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Review

Management of potato dry rot

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ABSTRACT

Dry rot is a postharvest fungal disease affecting potato (*Solanum tuberosum* L.) tubers. The disease, caused by several species of *Fusarium* such as *F. solani* var. *coeruleum*, *F. sambucinum*, *F. oxysporum*, *F. avenaceum*, *F. culmorum*, results in significant yield losses. Some *Fusarium* species associated with the disease produce toxins, which are implicated in mycotoxicoses of humans and animals. The pathogens cannot penetrate the tuber through the lenticels or in the absence of wounds and cause infection only if the potato skin is ruptured. The seed tuber is considered as the main source of inoculum although soil infested by *Fusarium* spp. also constitutes a source of inoculum. Control of the disease, once provided by the fungicide thiabendazole, is now difficult due to the appearance of thiabendazole-resistant strains and the lack of potato cultivars with high levels of resistance to dry rot. An integrated disease management program including detection strategies, appropriate cultural practices and storage conditions (including a wound healing period) along with the use of synthetic chemical fungicides as seed tuber and/or postharvest treatment is recommended to reduce incidence and severity of dry rot. Recent studies also indicate the possibility that generally recognized as safe (GRAS) compounds and microbial antagonists could eventually be integrated into dry rot management strategies.

1. Introduction

Dry rot is an economically important postharvest fungal disease affecting potato (*Solanum tuberosum* L.) tubers. Yield losses attributed to the disease in storage range from 6 to 25%, with up to 60% of tubers affected in some cases (Stevenson et al., 2001). The disease, caused by several species of *Fusarium* mainly *F. solani* var. *coeruleum* and *F. sambucinum* (Seppänen, 1981; Tivoli et al., 1986a; Singh et al., 1987; Theron and Holz, 1989; Hanson et al., 1996; Stevenson et al., 2001; Cullen et al., 2005; Peters et al., 2008a), appears as large, sunken, concentric rings; cavities underneath the rotted area are usually lined with *Fusarium* mycelia and spores of various colors (Howard et al., 1994; Stevenson et al., 2001). Some *Fusarium* species associated with the disease produce toxins, which are implicated in mycotoxicoses of humans and animals (Desjardins and Plattner, 1989; Kim et al., 1995b).

Control of dry rot has been achieved primarily by postharvest applications of thiabendazole, a benzimidazole fungicide (Secor and Gudmestad, 1999; Mecteau et al., 2002, 2008) because no high level resistance to the pathogens exists among commercially grown cultivars. However, many strains of the pathogens have become resistant to thiabendazole (Desjardins et al., 1993; Holley and Kawchuk, 1996; Platt, 1997), thus resulting in increased incidence and severity of dry rot (Secor and Gudmestad, 1999).

This review will discuss the typical symptoms of potato dry rot, causal agents, disease cycle, infection process, detection and management of the disease. Production of mycotoxins by *Fusarium* species associated with dry rot will be also discussed.

2. Symptoms

Fusarium dry rot initial symptoms appear on tuber at wound sites as shallow small brown lesions after approximately one month of storage. The lesions enlarge in all directions and the periderm eventually sinks and may wrinkle in concentric rings as the underlying dead tissue desiccates (Howard et al., 1994; Stevenson et al., 2001). Cavities underneath the rotted area are usually lined with *Fusarium* mycelia and spores of various colors. Fully rotted tubers become shrivelled and mummified. Dry rot lesions may be invaded by bacterial pathogens causing soft rot decay especially when tubers are wet or under high relative humidity storage conditions (Howard et al., 1994; Stevenson et al., 2001). *Fusarium* species that cause dry rot can also manifest themselves as seed tuber decay and in-field wilt (Secor and Gudmestad, 1999).

3. Causal organisms

Potato dry rot is caused by several species of *Fusarium*. Common causal agents are *F. solani* (Mart.) Sacc. var. *coeruleum* (Lib.

