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Potato Black Dot – The Elusive Pathogen, Disease Development and Management

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Abstract

Black dot caused by *Colletotrichum coccodes* was initially considered a mild disease of potato, mainly infecting weakened plants. In the past two decades the fungus has been reported to infect roots and stems relatively early in the growing season, be prevalent on potato and in field soil in major potato production regions of the world, cause early death of foliage by itself and in association with other pathogens, reduce plant and root growth, and to reduce potato yields. Furthermore, the tuber phase of the disease is recognized as a major problem in that unsightly blemishes reduce value of fresh market potatoes. *C. coccodes* has been dubbed an elusive pathogen because infections are latent, disease symptoms on foliage are often non-descript and can be confused with other potential causes, disease effects on potato yield have not been consistent, and the disease is not satisfactorily managed. Sources of variation on yield likely arise from genetic variation within the pathogen population; the host population such as potato cultivar, maturity class, and plant organs infected; environmental variables; cultural and management practices such as timing of fungicide application; crop duration; post-harvest conditions; and interactions of *C. coccodes* with other microbes and with potato cultivars. Considerable research has been done on potato black dot during the last two decades, the scope of this paper is to define our current understanding on the disease and summarize disease management strategies.

Resumen

La mancha negra, causada por *Colletotrichum coccodes*, fue considerada inicialmente una enfermedad ligera en papa, infectando principalmente a plantas débiles. En las dos décadas pasadas, se ha reportado al hongo infectando raíces y tallos relativamente temprano durante el ciclo de cultivo, y ser prevalente en papa y en suelo de campo en las principales regiones productoras de papa del mundo, causando muerte temprana del follaje por si misma y en asociación con otros patógenos, reduciendo los rendimientos de papa. Aún más, la fase de tubérculo de la enfermedad es reconocida como un problema mayor en que manchas desagradables reducen el valor de papas de mercado fresco. *C. Coccodes* ha sido considerado como un patógeno elusivo porque las infecciones son latentes, los síntomas de la enfermedad en el follaje a menudo no se describen y pueden confundirse con otras causas potenciales. Los efectos de la enfermedad en el rendimiento de la papa no han sido consistentes, y la enfermedad no se maneja satisfactoriamente. Las fuentes de variación en rendimiento es probable que surjan de la variación genética al interior de la población del patógeno; la población del hospedante tal como la variedad de papa, tipo de madures, y el órgano de la planta infectado; variables ambientales, prácticas culturales y de manejo, tales como el tiempo en la aplicación del fungicida, duración del cultivo, condiciones de post-cosecha, e interacciones de *C. coccodes* con otros microbios y con variedades de papa. Se ha hecho considerable investigación en la mancha negra de la papa durante las últimas dos décadas. El alcance de este artículo es el de definir nuestro entendimiento actual de la enfermedad y resumir las estrategias de su manejo.

Keywords *Colletotrichum coccodes* · Latent infection · Predisposition · Plant stress · Prior colonization

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Introduction

Black dot caused by the soil-borne fungus, *Colletotrichum coccodes* (Wallr.) S. J. Hughes, was initially considered a mild disease of potato, infecting weakened plants and not known to impact yield (Dickson 1926; Otazu et al. 1978; Scholte et al. 1985). The disease was described as common, but rarely of

economic importance on potato in England in 1965 (Chesters and Hornby 1965). Since then, the fungus has been reported to initially infect roots, stolons and stems relatively early in the growing season and be prevalent on potato and in field soil in major potato production regions of Europe, Israel, South Africa, the United States and other countries of the world (Andrivon et al. 1997; Barkdoll and Davis 1992; Dashwood et al. 1992; Heilmann et al. 2006; Jeger et al. 1996; Read and Hide 1995; Thirumalachar 1967). Furthermore, *C. coccodes* has been associated with early death of the potato crop by itself and in association with other pathogens (Kotcon et al. 1985; Mohan et al. 1992). A marked reduction in plant growth and tuber yield was first reported when sclerotia were added to field soil in Indiana (Stevenson et al. 1976), and premature vine death and yield losses of up to 30% were documented from foliar infections in Idaho and Washington State (Barkdoll and Davis 1992; Johnson 1994; Mohan et al. 1992). Total yield losses of 22 to 30% were observed for potato cultivars Alpha, Cara, Nicola, Agria, and Desiree in Israel (Tsror et al. 1999a) and additional yield reductions have been reported (Aqeel et al. 2008; Mohan et al. 1992; Read and Hide 1995; Thirumalachar 1967). Furthermore, the tuber phase of the disease is recognized as a major problem in that unsightly blemishes caused by *C. coccodes* reduce value of fresh market potatoes on red and light-skinned tubers (Andrivon et al. 1997; Lees and Hilton 2003). Tuber lesions are an especially serious problem with increasing demands for washed potatoes with high-quality appearance for prepacked, fresh market potatoes (Lees and Hilton 2003). Infected tubers in storage also suffer from dehydration losses (Hunger and McIntyre 1979).

However, the effect of black dot on potato yield is not fully known and losses are still found to be inconsistent (Barkdoll and Davis 1992; Pasche et al. 2010). Considerable research has been done on potato black dot during the last two decades, the scope of this paper is to define our current understanding on the disease and summarize disease management strategies.

The Elusive Pathogen

Colletotrichum coccodes has been dubbed an elusive pathogen for several reasons (Johnson 1994; Thirumalachar 1967). First, infections are latent. Infections of below and above ground stems occur relatively early in the growing season but disease symptoms of chlorosis and necrosis or signs of the pathogen such as sclerotia usually do not become evident until plants are stressed or begin to senesce near time of harvest (Johnson and Miliczky 1993b; Pasche et al. 2010). Likewise, tubers become infected in the field and may not develop obvious lesions until storage (Hamm and Johnson 2012). *C. coccodes* alone can cause deep lesions on potato tubers stored for extended periods at 5–15 °C (Glais and Andrivon 2004). The disease does not spread from tuber to tuber in commercial storage, but latent infections become symptomatic and lesions expand in size on previously infected tubers. Latent infections are characteristically caused by several species of *Colletotrichum* on a number of hosts (Zaitlin et al. 2000). Thus, latent infection is not unexpected for *C. coccodes* infecting potato. Second, disease symptoms on foliage are often non-descript and can be confused with Verticillium wilt, natural senescence and nitrogen deficiency, and tuber lesions can be confused with other blemish diseases (Tsror and Johnson 2000; Hunger and McIntyre 1979). Consequently, identifying the disease and evaluating the effects of the disease in research plots and grower fields is complex and the disease is often miss-diagnosed (Pasche et al. 2010). Third, disease effects on potato yield have not been consistent (Pasche et al. 2010); implying biotic and abiotic variables may interact with the pathogen and potato that are not fully understood. Sources of variation on yield likely arise from genetic variation within the pathogen population; the host population such as potato cultivar, maturity class, and plant organs infected; environmental variables; cultural and management practices such as timing of fungicide application; crop duration; post-harvest conditions; and interactions of *C. coccodes* with other microbes and potato cultivars (Andrivon et al. 1998; Aqeel et al. 2008; Brierley et al. 2015; Pasche et al. 2010; Peters et al. 2016). Therefore, more needs to be learned through timely observations during controlled experiments about these interacting factors on disease development and yield. Fourth, the disease is not satisfactorily managed.

