Dry Rot of Rutabaga Caused by *Fusarium avenaceum*

Rick D. Peters
Agriculture and Agri-Food Canada, Crops and Livestock Research Centre, 440 University Avenue, Charlottetown, PE, Canada C1A 4N6

Tharcisse Barasubiyi
Agriculture and Agri-Food Canada, Eastern Cereal and Oilseed Research Centre, 960 Carling Avenue, Ottawa, ON, Canada K1A 0C6

Joanne Driscoll
Prince Edward Island Horticultural Association, P.O. Box 2232, Charlottetown, PE, Canada C1A 8B9

Additional index words. Brassica napus, swede turnip, crucifers, fungal diseases

Abstract. The rutabaga, also known as the swede turnip, is grown on ~2000 ha in Canada. During the spring of 2006, a grower in Prince Edward Island noticed an advanced level of decay in his stored rutabagas (cv. Thompson Laurentian). About 80% of the stored crop was affected. Lesions on the surface of affected roots were circular to ovate and ranged in size from 10 to 50 mm. The lesions were light brown, with dark borders and some concentric zones evident near the perimeter of the affected tissue. Root tissue within the lesions was shrunken and often wrinkled. Sectioning the root through the lesion revealed an internal advancing dry rot, with an irregular border and cavities that contained white mycelium. Isolation from diseased tissues yielded fungal cultures, which were determined to be *Fusarium avenaceum* using morphological and molecular criteria. Successful completion of Koch’s postulates determined that *F. avenaceum* was indeed the causal agent of rutabaga dry rot. To the authors’ knowledge, this is the first report of *F. avenaceum* causing disease in rutabaga in Prince Edward Island, and likely only the second observation of its occurrence in North America. Dry rot, incited by *F. avenaceum*, may need to be considered as part of the spectrum of postharvest pathogens of rutabaga.

The rutabaga (*Brassica napus* L. [Napo-brassica group]), also known as the swede turnip, is a member of Brassicaceae, and in North America is predominantly grown in California, Colorado, Wisconsin, and Minnesota, as well as several Canadian provinces. In Canada, production occurs on ~1500 ha, with the majority in Ontario (41%), Quebec (29%), and the Atlantic provinces (25%) (Statistics Canada, 2007). Prince Edward Island rutabaga production accounts for ~13% of total production (Statistics Canada, 2007). Much of the crop is stored, sometimes for periods of up to 9 months. Rutabagas are commonly stored at temperatures near 0 °C to reduce postharvest decay, and at a relative humidity greater than 95% to reduce root shrinkage. Rutabagas are particularly susceptible to bruising, especially during mechanical harvesting, which can lead to the development of rot in storage. For this reason, much of the crop in some production areas, including Prince Edward Island, is hand-harvested.

During the spring of 2006, a grower in Prince Edward Island noticed an advanced level of decay in his stored rutabagas (cv. Thompson Laurentian). These rutabagas (9 ha) had been harvested in mid October (typical harvest period in Prince Edward Island). After shipments to early markets, ~122,500 kg (30% of the harvested crop) was placed into storage. Symptoms of disease were first noticed toward the end of January and, by mid February, 80% of the stored crop was affected and ultimately unmarketable. Lesions on the surface of affected roots were circular to ovate and ranged in size from 10 to 50 mm (Fig. 1A). The lesions were light brown, with dark borders and some concentric zones evident near the perimeter of the affected tissue. Root tissue within the lesions was shrunken and often wrinkled. Sectioning the root through the lesion revealed an internal advancing dry rot, with an irregular border and cavities that contained white mycelium (Fig. 1B).

Sections of root tissue (10 × 5 × 3 mm) were excised from the margins of lesions from five diseased rutabagas with a sterile scalpel. The sections were then surface sterilized in 0.6% sodium hypochlorite for 2 min, rinsed twice in sterile distilled water, and blotted dry on sterile filter paper (Whatman no. 4, Whatman, Florham Park, N.J.). Tissue pieces were then plated onto one-quarter strength potato dextrose agar (PDA) in small Petri dishes (60 × 15 mm; Fisher Scientific Co., Nepean, Ont.). Petri dishes were incubated in the dark at 22 °C for 7 d. After incubation, fungal growth was noted emanating from all tissue sections. Hyphal tips from the margins of actively growing cultures were removed with a sterile probe and plated onto PDA to generate pure cultures.

