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Effects of in-season crop-protection combined with postharvest applied fungicide on suppression of potato storage diseases caused by Fusarium pathogens

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ABSTRACT

The effects of fungicides and biofungicides applied as foliar sprays to potatoes during the growing season in combination with storage loading applications to control Fusarium dry rot in stored potato tubers were evaluated. The in-season treatments included in-furrow and foliar application of mefenoxam or phosphorous acid and foliar application of *Bacillus subtilis*. Storage treatments included phosphorous acid, *B. subtilis* and a 3-way mixture of azoxystrobin, fludioxonil and difenoconazole. The experiment utilized two storage temperatures, 10 °C (on cv. FL1879) and 4 °C (on cv. Goldrush). There was a significant interaction between field and storage treatment for Fusarium dry rot incidence. Field treatment with *B. subtilis* or mefenoxam followed by storage treatment with *B. subtilis*, the 3-way mixture of azoxystrobin, fludioxonil and difenoconazole or phosphorous acid resulted in reduced dry rot incidence. Field treatment strategies combined with storage applied fungicides and biofungicides were viable options for controlling dry rot incidence and could be adopted in potato production.

1. Introduction

Fusarium dry rot of potato (*Solanum tuberosum* L.) is a devastating postharvest disease worldwide and is caused by several *Fusarium* species (Boyd, 1972; Secor and Salas, 2001; Gachango et al., 2011a,b) with *Fusarium sambucinum* Fuckel. being the most aggressive species in Michigan (Wharton et al., 2007b; Gachango et al., 2012b). The first symptoms of Fusarium dry rot are usually dark depressions on the surface of the tuber (Peters et al., 2008a,b). In large lesions, the skin becomes wrinkled in concentric rings as the underlying dead tissue desiccates (Fig. 1). Necrotic areas shaded from light to dark chocolate brown or black characterize internal symptoms. This necrotic tissue is usually dry (hence the name "dry rot") and may develop at an injury such as a cut or bruise. The pathogen enters the tuber, often rotting out the center (Fig. 1). Rotted cavities are often lined with mycelia and spores of various colors from yellow to white to pink. Dry rot diagnosis may be complicated by the presence of other tuber pathogens. Soft rot bacteria (*Pectobacterium* spp.) often colonize dry rot lesions, especially when tubers have been stored under conditions of high relative humidity or tuber surfaces are wet (Secor and Salas, 2001).

Fusarium species are common in most soils where potatoes are grown and can survive as resistant spores free in the soil. There are two main opportunities in the potato crop cycle for *Fusarium* species to infect potato tubers in the spring and in the fall (Fig. 2). *F. sambucinum* and *F. solani* are commonly found on seed tubers in the spring. Potato seed tubers are maintained in storage at 3 °C, which is approximately the temperature at which *F. sambucinum* is dormant, and consequently, there is minimal development of dry rot in storage. However, some level of Fusarium dry rot is almost always present in commercially available seed. During the pre-planting phase of potato production, seed tubers are warmed to about 12 °C, then cut into seed pieces prior to planting. Tubers infected with *F. sambucinum* are particularly susceptible to the development of seed piece decay during this phase. In cases of severe disease, seed pieces may rot completely before planting. Alternatively, after planting, more than 50 % of sprouts developing on infected tubers may become diseased and killed outright before emergence (Choiseul et al., 2001). Damage at this stage results in delayed or non-emergence and is usually expressed as poor and uneven stands with weakened plants. Reduction in crop vigor then results from expenditure of seed energy used to produce secondary or tertiary sprouts to compensate for damage to primary sprouts.

Progeny tubers may become contaminated with *Fusarium* spores as they develop in the late summer and early fall. Tubers are

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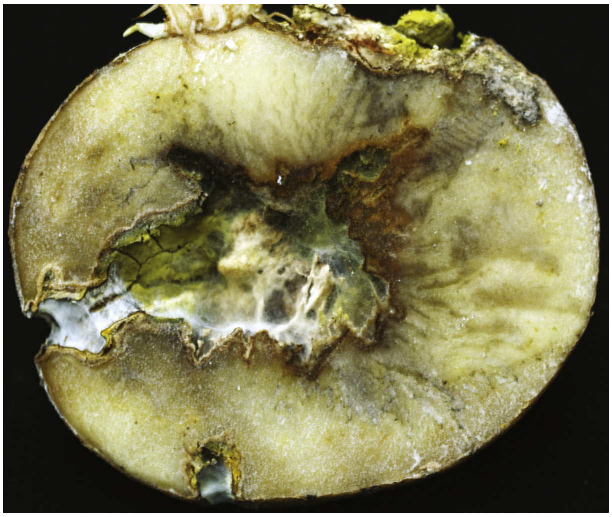

Fig. 1. Symptoms of dry rot caused by *Fusarium sambucinum* on cv. Snowden tuber (from Wharton et al., 2007a).

not usually infected until harvest because the pathogen cannot cause infection unless the potato skin is ruptured, which rarely occurs during the growing season. Wounds caused during harvest and handling provide dormant spores on the tuber surface with multiple points of entry into the tuber. Once the pathogen has penetrated the tuber skin, it begins to grow in the tuber tissue, causing dry rot lesions at the point of entry. In storage, dry rot develops most rapidly at about 90–95% relative humidity and temperatures of 15° to 20 °C. Lower humidity and temperatures retard infection and disease development. However, dry rot may continue to develop at the lowest temperatures safe for storage of potatoes (Wharton et al., 2007a,b).

