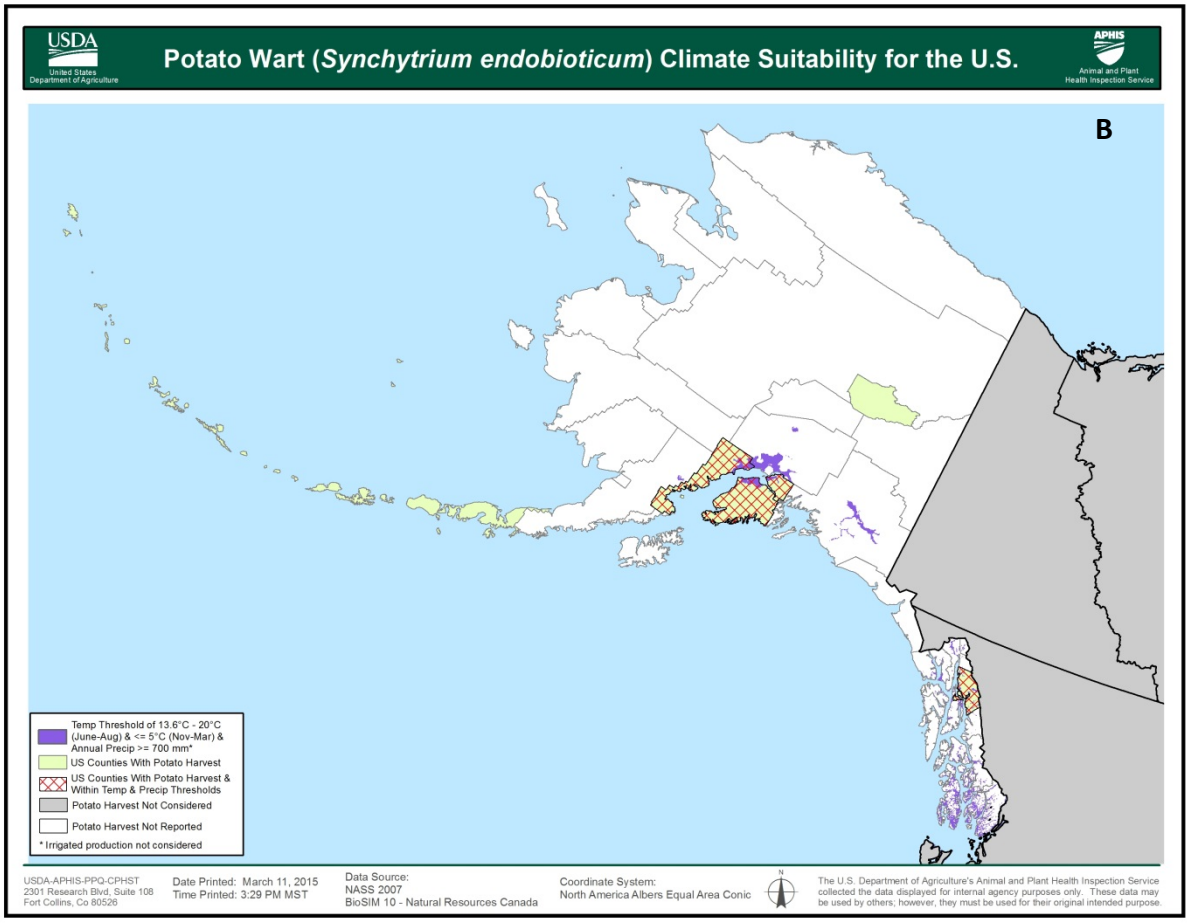


Figure 6: Climatic suitability for potato wart in the United State based on literature thresholds and current climate matching. A. Continental United States. B. Alaska. Courtesy of USD-APHIS-PPQ-CPHST (Lisa Kennaway).

S. endobioticum does better in cooler climates. It is not likely to become a serious problem in areas with warm, light, and well-drained soils (EPPO, 1982). Bojňanský (1960) states that the pathogen prefers areas with low summer temperatures (<18°C; 64.4°F), long, deep winters (≥ 160 days with temperatures below 5°C; 41°F), heavy precipitation (≥ 700 to 800 mm (27.5 to 31.5 in) per year, mostly during the summer), sufficiently aerated and cultivated soils, acidic soils, and areas where poor crop rotation is practiced (potatoes planted every 2 to 3 years).