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ex Sacc.) C. Booth (syn. *F. coeruleum* (Libert) Sacc.) (teleomorph: *Nectria haematococca* Berk. & Broome), *F. sambucinum* Fuckel (syns. *F. sulphureum* Schlechtlend., *F. roseum* var. *sambucinum* (Fuckel) SN. & H.) (teleomorph: *Gibberella pulicaris* (Fr.:Fr.) Sacc.), *F. oxysporum* Schlechtend.:Fr., *F. avenaceum* (Fr.:Fr.) Sacc. (teleomorph: *Gibberella* avenacea R.J. Cook), and *F. culmorum* (Wm. G. Smith) Sacc.; *F. solani* var. *coeruleum* and *F. sambucinum* being the most predominant agents (Seppänen, 1981; Tivoli et al., 1986a; Singh et al., 1987; Theron and Holz, 1989; Hanson et al., 1996; Stevenson et al., 2001; Cullen et al., 2005; Peters et al., 2008a). They generally grow rapidly on acidified potato-dextrose agar (PDA). *F. solani* var. *coeruleum* produces a dense, white mycelial mat that may develop a blue, blue-green or purple pigmentation with age. It forms macroconidia, microconidia, and chlamydospores in culture (Howard et al., 1994). *F. sambucinum* may produce a dense aerial mycelium when grown on PDA. When present, aerial mycelium may be white, tan, pink or reddish-brown. Microconidia are generally absent in culture, while chlamydospores and macroconidia are present (Howard et al., 1994).

Other species considered of minor importance have been reported to cause dry rot including *F. acuminatum* Ellis & Everh. (teleomorph: *Gibberella acuminata* Wollenweb.), *F. crookwellense* L.W. Burgess, P.E. Nelson & T.A. Toussoun (syn. *F. cerealis* (Cooke) Sacc.), *F. equiseti* (Corda) Sacc. (teleomorph: *Gibberella intricans* Wollenweb), *F. scirpi* Lambotte & Fautrey, *F. semitectum* Berk. & Ravenel, *F. sporotrichioides* Sherb., *F. tricinctum* (Corda) Sacc. (Seppänen, 1981; Singh et al., 1987; Theron and Holz, 1989; Hanson et al., 1996; Cullen et al., 2005), *F. oxysporum* f. sp. *tuberosi* W.C. Snyder & H.N. Hansen (Manici and Cerato, 1994), *F. torulosum* (Berk. & M.A. Curtis) Nirenberg (Gachango et al., 2011a, 2012), *F. graminearum* Schwabe (teleomorph: *Gibberella zeae* (Schwein.) Petch) (Ali et al., 2005; Peters et al., 2008b; Gachango et al., 2012), *F. sambucinum* var. *coeruleum* Wollenw., and *F. oxysporum* var. *redolens* (Wollenw.) Gordon (Seppänen, 1981).

The relative frequency of *Fusarium* spp. causing potato dry rot varies with location. In North America and in different regions of Europe, *F. sambucinum* is the most prevalent cause of dry rot (Boyd, 1972; Tivoli and Jouan, 1981; Kawchuk et al., 1994; Hanson et al., 1996; Stevenson et al., 2001; Cullen et al., 2005; Ocamb et al., 2007; Peters et al., 2008b; Estrada et al., 2010). *F. coeruleum* is the most predominant pathogen associated with the disease in the United Kingdom (McKee, 1952; Hide and Cayley, 1985; Wastie et al., 1989; Satyaprasad et al., 1997; Peters et al., 2008a) although *F. sambucinum* occasionally causes severe losses in this country (Boyd and Tickle, 1972; Hide et al., 1992). The relative frequency of the *Fusarium* species causing dry rot is not only influenced by crop location but also by other factors such as cultivar(s) used, fungicide(s) applied and seed tuber source(s) (Singh et al., 1987; Peters et al., 2008a).

F. avenaceum, *F. equiseti*, and *F. graminearum* are generally considered of lesser importance as compared to *F. sambucinum* and *F. coeruleum*; they can however cause severe amounts of disease or can be the predominant cause of disease (Moore, 1945; Seppänen, 1981; Choiseul et al., 2007; Estrada et al., 2010; Gachango et al., 2012). For example, a 3-year survey conducted between 1997 and 2000 to identify the fungi causing dry tuber rots in Scottish seed tuber potato stocks showed that *F. avenaceum* was the greatest cause of the disease (Choiseul et al., 2007). A 2004–2005 survey of potatoes from stores in the north-central potato-producing region of the USA showed that *F. graminearum* along with *F. sambucinum* were the predominant causes of the disease (Estrada et al., 2010).