Dry rot affects both tubers in storage and seed tuber pieces in the field (Wharton et al., 2007a). Losses associated with dry rot have been estimated from 6 to 25%, and occasionally losses as high as 60% have been reported during long-term storage (Chelkowski, 1989; Secor and Salas, 2001). In addition to the damage inflicted on tubers, *Fusarium* species also produce toxins such as trichothecenes, harmful to humans and animals (Desjardins and Plattner, 1989). In Michigan potato production, *F. sambucinum* was the predominant species affecting potatoes in storage and causing seed piece decay after planting (Lacy and Hammerschmidt, 1993) and since then dry rot has been reported in most of the seed lots (Kirk and Wharton, 2008) and additionally causing sprout rotting in seed tubers (Wharton et al., 2006). *F. oxysporum* was the predominant species isolated from seed tubers in Michigan in a survey conducted between 2008 and 2009 by Gachango et al. (2012b).

Measures for controlling dry rot in storage are limited. There is no commercially grown potato cultivar that is resistant to dry rot in

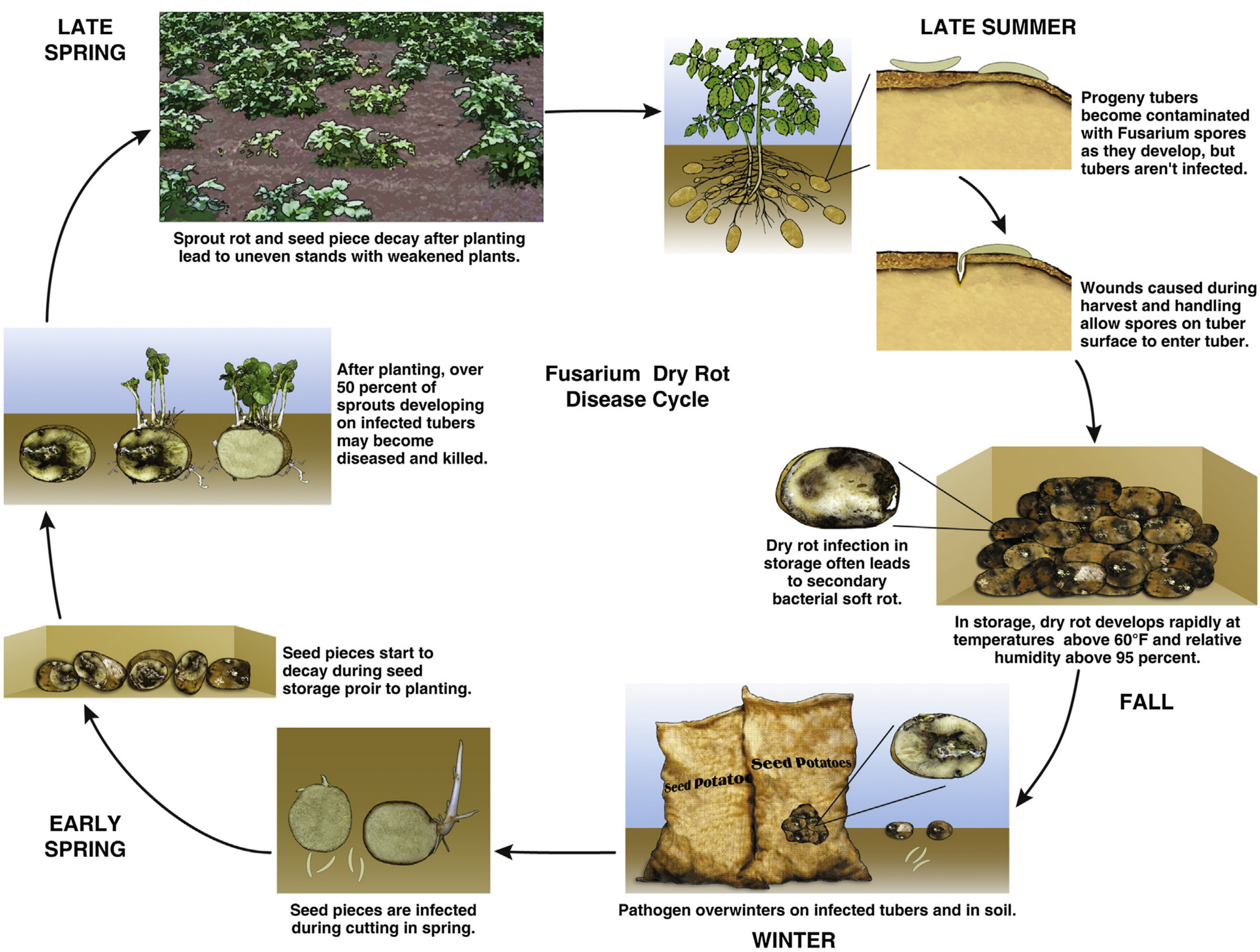

Fig. 2. Fusarium dry rot disease cycle (from Wharton et al., 2007a).