A map developed by USDA-APHIS-PPQ-CPHST (2014) shows the geographical area in the continental United States and Alaska with both suitable hosts and appropriate climate conditions (combination of temperature and precipitation) for this pathogen (Fig. 6). USDA-APHIS-PPQ-CPHST allowed for a wide temperature range based on the scientific literature about the temperature requirements for infection to occur and disease to develop and climate matching with areas currently known to have the pathogen established. Counties outlined in red have areas with suitable environmental

conditions (shown in purple) at some point during the growing season and have potato production. Even if the areas of purple shading in a red outlined county are not visible, there are areas (though minimal) in that county with suitable temperature and moisture conditions at some point in the growing season. The light green counties indicate counties with potato harvest. Based on these maps, the states at risk for establishment of *S. endobioticum* include: Alaska, California, Colorado, Idaho, Iowa, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Montana, New Hampshire, New Jersey, New York, North Carolina, Ohio, Oregon, Pennsylvania, Rhode Island, Tennessee, Vermont, Virginia, West Virginia, Washington, and Wisconsin.

Survey

CAPS-Approved Method*: Visual survey of potato tubers for symptoms at harvest.

This survey should only be considered in the states with appropriate climate conditions and suitable hosts for this pathogen: Alaska, California, Colorado, Idaho, Iowa, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Montana, New Hampshire, New Jersey, New York, North Carolina, Ohio, Oregon, Pennsylvania, Rhode Island, Tennessee, Vermont, Virginia, West Virginia, Washington, and Wisconsin (Fig. 6).

A visual inspection of tubers can be an effective survey methodology due to the characteristic warty, cauliflower-like symptoms present on the tubers. However, since symptoms are not diagnostic, a laboratory confirmation is necessary to confirm the presence of potato wart.

Wart disease can be detected in potato plants from the flowering period to the time of harvest. Inspection at harvest will preclude unnecessary destruction of plants. If a plant is suspected of having wart, a sample of it will be sent to a designated lab (CPHST Beltsville) for confirmation at:

USDA-APHIS-PPQ-CPHST
BARC-East, Bldg. 580
Powder Mill Road
Beltsville, MD 20705-2350
Phone: (301) 504-7100, VOIP: (301) 313-9200
Group E-mail Address: APHIS-PPQCPHSTBeltsvilleSampleDiagnostics@aphis.usda.gov

Samples will be submitted in sturdy, sealed plastic bags and will be double bagged. Samples should be placed in a cooler. The outer bag will be clearly labeled with a sample number. This will correspond to a survey form including the sample number, location of the field or plot, name of owner/operator, and date. In large fields it will be beneficial to record the location within the field where the sample originated in case the field must be resurveyed. Flagging or other markers can be used, but may be moved or removed.

Alert: Disinfest all sampling equipment and personal protective equipment (rubber boots, etc.) after each field. Keep the samples as cool as possible. Contact the laboratory by phone prior to shipping the samples via overnight delivery service.

Survey Site Selection:
"Surveys should be focused on areas where potatoes are grown, either commercially or on a small scale. Survey areas can also occur in certain areas that are likely to harbor the pathogen including: gateways, potato cutting areas, locations of cull or storage piles, low lying areas (samples should not be taken when areas are overly wet), and sites where Potato wart has previously been reported" (NPRG, 2009).

*For the most up-to-date methods for survey and identification, see Approved Methods on the CAPS Resource and Collaboration Site, at http://caps.ceris.purdue.edu/.

Literature-Based Methods:

Soil survey: Soil sampling should only occur for a delimiting survey once a positive find is confirmed in the field. Soil cores should be collected by using a standard ¾-inch (1.9 cm) soil probe (Oakfield model L or equivalent). Each core will consist of a probe to a depth of 8 inches (20 cm). Cores will be collected in a grid pattern using the following guidelines:

Table 1. Delimiting Survey Soil Sampling Guidelines

Field Size	Grid Pattern[1]
< ½ Acre (A)	2 x 2
½ - 1 A	4 x 4
> 1 A	8 x 8

A minimum sample size of 1000 cubic centimeters (cc) of soil (a composite sample of no fewer than 20 probes) will be taken from each field.

Soil will be collected as 5-pound samples in sturdy paper bags. When each sample is collected, the bag will be closed securely and placed in another paper bag (double bagged). The outer bag will be clearly labeled with a sample number. This will correspond to a survey form which will include the sample number, location of the field or plot, name of owner/operator, and date. In large commercial fields it will be beneficial to record the location (GPS coordinates) within the field where the sample originated in case the field must be resurveyed for confirmation. Flagging or other markers can be used, but may be moved or removed inadvertently (NPRG, 2009).

The summer sporangia can be found during the growing season in young warts. The winter sporangia can also be found in plant tissue during the growing season as well as in decomposing warts (Byrne, 2008).

Sporangia can be concentrated in soil samples by using the wet-sieving (Pratt, 1976) or centrifugation method (Wander et al., 2007). The soil extracts are then viewed

microscopically to check for the presence of sporangia (Byrne, 2008). Dry sieving is also used as well as floatation on chloroform (CABI, 2013).