All isolates were morphologically similar, so one was chosen for more detailed identification. To identify the fungal isolate, the following procedures were carried out using DNA sequencing methods and micromorphological observations. Pure cultures were grown in 9-cm polystyrene Petri dishes on PDA for 5 d at 22 °C in the dark. Fungal mycelia were scraped from the surface of the agar using a sterilized scalpell, and total genomic DNA was extracted using the DNeasy Plant Mini Kit (QIAGEN, Valencia, Calif.) according to the manufacturer’s instructions. One microlitr of aliquot was then analyzed on a 1.5% agarose gel in 0.5x Tris–borate–ethylenediamine tetraacetic acid (EDTA) buffer to estimate the concentration and quality of the DNA.

Specific primer sets were used to amplify a 700-bp fragment of the translation elongation factor α gene (EFl-α) region (Barasubiyi et al., 2005). Polymerase chain reaction (PCR) amplification was carried out in an Eppendorf (Brinkmann Instruments, Westbury, N.Y.). Each reaction mixture contained 10 ng (usually 1 µl) diluted template genomic DNA, 1X Titanium Taq buffer (10X), 3.5 mM MgCl₂ included in 10X buffer, 0.1 mM each of four deoxyribonucleotide triphosphates, 0.08 µM of each primer, and 1 U of Titanium Taq DNA polymerase in a total of 10 µl. An initial denaturation step of 95 °C for 3 min was followed by 34 amplification cycles of denaturation (the first five cycles at 95 °C for 45 s, annealing at 68 °C for 45 s, and extension at 72 °C for 90 s). Cycles 6 to 10 and 11 to 34 used the same parameters except that the annealing temperature was 66 and 62 °C, respectively. After the 34 cycles were completed, the amplicons were incubated for an additional 8 min at 72 °C for a final extension. A negative control (without DNA template) was used to test for DNA contamination of reagents and reaction mixtures. To estimate the concentration and quality of PCR, 1 µl ampcion from the PCR reaction was run on a 1.5% agarose gel with 0.5 µg ethidium bromide/milliliter and 0.5X Tris borate EDTA buffer for 45 min. DNA bands were visualized on an ultraviolet transilluminator.

Sequencing of the EF1-α template was performed using the BigDye Terminator V2.0 ready reaction kit from Applied Biosystems (Forster City, Calif.) and 0.5X reactions. An estimated 10 to 20 ng of each ampcion was sequenced in both directions using the same primers described earlier for PCR. Sequencing reactions were analyzed with the ABI 3100-Avant automated sequencer.
The culture was also grown on Spezieller Nährstoffarmer Agar (Nirenberg, 1981) with filter paper at room temperature (≈22 °C under mixed near-ultraviolet light), examined microscopically, and compared with taxonomic descriptions by Nelson et al. (1983) and Gerlach and Nirenberg (1982). The results obtained by DNA sequencing of EF-1α were supported by micromorphological observations (shape of macroconidia). The culture was identified as *Fusarium avenaceum* (Fr.:Fr.) Sacc., and it was deposited in the Canadian Collection of Fungal Cultures, Ottawa, under the accession no. DAOM 237752.

To complete Koch’s postulates, healthy rutabagas were inoculated with the isolate of *F. avenaceum*. Five rutabagas (cv. Thompson Laurentian) were washed and air-dried before inoculation. Three wounds (15 mm in depth, 5 mm in diameter) were made with a cork borer on the sides of the root equidistant from each other. One wound in each root was inoculated with a small disk (5-mm diameter) of PDA (control). The other two wounds on each root were each inoculated with a disk of PDA containing active mycelium of *F. avenaceum* taken from the margin of a 7-d-old culture growing on PDA. After inoculation, the excised root tissue plugs were placed back into the cavities created by their extraction to seal the wounds. Each rutabaga was then placed in a paper bag (Harvest Gold, 4.54 kg; PEI Bag Co. Ltd., Summerside, PEI) and stored in the dark at 10 °C and 95% relative humidity in a vegetable storage facility for 5 weeks.

After 5 weeks in storage, the inoculated rutabagas were examined for external and internal symptoms of disease. Symptoms of disease were like those seen in the original diseased samples, including the development of typical dry rot in internal tissues (Fig. 1B). Control wounds inoculated with PDA disks only developed no disease symptoms (Fig. 1B). Isolations from diseased lesions using the same protocols described previously always yielded isolates of *F. avenaceum*, as determined by micromorphological observations (shape of macroconidia).