Pathogen Diversity

C. coccodes is an asexual fungus with no known sexual reproduction. Fusion of vegetative hyphae is the only known means for exchanging genetic material among isolates. Nevertheless, the fungus is morphologically and genetically diverse (Aqeel et al. 2008; Chesters and Hornby 1965). Variation in aggressiveness of isolates exists on tomato (Loprieno and Guglielminetti 1962), strawberry (Maas and Howard 1985) and potato (Aqeel et al. 2008; Nitzan et al. 2002, 2006c; Nitzan and Tsror 2003). A continuum in the range of aggressiveness likely exists, as illustrated with an isolate of *C. coccodes* pathogenic on eastern black nightshade but non-pathogenic on potato (Andersen and Walker 1985). Differences in aggressiveness within the *C. coccodes* population partially explain inconsistent results on the effects of the fungus on potato yield.

Diversity of *C. coccodes* populations has been studied using nitrate non-utilizing (nit) mutants method for designating vegetative compatibility groups (VCGs). Sub populations of *C. coccodes* from Europe, Israel, North America, Australia and South Africa were characterized by VCG analysis and pathogenicity. In isolates originated from Europe-Israel (EU/I) 8 VCGs were detected with EU/I VCGs 5 and 6 being the most aggressive on potato, while isolates from

EU/I-VCG-1 were the least aggressive (Nitzan et al. 2002; Shcolnick et al. 2007). In North-American (NA) isolates, 7 different VCGs were identified with NA-VCG-2 and 5 being the most aggressive on potato (Nitzan et al. 2006c). In Australian isolates (AUS), 6 VCGs were defined, with AUS-VCG-4 being the most aggressive and AUS-VCG-3 the least (Ben-Daniel et al. 2010b). Isolates from South-Africa (SA) could not be characterized or assigned to VCGs because nit mutants were generated in a very low frequency (L. Tsror unpublished data). Limited interactions between sub-populations from the various continents were observed. Isolates assigned to AUS-VCG-4 anastomosed with isolates assigned to EU/I-VCG-7 and NA-VCG-5 (which also anastomosed with each other). Isolates assigned to EU/I-VCG-6 anastomosed with isolates assigned to NA-VCG-2 and isolates assigned to AUS-VCG-2 anastomosed with isolates assigned to EU/I-VCG-2. The linkage between subpopulations could result from the limited exchange of seed tubers among continents, or could be due to gene flow, selection, or a limited number of polymorphic vegetative incompatibility genes (Ben-Daniel et al. 2010b).

Diversity of *C. coccodes* global sub-populations was molecularly characterized by amplified fragment length polymorphism (AFLP) analysis (Heilmann et al. 2006; Alananbeh et al. 2014; Alananbeh and Gudmestad 2016). Within the global population (EU/I, NA, AUS and SA) five VCG/AFLP sub-populations (1, 2, 3, 4/5 and 6/7) were defined (Alananbeh et al. 2014). These were distributed between two phylogeny clusters, designated Cc-A and Cc-B. Cluster Cc-A was unique and composed of isolates from VCG/AFLP6/7 only, whereas cluster Cc-B was composed of the remaining four VCG/AFLP sub-populations. VCG/AFLP4/5 was the most common VCG globally, followed by VCG/AFLP2. Most of the variation among the four geographic regions originated from within population differentiation (84%). These studies indicate that the global population of *C. coccodes* exists as one large population with five main VCG/AFLPs worldwide, and they are probably of the same origin. However, geographic isolation likely caused these populations to differentiate and form distinct sub-clusters (Alananbeh et al. 2014). Within the NA (nine states in USA) sub-population, four VCG/AFLP were defined (Alananbeh and Gudmestad 2016). Diversity within NA states accounted for 73% of the total genetic diversity, and among populations accounted for 27%. These results suggest that several AFLP/VCGs are widely distributed in NA, and they form a single large population of *C. coccodes.*

A second species of *Colletotrichum*, *C. nigrum*, is morphologically similar but molecularly different to *C. coccodes* (Liu et al. 2013). *C. nigrum*, as well as *C. coccodes*, caused anthracnose symptoms on tomato fruit (Rivera et al. 2016) and may infect potato. The prevalence and effect of *C. nigrum* on potato is not known and needs to be quantified.

Host-Pathogen Interaction

Secreted pectate lyase (PL), a cell-wall-degrading enzyme, plays a major role in fungal pathogenicity. Highly aggressive *C. coccodes* isolates were shown to secrete PL earlier than its secretion by mildly aggressive isolates (Ben-Daniel et al. 2012). The PL gene (CcpelA) cloned from a highly aggressive isolate (US-41), showed high homology (50–70%) to pectate lyases from other fungi. Gene disrupted mutants of highly aggressive isolates showed reduced aggressiveness to tomato fruits and their PL secretion was impaired. On the other hand, over expression of the CcpelA gene in a mildly aggressive isolate (Si-60), increased its aggressiveness and enhanced PL secretion (Ben-Daniel et al. 2012).

pH modulation is a major factor in fungal pathogen-host interaction. The effect of *C. coccodes* growth on pH value at the infected tuber area was evaluated using pH indicators (phenol red and thymol blue) and imaging system. The pH of the infected area increased from potato native pH ~6.0 to 7.4–8.0 whereas no change in pH value was observed in the non-inoculated control. The pH shift stopped abruptly at the margin of the lesion. A correlation between the morphological symptoms of the infected area and the visualized pH was observed (Tardi-Ovadia et al. 2016).