4. Infection process and potato tuber tissue reaction

Fusarium spp. cannot penetrate the tuber through the lenticels or in the absence of wounds. The pathogen causes infection only if the potato skin is ruptured (Boyd, 1972; Nielsen and Johnson, 1972; Secor and Gudmestad, 1999; Stevenson et al., 2001). Infecting hyphae are at first intercellular, becoming intracellular in dead cells. Histological studies conducted with *F. coeruleum* and *F. avenaceum* showed that *F. coeruleum* grew through the intercellular spaces, the adjacent host cells remaining alive for some time whereas *F. avenaceum* killed and penetrated the cells with which it came into contact (McKee, 1954; Stevenson et al., 2001). Lesions near the site of infection may be limited by continuous layer of wound periderm cells with suberin deposition (O'Brien and Leach, 1983; Stevenson et al., 2001). Deposition of suberin alone is not sufficient to prevent the spread of hyphae of the pathogen as wound periderm does but wounded areas need to be sealed with suberin before wound periderm will form (O'Brien and Leach, 1983; Stevenson et al., 2001). Wound healing can minimize dry rot severity by walling off infection sites and preventing lesions from expanding (Stevenson et al., 2001).

F. sambucinum infected tuber tissues were shown to accumulate numerous sesquiterpenes including the phytoalexins rishitin, lubimin, phytuberin, and phytuberol (Corsini and Pavek, 1980; Kuc, 1982; Desjardins et al., 1989; Ray and Hammerschmidt, 1998). Their accumulation was shown however to have no significant effect on the course of dry rot (Ray and Hammerschmidt, 1998) considering that highly virulent strains of the pathogen tolerate phytoalexins (Desjardins and Gardner, 1989). While no correlation between total sesquiterpenes concentrations or concentrations of individual sesquiterpenes in tuber tissues and dry rot resistance was observed (Desjardins et al., 1995; Ray and Hammerschmidt, 1998), genetic studies indicated a correlation between tolerance to rishitin, ability to metabolize rishitin, and virulence of *F. sambucinum* (Desjardins and Gardner, 1989, 1991; Desjardins et al., 1992). The mechanism allowing the fungus to tolerate phytoalexins was further investigated by Fleiβner et al. (2002). Their work strongly suggested that the presence of an ATP-binding cassette multidrug-resistance transporter, which secretes actively the toxic substances present in cells, is necessary for tolerance of *F. sambucinum* to rishitin and virulence on potato tubers. Gene *Gpabc1* coding for this transporter is required for tolerance to phytoalexins and for virulence on potato (Fleiβner et al., 2002).

Increases in lignin content, and polyphenol oxidase and peroxidase activities were also observed in tuber infected tissues. These increases were correlated with the amount of tissue infected rather than the resistance of the tissue to infection (Ray and Hammerschmidt, 1998). Lignin accumulation did not prevent dry rot progression (Ray and Hammerschmidt, 1998).

5. Disease cycle

Dry rot is both a soil- and seed tuber-borne disease (Nielsen and Johnson, 1972; Burgess, 1981; Adams and Lapwood, 1983; Leach, 1985; Choiseul et al., 2001; Cullen et al., 2005; Peters et al., 2008a,b). Soil infested by *Fusarium* spp. surviving for many years as fungal propagules in the soil or as colonizers of living plants or crop debris constitutes a source of inoculum (Nielsen and Johnson, 1972; Burgess, 1981; Leach, 1985; Peters et al., 2008b). The seed tuber is considered as the main source of inoculum (Cullen et al., 2005). Planting contaminated or infected seed tubers results in soil infestation around progeny tubers (Adams and Lapwood, 1983). Contaminated soil adhering on the surface of tubers and equipment will eventually infect tubers through wounds caused by mechanized harvesting and handling. Dry rot will develop during storage, most rapidly when tubers are stored at high humidity and temperatures of 15–20 °C. Dry rot will develop even at the lowest temperatures used for storing potatoes (Stevenson et al., 2001). Lui and Kushalappa (2002) developed models incorporating the most important postharvest factors influencing potato tuber infection