*Fusarium avenaceum* is ubiquitous in soil and often able to colonize plant wounds, sometimes leading to disease development. As mentioned, rutabagas are susceptible to bruising during harvest operations, which in turn also makes them susceptible to opportunistic wound pathogens. Although the diseased rutabagas described here were hand-harvested, root damage can still occur when harvested

![Fig. 1. (A) External symptoms of dry rot of rutabaga incited by *Fusarium avenaceum*. (B) Internal decay of rutabaga after artificial inoculation of wounded roots with *F. avenaceum*. Note the control inoculation (agar disk only) on the left.](image-url)
roots are gathered and tossed into crates or handled before entering storage. In addition, tractors used to apply pest control products can sometimes damage roots before harvest. Minimizing root injury would be a key cultural practice to reduce storage rot, in addition to maintaining low storage temperatures. Thiabendazole has excellent efficacy for control of dry rot of potato incited by *F. avenaceum* (Hide et al., 1992; Satyaprasad et al., 1997) and is available as a postharvest application; however, this product is currently not registered for use on rutabaga. Chemical control may become an option in situations when *Fusarium* dry rot of rutabaga becomes entrenched.

**Literature Cited**

Barasubiye, T., K. Seifert, A. Tenuta, S. Rioux, T. Anderson, T. Welacky, and C.A. Levesque. 2005. Monitoring of *Fusarium* species in soybean roots by DNA array hybridization. Phytopathology 95:S59.

Booth, C. 1971. The genus *Fusarium*. Commonwealth Mycological Inst., Kew, UK.

Creelman, D.W. 1962. Summary of the prevalence of plant diseases in Canada in 1961: Diseases of vegetables and field crops. Can. Plant Dis. Surv. 42:46–71.

Dillard, H.R., R.R. Bellinder, and D.A. Shah. 2004. Integrated management of weeds and diseases in a cabbage cropping system. Crop Prot. 23:163–168.

Dillard, H.R. and A.C. Cobb. 2006. *Fusarium avenaceum*, a new fungal head rot of cabbage in New York. Phytopathology 96:530.

Howard, R.J., J.A. Garland, and W.L. Seaman (eds.). 1994. Diseases and pests of vegetable crops in Canada. Can. Phytopathol. Soc. and Entomol. Soc. Can., Ottawa.

Geeson, J.D. 1983. Brassicas, p. 125–156. In: C. Dennis (ed.). Post-harvest pathology of fruits and vegetables. Academic Press, London.

Geiser, D.M., M. Delmar Jimenez-Gasco, S. Kang, I. Makalowska, N. Veeraraghavan, T.J. Ward, N. Zhang, G.A. Kulda, and K. O’Donnell. 2004. FUSARIUM-ID v.1.0: A DNA sequence database for identifying *Fusarium*. Eur. J. Plant Pathol. 110:473–479.

Gerlach, W. and H. Nirenberg. 1982. The genus *Fusarium*: A pictorial atlas. Mitt. Biol. Bund. Land- u. Forstw. Berlin-Dahlem 209:1–406.

Hide, G.A., P.J. Read, and S.M. Hall. 1992. Resistance to thiabendazole in *Fusarium* species isolated from potato tubers affected by dry rot. Plant Pathol. 41:745–748.

Lange, R.M., L.M. Harrison, and P.D. Khurshid. 2000. A new *Fusarium* wilt of canola in Alberta, Canada in 1999. American Phytopathological Society, 2000 Pacific Division meeting abstracts. Publication no. P-2000-0033-PCA. 27 Mar. 2007. <www.apsnet.org/meetings/div/pc00abs.asp>.

Mercier, J., J. Mahkhouf, and R.A. Martin. 1991. *Fusarium avenaceum*, a pathogen of stored broccoli. Can. Plant Dis. Surv. 71:161–162.

Nelson, P.E., T.A. Toussoun, and W.F.O. Marasas. 1983. *Fusarium* species, an illustrated manual for identification. Pennsylvania State University, University Park, Pa.

Nirenberg, H.I. 1981. A simplified method for identifying *Fusarium* species occurring on wheat. Can. J. Bot. 59:1599–1609.

Rimmer, S.R., V.I. Shattuck, and L. Buchwaldt (eds.). 2006. Compendium of Brassica diseases. APS Press, St. Paul, Minn.

Satyaprasad, K., G.L. Bateman, and P.J. Read. 1997. Variation in pathogenicity on potato tubers and sensitivity to thiabendazole of the dry rot fungus *Fusarium avenaceum*. Potato Res. 40:357–365.

Statistics Canada. 2007. Fruit and vegetable production. February 2007. Catalogue no. 22-003-XIB, Vol. 75, no. 2, Statistics Canada, Agriculture Division, Crops Section, Ottawa, Ontario. 40 pp. 27 Mar. 2007. <www.statcan.ca/english/freepub/22-003-X/22-003-XIB22006002.pdf>.

Tahvonen, R., J. Hollo, A. Hannukkala, and A. Kurpa. 1984. *Rhizoctonia solani* damping-off on spring turnip rape and spring rape *Brassica* spp. in Finland. J. Agr. Sci. Fin. 56:143–154.