Host Range

The known host range of *C. coccodes* is wide and expanding with additional research. Solanaceous crops such as potato (*Solanum tuberosum*), tomato (*Lycopersicon esclentum*), litchi tomato (*Solanum sisymbriifolium*) and pepper (*Capsicum annuum*) and more than 50 additional plants from 17 plant families primarily in Cucurbitaceae, Leguminosae and Solanaceae are known hosts (Chesters and Hornby 1965; Frederick et al. 2017; Nitzan et al. 2006b; Raid and Pennypacker 1987). Anthracnose symptoms occur on tomato fruit (Dillard and Cobb 1997) and the fungus might be carried in tomato seeds (Ben-Daniel et al. 2010a). It is also a pathogen of pepper seedlings and strawberry (Hong and Hwang 1998; Maas and Howard 1985), although, *C. acutatum* is the most prevalent *Colletotrichum* spp. infecting strawberry (Freeman et al. 1998). Many infected plant species remained asymptomatic or symptoms appeared as inconspicuous chlorotic or necrotic flecks from artificial inoculations (Nitzan et al. 2006b; Raid and Pennypacker 1987). Rotation crops such as yellow mustard (*Guillenia flavescens*), spring canola (*Brassica napus*), and soybean (*Glycine max*) had a high infection potential when artificially inoculated; whereas, alfalfa (*Medicago sativa*), oat (*Avena sativa*) and Timothy grass (*Phleum pretense*) had a low infection potential and barley (*Hordeum vulgare*), maize (*Zea mays*), rye (*Secale cereale*), wheat (*Triticum aestivum*) and orchard grass (*Dactylis glomerata*) were not infected (Nitzan et al. 2006b). Selected rotation crops and weedy hosts, especially eastern black

nightshade (*Solanum ptycanthum*) and velvetleaf (*Abutilon theophrasti*) are asymptomatic when infected but potentially serve as alterative inoculum sources in potato and tomato fields.

C. coccodes was isolated from rhizomes from asymptomatic peppermint (*Mentha x piperita*) collected from a commercial mint field where potatoes had been previously grown. Isolates from a potato stem, another from a potato tuber and one from peppermint were significantly more aggressive than an isolate from tomato when potato roots of young plants were drenched with conidial suspensions (Geary 1999).

The Disease

Symptoms, Signs

Colletotrichum coccodes colonizes underground stems, stolons, daughter tubers, roots and foliage (Johnson 1994; Johnson and Miliczky 1993a). Distinct below- and above-ground phases of the disease are evident from the quantity of the pathogen detected in various plant parts over the course of the growing season (Johnson and Miliczky 1993b). Small, macroscopic (0.1 to 0.5 mm), black sclerotia present on infected tissues are survival structures of the fungus, making them a sign of the disease. Sclerotia are not formed by all isolates of *C. coccodes,* and a sole reliance on their presence in plant tissue could be misleading in disease diagnosis. Brown lesions form on infected roots and the root cortex rots and sloughs off when infection is severe (Pasche et al. 2010). An amethyst coloration is sometime associated with the remnants of the vascular tissues after partial decomposition of the stem and root cortex (Dickson 1926). The cortical root rot phase of black dot leads to direct plant losses, especially when occurring by mid-growing season. Roots have been noted as the most susceptible plant organ to infection (Andrivon et al. 1998). However, roots and stolons were colonized less frequently than above- and belowground stems when considering multiple inoculum sources from soil-, aerial- and tuber-borne inocula (Pasche et al. 2010).

Lesions on leaflets, petioles and above-ground stems from air brown inoculum are initially water soaked and soon turn dark brown to black and resemble those caused by *Alternaria solani* but do not contain concentric rings. Size of lesions vary from 0.3 to >7 mm. Wounding of foliage from wind-blown sand followed by wet periods greater than 12 h favored foliar lesions when inoculum was present. Chlorosis and dropping of lower canopy leaves occurred in the field from either foliar infection or infection of roots and belowground stems with soil-borne inoculum (Johnson 1994; Johnson and Miliczky 1993a).

Both surface and latent infection within the vascular bundles of the tubers may occur (Tsror et al. 1999a). Infection of potato tuber surfaces results in the development of silvery to brown lesions with poorly defined margins, later characterized by the production of sclerotia under humid conditions (Ingram and Johnson 2010). Tuber lesions are similar to those caused by *Helminthosporium solani*, causal agent of silver scurf. Confirmation of these two diseases requires incubation of infected tubers at a high relative humidity for up to 14 days and examining tubers with a hand lens or microscope to observe the characteristic black microsclerotia of black dot or conidia of *H. solani* (Hamm and Johnson 2012). Black dot lesions are more evident on tubers of thin-skinned than thick-skinned or russet-type potato cultivars. The cultivar Dakota Pearl (Fig. 1) is very susceptible to damage caused by *C. coccodes*.

Effect on Yield

The effect of the disease on yield is a reduction of tuber weight, especially in large tubers (Andrivon et al. 1997; Aqeel et al. 2008; Denner et al. 1998; Johnson and Miliczky 1993a; Tsror et al. 1999b). For example, total weight of tubers was significantly reduced and weight of tubers greater than 113 g was significantly reduced 3 of 3 years in the field in the Columbia Basin of Washington State when foliage was lightly sandblasted and then inoculated with conidial suspensions (Johnson 1994). Reductions of tuber weight were significantly higher following root than foliar inoculations of cultivars Russet Burbank, Umatilla Russet, and Russet Norkotah evaluated under greenhouse conditions (Aqeel et al. 2008).

In a study demonstrating the pathogenic nature of *C. coccodes* on potato, stolons of nine of nine cultivars evaluated were severely damaged, collapsed and shriveled when either roots or the exposed collar regions of haulms were inoculated. Only five of the nine cultivars had lesions on the collar of haulms and on roots. The killing of the stolons took place early

Fig. 1 Coalesced lesions on a Dakota Pearl tuber caused by *Colletotrichum coccodes*. Photo courtesy by Eugenia Banks

in some cases, leaving developing tubers detached from the mother plant. Such plant yielded "only few, pebble-like tubers of little commercial value" (Thirumalachar 1967). Damaged and girdled stolons account for reduction of tuber weight.

Significant yield reductions of 22 to 30% were observed in five selected cultivars (including early-maturing ones) under field conditions in Israel (Tsror et al. 1999b). *C. coccodes* inoculation also resulted in reduction of the quality of daughter tubers. *C. coccodes* contamination of dry stems at harvest was relatively high, indicating that infection not only affects potato yield and the tuber quality, but also infests soil and serves as an important source of inoculum for future potato crops.

Yield of some early-season cultivars have not been shown to be affected by *C. coccodes,* and the lack of measurable yield reduction in early-maturing cultivars may be due to a rapid rate of physiological senescence or a high degree of susceptibility to Verticillium wilt, or both, which might not allow *C. coccodes* adequate time to reduce yield (Mohan et al. 1992).