The recommended method by EPPO "consists of wet sieving a soil sample of 100 g with an electronic sieve shaker, drying the sediment on filter paper, and after dissolving the material centrifuging three times with chloroform or $CaCl_2$, collecting on filter paper and resuspending in lactoglycerol for counting under the microscope. It has the disadvantage that organic matter is not removed from the sample, which makes counting difficult (Van Leeuwen et al., 2005)" (Wander et al., 2007).

The Hendrickx centrifuge method involves mixing a 1 L suspension of tap water with at most 200 g soil. The following is added in order to the centrifuge: 1) separation liquid, 2) water, 3) 100 to 500 ml soil suspension, and 4) kaolin suspension. After centrifuging, "supernatant of water and $CaCl_2$ solution containing the sporangia is collected in a small beaker through the hollow shaft of the rotor" (Wander et al., 2007).

A rapid method to detect zoosporangia in soil using inexpensive equipment and non-toxic reagents was developed by Zelya and Melnik (1998).

Soil bio-assay:
"A bioassay crop is used to determine the viability of resting spores or sporangia in the soil. Soil is collected from areas which are suspected of being infested. The soil will then be used to grow susceptible plants under controlled conditions, such as in a greenhouse or growth chamber which is conducive to disease development. The average greenhouse or growth chamber temperature should remain between 18-21ºC (65–70ºF). If the temperature rises above 21ºC (70ºF), development of warts may be inhibited. Up to three successive cropping cycles may be required to rule out the possibility of viable resting spores.

Key Diagnostics/Identification

CAPS-Approved Method*:

Morphological: Plant material with warts is examined for sporangia. Affected tissue should be mounted in water and observed at 100 to 400x magnification under a light microscope.

Because this pathogen is on the Federal Select Agent list, suspected positives tuber samples should be sent for confirmation to the USDA-APHIS-PPQ-CPHST lab in Beltsville, MD under permit. Molecular protocols are available in the CPHST Beltsville laboratory for confirmatory testing.

*For the most up-to-date methods for survey and identification, see Approved Methods on the CAPS Resource and Collaboration Site, at http://caps.ceris.purdue.edu/.

Literature-Based Methods:

This pathogen cannot be grown on artificial culture media.

PCR: PCR methods for detecting *S. endobioticum* have been developed using the ITS region of the multi copy rDNA gene (Lévesque et al., 2002; Niepold and Stacheqicz, 2004; Byrne, 2008). An improved real-time PCR assay has recently been developed to detect *S. endobioticum* winter spores in soil and plant extracts (van Gent-Pelzer et al., 2010). PCR allows for detection and quantification of the pathogen in both soil and host tissues.

A soil assay using PCR primers and DNA probes is available and currently being used in Canada and the Netherlands for detection of *S. endobioticum*. This method detects *S. endobioticum* sporangia in soil extracts (Lévesque et al., 2002; van den Boogert et al., 2005). A national diagnostic protocol for Australia is also available (AU-DAFF, 2011).

Microarray: Work has been carried out to develop microarrays to detect this pathogen along with other important viral pathogens of potatoes using the 18S region of rDNA (Abdullahi et al., 2005)

Pathotype Identification: Pathotype identification can be done using the Spieckermann method, the Glynne-Lemmerzahi method, or field tests all of which are explained in EPPO (2003a).

Easily Confused Species

Symptoms may be confused with powdery scab (*Spongospora subterranea* f. sp. *subterrenea*) (Byrne, 2008), bud proliferation (DEFRA, n.d.), and potato smut (*Thecaphora solani*) (Franc, 2007). Powdery scab spore balls can be differentiated from winter sporangia of *Synchytrium endobioticum* through microscopic examination as spore balls are made up of many small cysts (AU-DAFF, 2011).

Potato wart may also be confused with “false wart”, which is caused by environmental conditions. Outgrowths caused by “false wart” are the same color as the tuber, unlike potato wart (USDA, 1990). Some other wild plant species found in potato fields may be infected with *Synchytrium* species other than *S. endobioticum* (EPPO, 2003a). The sporangia may also be confused with pollen grains, which can also be found in the soil (Byrne, 2008).

References

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This datasheet was developed by USDA-APHIS-PPQ-CPHST staff. Cite this document as:

Molet. T., Mackesy, D., and Sullivan, M. 2014. CPHST Pest Datasheet for *Synchytrium endobioticum*. USDA-APHIS-PPQ-CPHST.

Updates

June, 2014: Updated to include the eradication from Austria.
September, 2014: Updated to include Georgia in known distribution.
October, 2014: Updated to include Denmark in known distribution.
March 2015: Added updated risk maps to the pest datasheet.
January 2016: Updated list of states at risk from potato wart.