Source of Inoculum

Inoculum is soil-, seed-tuber-, and air-borne. Sclerotia in soil are a major source of inoculum (Johnson et al. 1997; Tsror et al. 1999a) and infection frequencies of sclerotia from soil generally are greater than infection frequencies from tuber-borne inoculum on stems and daughter tubers (Denner et al. 1998; Dung et al. 2012; Lees et al. 2010; Nitzan et al. 2005, 2008). Soil infestations can cause disease symptoms so severe that they mask the effects of infected seed tubers (Dashwood et al. 1992; Dung et al. 2012; Read and Hide 1995). Soil-borne sclerotia originate mainly from infected plants from a prior growing season but may also blow with dust from adjacent fields or be carried with soil on equipment. Sclerotia can remain viable in soil at least eight years (Hide and Read 1991). An increase in soil inoculum increased foliar chlorosis and necrosis, and development of sclerotia on roots and stems under greenhouse conditions (Nitzan et al. 2008) and concentrations of *C. coccodes* in field soil were correlated with plant colonization and black dot development in the field (Barkdoll and Davis 1992; Davis and Everson 1986; Pasche et al. 2010). The threshold for moderate to severe disease development was at a relatively low level of soil inoculum in a greenhouse (Nitzan et al. 2008) and field trials (Pasche et al. 2010).

Infected seed tubers and possibly infested tare dirt associated with seed tubers are main sources for long distant dispersal of the pathogen to previously non-infested fields (Barkdoll and Davis 1992; Dung et al. 2013; Johnson et al. 1997). The fungus grew from infected seed pieces to roots and along stolons towards daughter tubers and colonized belowground tissues progressively away from an infected seed piece at a rate of 1 mm per day (Andrivon et al. 1998; Ingram and Johnson 2010). Length of stolons and growing season are likely factors determining severity of infection of daughter tubers.

Sclerotia from soil may become air-borne and be a contributor to the initial development of the disease. A high potential for foliar infection is present in windy regions with sandy soil, such as the Columbia Basin, where wounds from blowing sand provide entry avenues for the fungus. Rain and sprinkler irrigation may disseminate conidia and microsclerotia via water splashing, and provide moisture needed for germination and infection (Johnson and Miliczky 1993a). Secondary infections on foliage involving water-splashed conidia from acervuli may occur in humid, moist environments.

Asymptomatic infections of below- and above-ground stems occur relatively early in the growing season, generally on a small proportion of plants and increase in numbers until a large proportion of plants are latently infected in fields by mid-season (Johnson and Miliczky 1993b; Pasche et al. 2010). The increase in number of infected plants over time is due to soil (microsclerotia) or air-borne (microsclerotia and conidia) inoculum contacting plant tissues at different times. The pathogen then spreads within and colonizes aboveground stems. Colonization and spread within stems is most rapid as plants enter the tuber bulking stage (growth state 4 (Miller and Hopkins 2008)), and foliage starts to senesce (Nitzan et al. 2006a). Microsclerotia then become visually evident during plant stress or at plant senescence.

Disease Associated and not Associated with Plant Stress

Sclerotia production on stems, stolons and roots is promoted by plant stress and plant senescence (Johnson and Miliczky 1993b) or natural plant maturity, which is a type of plant stress (Otazu et al. 1978). Sclerotia also form on non-senescent plants with no known indication of prior stress. For example, immature microsclerotia are shown in Fig. 2 on the belowground stems of Ranger Russet collected from a plant at initial plant flowering growth stage in a commercial field in the

Fig. 2 Immature sclerotia of *Colletotrichum coccodes* seen as black specks between the second and third nodes from the left on a belowground stem of Ranger Russet collected at initial flowering

Columbia Basin. *C. coccodes* is not an aggressive saprophyte and is not able to initially colonize dead potato tissue in the field. Aggressive saprophytes outcompete *C. coccodes* for substrate on dead and dying potato tissue under field conditions. *C. coccodes* is well adapted at latently infecting and colonizing host tissue while the host is alive (Nitzan et al. 2006a) when saprophytes are at a competitive disadvantage. As the host dies, *C. coccodes* already has control of the colonized tissue and microsclerotia develop as the host tissues senesce. Establishment in host tissue through parasitism gives the parasite a competitive advantage over saprophytes for the substrate when the host dies. The phenomenon is called prior colonization (Bruehl 1987).

Various plant stresses or sub-optimal growing conditions caused by biotic and abiotic factors may also increase susceptibility or predispose potato tissues to increased symptom and microsclerotia development when infected with *C. coccodes* (Johnson and Miliczky 1993b; Otazu et al. 1978). Predisposition is an internal degree of susceptibility resulting from external causes. High or low ambient temperatures, soil moisture extremes, low light intensity and short duration, imbalances in mineral nutrition and plant wounding from blowing sand are some of many potential types of plant stresses that may promote predisposition (Yarwood 1959).

Yield losses are greater in fall-grown than spring-grown potato crops in Israel when day length is decreasing verses increasing (Tsror et al. 1999b). Disease symptoms and fungal colonization were enhanced under short day growth conditions of 8 h light and16 hours dark in controlled greenhouse experiments (Tsror 2004). Light duration may play an important role in *C. coccodes* infection and may be one of the factors responsible for the severe yield losses observed in autumn similarly to *V. dahliae* causing more severe symptoms in fall than in spring (Krikun and Orion 1979). Decreasing and short day length likely stressed potato plants, promoting greater yield reductions when infected with *C. coccodes*.

Late season application of the herbicide metribuzin increased incidence of stems with black dot symptoms in Russet Burbank at one of two field locations in Idaho (Mohan et al. 1992). Metribuzin is used for the control of broad-leaf weeds in potato and tomato fields and can stress potato plants to various degrees, sometimes producing symptoms of herbicide damage.

Interactions with Other Pathogens

Co-infection by both *C. coccodes* and *V. dahliae* has occurred in the field in Israel resulting in enhanced disease symptoms compared to when either pathogen was inoculated separately (Tsror and Hazanovsky 2001). Wilt symptoms and yield loss was not observed for the cultivar Katahdin when inoculated solely with *C. coccodes*, but wilt symptoms and yield reduction were increased over those caused by inoculation solely with *Verticillium dahliae* when the cultivar was inoculated (soil infestation) with both pathogens in a greenhouse (Mohan et al. 1992). Yield loss for the highly susceptible cv. Amethyst by *V. dahliae* was almost double in the presence of *C. coccodes* in the Netherlands (Scholte et al. 1985), and *C. coccodes* was recognized as a contributing cause of the potato early dying complex in the mid-western United States (Kotcon et al. 1985).

However, the association of *C. coccodes* with potato was not dependent on infection by *V. dahliae* in that the proportion of plants infected with *C. coccodes* increased and peaked earlier than the proportion of plants infected with *V. dahliae* in the Columbia Basin (Johnson and Miliczky 1993b). The coincident infection by *C. coccodes* and *V. dahliae* was also less severe than expected if infections were associated in another study (Goodell et al. 1982).

Nutrient Stress and Black Dot Development

Plant stress from nutrient deficiency or imbalance may also increase colonization of potato roots by the black dot fungus. In controlled experiments, Russet Burbank plants produced in tissue culture were grown in a hydroponic solution in growth chambers under temperature and light conditions favoring potato growth. Plant nutrients were supplied at a level where there were no deficiencies or toxicities except for nitrogen (N). Nitrogen (ammonium nitrate) was supplied at 5, 40, 160, and 640 ppm to create plant stress from a nitrogen deficiency and excess. Established plants were inoculated with a spore suspension and allowed to grow. Nitrogen treatments were arranged in a Latin square experimental design, *C. coccodes* had three replicates within two subsamples per replicate. Plants were then destructively sampled at 1, 2 and 3 weeks after inoculation and roots were assayed for *C. coccodes*. Infection severity was assessed by plating roots on modified potato dextrose agar and then determining the extent of infection. The experiment was expanded to test for the effects of potassium (K) (0, 10, 80, and 160 mg K/l potassium nitrate) and phosphorous (P) (0.032, 0.128, 1.00 and 8.2 ml phosphoric acid/l) on black dot development. Experiments were repeated.

Root colonization was greatest at the lowest level of N (5 ppm). Root colonization decreased as N concentration increased to 160 ppm, which was the optimum level of N (based on plant biomass), and then increased as N increased to 640 ppm. Greatest root colonization for K treatments occurred at the lowest level of K (0 mg K) and decreased as K concentration increased to 80 mg (the optimal level of K) and then slightly increased as K concentration increased to 160 mg Mg K (Fig. 3). The same pattern was seen when phosphorous (P) was tested. The greatest root colonization occurred at the lowest level of P (0.032 ml) and then decreased as P concentration increased to the optimal level of P (1.00 ml). Potato roots were

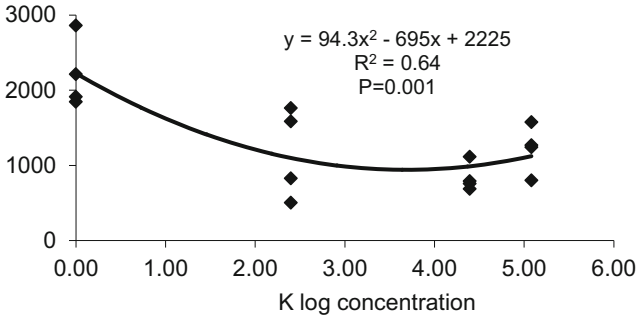

Fig. 3 Root colonization curve for *Colletotrichum coccodes* on Russet Burbank grown in hydroponic culture with various levels of potassium. Non-transformed values of K were 0, 10, 80 and 160 mg

more extensively colonized by the black dot fungus when plants were stressed by a lack and excess of N (Blaisdell et al. 2009), and K (Geary et al. 2009). An increase in disease was not observed when P was adjusted to suboptimum levels compare with the optimum level.

Water Stress and Black Dot Development

Black dot has also been associated with excessive water and with periods of drought (Read and Hide 1988; Stevenson et al. 1976). Water saturated soil from over-irrigation depletes soil oxygen which plant roots need for cellular respiration. Crop plants are subsequently stressed and become more susceptible to pathogens such as *C. coccodes*. For example, severity of black dot increased when Umatilla Russet plants were stressed from excessive irrigation in a greenhouse study. Root weight, density of sclerotia on roots, density of sclerotia on stems and infection of progeny tubers were all negatively affected by *C. coccodes* at an excessive water treatment. Severity of black dot was greater when plants were stressed by excessive irrigation water than when plants were optimally watered. Plant height, SPAD chlorophyll, tuber number and yield were not affected by *C. coccodes* (Cummings and Johnson 2014). Brierley et al. (2015) reported that irrigation increased the severity of disease on tubers in two trials but its effect was less significant when rainfall was high. In observations in commercial fields in Israel severity of black dot increased in rows where irrigation was disrupted and at the edges of fields which also is characterized by uneven irrigation (Tsror unpublished).

Temperature Stress and Black Dot

Considerable yield losses due to *C. coccodes* have been documented in the relatively hot climates of Israel (Tsror et al. 1999b) and the Columbia Basin (Johnson 1994), and relatively high incidences of black dot have been observed in commercial fields during unusually high temperatures in temperate regions (Andrivon et al. 1997; Buonaurio 2002; Otazu et al. 1978). The potato is characteristically a plant of temperate, cool regions (Thurston 2001); whereas, *C. coccodes* grows well at relatively high temperatures for a plant pathogenic fungus. Optimum temperatures for germination and infection is 22 to 28 C (Dillard 1988; Dillard and Cobb 1997). Temperature directly affects metabolism, plant growth and disease severity disease, and increases in disease severity can result when ambient temperatures favor pathogen growth over plant growth (Bruehl 1987).

Crop Rotation and Incidence of Black Dot

Short rotations (relatively few number of rotation crops) between potato crops cause plant stress in that inoculum of *C. coccodes* and other soil-borne pathogens often increase from one planting of potatoes to the next and impact the crop. In a study in the Columbia Basin (Johnson and Cummings 2015), incidence of black dot on tubers of Norkotah was higher when the crop was rotated 1 to 3 years out of potato than for longer rotations. Incidence of black dot decreased significantly as the number of years between potato crops increased. *C. coccodes* was detected in fields out of potato for 10 and 15 years, but incidence of detection of the fungus was low after six or more years out of potato production. Black dot also increased significantly as the number of previous potato crops increased. Both factors, years between potato crops and number of previous potato crops, accounted for a large proportion of disease incidence for black dot ($P < 0.0001$, $R^2 = 0.87$) using multivariate analysis (Johnson and Cummings 2015). Weed hosts may account for long-term persistence of the fungus in soil (Nitzan et al. 2006b).

Cultivar Resistance

Potato cultivars resistant to *C. coccodes* are not commercially available. However, cultivars vary in susceptibility and extremely susceptible cultivars require extra disease management and should be avoided for production unless providing market advantages. The late-maturing cultivar Russet Burbank was more susceptible than the early-maturing cultivar Norgold Russet in Idaho (Mohan et al. 1992). Umatilla Russet, a moderately long-season cultivar was also very susceptible in field tests in North Dakota/Minnesota and susceptible in tests in Washington (Aqeel et al. 2008; Nitzan et al. 2009). Moderately long-season cultivars Ranger Russet and Shepody were very susceptible in Washington (Nitzan et al. 2009). When considering only tuber symptoms in a study in Europe, however, late maturing cultivars had a lower disease frequency than other maturity classes (Andrivon et al. 1997). Resistance of stems, stolons and roots may not be positively correlated with that of tubers. Assessments of disease development on underground plant parts (stems, stolons and roots) revealed that cultivar resistance of the plant genotype assessed acted only at the tuber level, as disease symptoms on other

parts were often high irrespective of published disease resistance ratings (Brierley et al. 2015).

Because of a large genotype by environment interaction (Nitzan et al. 2009) and interaction among potato genotypes and *C. coccodes* strains (Andrivon et al. 1998) quantitative or partial resistance is likely the disease resistance of choice for black dot management (Johnson and Gilmore 1981). Germplasm resistant to stem and root invasion by *C. coccodes* is available (Nitzan et al. 2009). Four of 46 selections evaluated for three years under field conditions in the Columbia Basin had less black dot on stems than standard cultivars all three years. However, the estimate of heritability of resistance to stem colonization by *C. coccodes* was low at 0.13 with confidence intervals between zero and 0.68, indicating that progress in developing resistant cultivars will be difficult (Nitzan et al. 2009). Adding to this difficulty would be combining resistance to other pathogens and traits for yield and tuber quality.

Disease Management

The issue of *C. coccodes* being a weak pathogen is much broader than once thought (Pasche et al. 2010). Black dot is a major concern as a tuber blemish disease on potatoes intended for fresh markets (Andrivon et al. 1997). Furthermore, *C. coccodes* is a threat to potato health and tuber production even though tuber yields are not always negatively impacted (Pasche et al. 2010). Potato crops in agroecosystems will encounter predisposing stresses of various types and interactions on potato are likely between *C. coccodes* and other potato pathogens such as *V. dahliae.* Black dot is currently recognized is an economically important disease justifying management (Tsror and Johnson 2000).

A holistic approach toward potato health (Rowe and Powelson 2008) is needed to manage potato black dot. Both the pathogen and plant stress must be managed. Sources of crop stress include short rotations between potato crops, plant nutrient imbalance, improper irrigation practices, and co-infection with other pathogens including *V. dahliae* and *S. subterranea* f. sp. *subterranea*. Disease management tactics when viable should also be integrated and directed at all potential pathogens and stresses that may increase black dot.

Detection and quantification of *C. coccodes* in seed-tubers and field soil before planting potatoes is important to assess disease risk (Peters et al. 2016). Clearly, avoiding highly infected seed lots and fields with infested soil is recommended. Bioassay of potato roots in soil and quantitative real-time PCR of soil are available and capable of detecting low levels of the pathogen in soil (Brierley et al. 2009; Carnegie et al. 2003; Cullen et al. 2002). Damage thresholds need to be established for various levels of the pathogen.

Potato seed lots with high incidences of infected or infested tubers and excessive tare dirt should be avoided. Physical surfaces that contact freshly cut tubers should be cleaned of grime and tare dirt regularly during the seed handling and cutting processed to remove potentially infested materials that may infected cut seed-tuber surfaces. Fields should be selected for planting with well-drained soil. Soil compaction and over irrigation should be avoided. Adequate nutrients may be especially important just before and during the bulking phase of plant growth. Young plants need to be protected from blowing sand which may increase the incidence of foliar infections. Co-infection with other pathogens, especially the Verticillium wilt fungus will increase damage in many potato cultivars (Tsror and Hazanovsky 2001). Consequently black dot management also includes managing Verticillium wilt and other potential causes of early crop death.

Lengthening the time between potato crops reduces the effect of soil-borne inoculum *of C. coccodes* and other pathogens. Data from the Columbia Basin indicate that more than five years out of potatoes are needed to substantially reduce the effects of disease from soil-borne inoculum. However, incidence of infected tubers decreased 10% per year out of potatoes after the first three years (Johnson and Cummings 2015). Potatoes should be planted on well-drained soil and the crop should not be excessively irrigated when irrigation is available. Weeds that may become infected with *C. coccodes* in fields, and planting infected seed tubers will likely undo the beneficial effects of relatively long-term rotations and other sanitation practices that reduce soil inoculum levels.

Early harvest of potatoes may reduce tuber infections by minimizing exposure of tubers to the pathogen. In field trails over four growing seasons, black dot severity at harvest increased with increasing crop duration. Soil inoculum and crop duration together provided a reasonable prediction of black dot severity at harvest and after a 20-week storage period (Peters et al. 2016).

Soil fumigation with commercially available fumigants has not effectively reduced black dot (Stevenson et al. 1976) and in some situations may have increased disease severity (Tsror et al. 1994). Soil solarisation with tarping for eight weeks and deep tillage with moldboard ploughing to a depth of 30 cm has reduced incidence of black dot (Denner et al. 2000). Deep tillage removes infected plant material on the soil surface and buries it sufficiently deep as not to be an effective source of inoculum for plant infection during the growing season following incorporation. In addition, deep tillage allows infected material to be more susceptible to decomposition by soil microbes (Sumner et al. 1981).

Seed treatment and in-furrow fungicide applications have not effectively reduced incidence of black dot; however, foliar applications of strobilurins fungicides such as azoxystrobin and pyraclostrobin applied before plant row closure have reduced disease incidence in stems and daughter tubers

(Brierley et al. 2015; Cummings and Johnson 2008; Nitzan et al. 2005). Improved potato yield occurred approximately 1 in 3 years from the early foliar applications in the Columbia Basin. Fungicides applied soon after emergence may be the most effective timing in reducing stem colonization since infections that occur early in the growing season are most likely to become symptomatic (Pasche et al. 2010).

Application of strobilurin fungicides at 40–62 days after planting significantly reduced black dot sclerotia in upper and lower stems and tubers in the Columbia Basin (Cummings and Johnson 2008; Nitzan et al. 2005). Effective fungicides need to be applied early in the growing season before infection. For example, incidence of infected stems treated with azoxystrobin, pyraclostrobin, fluoxastrobin, and mandipropamid + difenoconazole was significantly less than the non-treated control plants when fungicides were applied prior to inoculation. However, fungicide application after inoculation did not significantly reduce infection. Chlorothalonil and mancozeb were not effective in preventing infection by *C. coccodes* (Ingram et al. 2011). Resistance to strobilurin fungicides developed in populations of *Alternaria solani* (Pasche et al. 2004) and the fungicides need to be managed to avoid selecting insensitive strains of *C. coccodes* in managing black dot. Effective fungicides should not be the only tactic for disease control. Rather, an integrated approach incorporating diagnostics, risk assessment, and disease management tactics that reduce initial inoculum and the rate of disease development and appropriate potato storage conditions are more likely to provide more durable disease control (Lees and Hilton 2003).

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References

Alananbeh, K.M., and N.C. Gudmestad. 2016. Genetic diversity of *Colletotrichum coccodes* in the United States using amplified fragment length polymorphism analysis. Journal General. *Plant Pathology* 82: 199–211.

Alananbeh, K.M., L. (Lahkim) Tsror, and N.C. Gudmestad. 2014. Genetic diversity of a global population of *Colletotrichum coccodes* using amplified fragment length polymorphism markers. *American Journal of Potato Research* 91: 75–87.

Andersen, H.N., and H.L. Walker. 1985. *Colletotrichum coccodes*; a pathogen of eastern black nightshade (*Solanum ptycanthum*). *Weed Science* 33: 902–905.

Andrivon, D., K. Ramage, C. Gurein, J.M. Lucan, and B. Jouan. 1997. Distribution and fungicide sensitivity of *Colletotrichum coccodes* in French potato-producing areas. *Plant Pathology* 46: 722–728.

Andrivon, D., J.M. Lucas, C. Guerin, and B. Jouan. 1998. Colonization of roots, stolons, tubers, and stems of various potato (*Solanum tuberosum*) cultivars by the black-dot fungus *Colletotrichum coccodes*. *Plant Pathology* 47: 440–445.

Aqeel, A.M., J.S. Pasche, and N.C. Gudmestad. 2008. Variability in morphology and aggressiveness among North American vegetative compatibility groups *of Colletotrichum coccodes*. *Phytopathology* 98: 901–909.

Barkdoll, A.W., and J.R. Davis. 1992. Distribution of *Colletotrichum coccodes* in Idaho and variation of pathogenicity on potato. *Plant Disease* 76: 131–135.

Ben-Daniel, B., D. Bar-Zvi, and L. Tsror Lahkim. 2010a. Infection of tomato seeds by *Colletotrichum coccodes*. *Phytoparasitica* 38: 167–174.

Ben-Daniel, B., D. Bar-Zvi, and L. Tsror Lahkim. 2012. Pectate lyase affects pathogenicity in natural isolates of *Colletotrichum coccodes* and in pelA gene-disrupted and over expressing mutant lines. *Molecular Plant Pathology* 13: 187–197.

Ben-Daniel, B., D. Bar-Zvi, D. Johnson, R. Harding, M. Hazanovsky, and L. Tsror Lahkim. 2010b. Vegetative compatibility groups in *Colletotrichum coccodes* subpopulations from Australia and genetic links with subpopulations from Europe/Israel and North America. *Phytopathology* 100: 271–278.

Blaisdell, B., B. Geary, J. Morton, M. Kearns, and D.A. Johnson. 2009. Available nitrogen levels influence *Colletotrichum coccodes* infection severity of Russet Burbank potato roots. *Phytopathology* 99: S12.

Brierley, J.L., A.J. Hilton, S.J. Wale, J.C. Peters, P. Gladders, N.J. Bradshaw, F. Ritchie, K. MacKenzie, and A.K. Lees. 2015. Factors affecting the development and control of black dot on potato tubers. *Plant Pathology* 64: 167–177.

Brierley, J.L., J.A. Stewart, and A.K. Lees. 2009. Quantifying potato pathogen DNA in soil. *Applied Soil Ecology* 41: 234–238.

Bruehl, G.W. 1987. *Soilborne plant pathogens*. New York: Macmillan Publishing Company 368 pgs.

Buonaurio, R. 2002. Occurrence of black dot of potato caused by *Colletotrichum coccodes* in central Italy. *Plant Disease* 86: 562.

Carnegie, S.F., J.W. Choiseul, and A.M.I. Roberts. 2003. Detection of *Colletotrichum coccodes* and *Helminthosporium solani* in soils by bioassay. *Plant Pathology* 52: 13–21.

Chesters, C.G.C., and D. Hornby. 1965. Studies on *Colletotrichum coccodes* 1. The taxonomic significance of variation in isolates from tomato roots. *Transaction of the British Mycological Society* 48: 573–581.

Cullen, D.W., A.K. Lees, I.K. Toth, and J.M. Duncan. 2002. Detection of *Colletotrichum coccodes* from soil and potato tubers by conventional PCR and real-time quantitative PCR. *Plant Pathology* 51: 281–292.

Cummings, T.F., and D.A. Johnson. 2008. Effectiveness of early-season, single applications of azoxystrobin for the control of potato black dot as evaluated by three assessment methods. *American Journal of Potato Research* 85: 422–431.

Cummings, T.F., and D.A. Johnson. 2014. Effects of soil water level, black dot (*Colletotrichum coccodes*) infested soil and nutrient depletion on potato in a controlled environment. *American Journal of Potato Research* 91: 327–336.

Dashwood, E.P., R.A. Fox, and D.A. Perry. 1992. Effect of inoculum source on root and tuber infection by potato blemish disease fungi. *Plant Pathology* 41: 215–223.

Davis, J.R., and D.O. Everson. 1986. Relationship of *Verticillium dahliae* in soil and potato tissue, irrigation method, and N-fertility to Verticillium wilt of potato. *Phytopathology* 76: 730–736.

Denner, F.D.N., C.P. Millard, and F.C. Wehner. 1998. The effect of seed- and soil-borne inoculum of on the incidence of black dot on potatoes. *Potato Research* 41: 51–56.

Denner, F.D.N., C.P. Millard, and F.C. Wehner. 2000. Effect of soil solarisation and mouldboard ploughing on black dot of potato, caused by *Colletotrichum coccodes*. *Potato Research* 43: 195–201.

Dickson, B.T. 1926. The black dot disease of potato. *Phytopathology* 16: 23–40.

Dillard, H.R. 1988. Influence of temperature, pH, osmotic potential, and fungicide sensitivity on germination of conidia and growth from sclerotia of *Colletotrichum coccodes* in vitro. *Phytopathology* 78: 1357–1361.

Dillard, H.R., and A.C. Cobb. 1997. Disease progress of black dot on tomato roots and reduction in incidence with foliar applied fungicides. *Plant Disease* 81: 1439–1442.

Dung, J.K.S., P.B. Hamm, J.E. Eggers, and D.A. Johnson. 2013. Incidence and impact of *Verticillium dahliae* in soil associated with certified potato seed lots. *Phytopathology* 103: 55–63.

Dung, J.K.S., J.T. Ingram, T.F. Cummings, and D.A. Johnson. 2012. Impact of seed lot infection on the development of black dot and Verticillium wilt of potato in Washington. *Plant Disease* 96: 1179–1184.

Frederick, Z.A., T.F. Cummings, C.R. Brown, R.A. Quick, and D.A. Johnson. 2017. Evaluation of *Solanum sisymbriifolium* as a potential inoculum source of *Verticillium dahliae* and *Colletotrichum coccodes*. *Plant Disease* 101: 1300–1305.

Freeman, S., T. Katan, and E. Shabi. 1998. Characterization of *Colletotrichum* species responsible for anthracnose diseases of various fruits. *Plant Disease* 82: 596–605.

Geary, B.D. 1999. Epidemiology of silver scurf and black dot, two tuber diseases of potato. Ph.D. Thesis. Department of Plant Pathology, Washington State University. 130 pgs.

Geary, B., M.J. Kearns, E. Song, B. Blaisedel, D.A. Johnson, B.G. Hopkins, and V.D. Jolly. 2009. Infection severity of *Colletotrichum coccodes* in Russet Burbank potatoes with respect to environmental potassium. *Phytopathology* 99: S41.

Glais, I., and D. Andrivon. 2004. Deep sunken lesions – an atypical symptom on potato tubers caused by *Colletotrichum coccodes* during storage. *Plant Pathology* 53: 254.

Goodell, J.J., M.L. Powelson, and T.C. Allen. 1982. Interrelations between potato virus X, *Verticillium dahliae*, and *Colletotrichum atramentarium* in potato. *Phytopathology* 72: 631–634.

Hamm, P.B., and D.A. Johnson. 2012. Silver scurf and black dot development on fresh marketed Russet Norkotah tubers in storage. Potato Progress XII, No 17. 4pp.

Heilmann, L., N. Nitzan, D.A. Johnson, J.S. Pasche, C. Doetkott, and N.C. Gudmestad. 2006. Genetic variability in the potato pathogen *Colletotrichum coccodes* as determined by amplified fragment length polymorphism and vegetative compatibility group analyses. *Phytopathology* 96: 1097–1107.

Hide, G.A., and P.J. Read. 1991. Effects of rotation length, fungicide treatment of seed tubers and nematicide on diseases and the quality of potato tubers. *Annual of Applied Biology* 119: 77–87.

Hong, J.K., and B.K. Hwang. 1998. Influence of inoculum density, wetness duration, plant age, inoculation method, and cultivar resistance on infection of pepper plants by *Colletotrichum coccodes*. *Plant Disease* 82: 1079–1083.

Hunger, R.M., and G.A. McIntyre. 1979. Occurrence, development and losses associated with silver scurf and black dot on Colorado potatoes. *American Potato Journal* 56: 289–306.

Ingram, J., and D.A. Johnson. 2010. Colonization of potato roots and stolons by *Colletotrichum coccodes* from tuberborne inoculum. *American Journal of Potato Research* 87: 382–389.

Ingram, J., T.F. Cummings, and D.A. Johnson. 2011. Response of *Colletotrichum coccodes* to selected fungicides using a plant inoculation assay and efficacy of azoxystrobin applied by chemigation. *American Journal of Potato Research* 88: 309–317.

Jeger, M.J., G.A. Hide, P.H.J.F. Van den Boogert, A.J. Termoshuizen, and P. Van Baarlen. 1996. Pathology and control of soil-borne fungal pathogens of potato. *Potato Research* 39: 437–469.

Johnson, D.A. 1994. Effect of foliar infection caused by *Colletotrichum coccodes* on yield of Russet Burbank potato. *Plant Disease* 78: 1075–1078.

Johnson, D.A., and T.F. Cummings. 2015. Effect of extended crop rotations on incidence of black dot, silver scurf and Verticillium wilt of Potato. *Plant Disease* 99: 257–262.

Johnson, D.A., and E.C. Gilmore. 1981. Breeding for resistance to pathogens in wheat. In *Biology and breeding for resistance to arthropods and pathogens in agricultural plants*, ed. M.K. Harris, 263–275. College Station: Texas A & M Univ. and Univ. of Calif.

Johnson, D.A., and E.R. Miliczky. 1993a. Effects of wounding and wetting duration on infection of potato foliage by *Colletotrichum coccodes*. *Plant Disease* 77: 13–17.

Johnson, D.A., and E.R. Miliczky. 1993b. Distribution and development of black dot, Verticillium wilt, and powdery scab on Russet Burbank potatoes in Washington State. *Plant Disease* 77: 74–79.

Johnson, D.A., R.C. Rowe, and T.F. Cummings. 1997. Incidence of *Colletotrichum coccodes* in certified potato seed tubers planted in Washington State. *Plant Disease* 81: 1199–1202.

Kotcon, J.B., D.I. Rouse, and J.J.E. Mitchell. 1985. Interactions of *Verticillium dahliae*, *Colletotrichum coccodes*, *Rhizoctonia solani*, and *Pratylenchus penetrans* in the early dying syndrome of Russet Burbank potatoes. *Phytopathology* 75: 68–74.

Krikun, J., and D. Orion. 1979. Verticillium wilt of potato: Importance and control. *Phytoparasitica* 7: 107–116.

Lees, A.K., and A.J. Hilton. 2003. Black dot (*Colletotrichum coccodes*): An increasingly important disease of potato. *Plant Pathology* 52: 3–12.

Lees, A.K., J.L. Brierley, J.A. Stewart, A.J. Hilton, S.J. Wale, P. Gladders, N.J. Bradshaw, and J.C. Peters. 2010. Relative importance of seed-tuber and soil-borne inoculum in causing black dot disease of potato. *Plant Pathology* 59: 693–702.

Liu, F., L. Cai, P.W. Crous, and U. Damm. 2013. Circumscription of the anthracnose pathogens *Colletotrichum lindemuthianum* and *C. nigrum*. *Mycologia* 105: 844–860.

Loprieno, N., and R. Guglielminetti. 1962. Investigations on tomato anthracnose III. The influence of amino acids on the growth of *Colletotrichum coccodes* (Wallr.) Hughes. *Phytopathology Zeitschrift* 45: 312–320.

Maas, J.L., and C.M. Howard. 1985. Variation of several anthracnose fungi in virulence to strawberry and apple. *Plant Disease* 69: 164–166.

Miller, J.S., and B.G. Hopkins. 2008. Checklist of a holistic potato health management plan. In *Potato Health Management*, ed. D.A. Johnson, 2nd ed. St. Paul: American Phytopathological Society.

Mohan, S.K., J.R. Davis, L.H. Sorensen, and A.T. Schneider. 1992. Infection of aerial parts of potato pants by *Colletotrichum coccodes* and its effects on premature vine death and yield. *American Potato Journal* 69: 547–559.

Nitzan, N., T.F. Cummings, and D.A. Johnson. 2005. Effect of seed-tuber generation, soilborne inoculum, and azoxystrobin application on development of potato black dot caused by *Colletotrichum coccodes*. *Plant Disease* 89: 1181–1185.

Nitzan, N., T.F. Cummings, and D.A. Johnson. 2008. Disease potential of soil-and tuberborne inocula of *Colletotrichum coccodes* and black dot severity on potato. *Plant Disease* 92: 1497–1502.

Nitzan, N., M. Evans, and D.A. Johnson. 2006a. Colonization of potato plants after aerial infection by *Colletotrichum coccodes*, causal agent of potato black dot. *Plant Disease* 90: 999–1003.

Nitzan, N., M.A. Evans, T.F. Cummings, D.A. Johnson, D.L. Batchelor, C. Olsen, K.G. Haynes, and C.R. Brown. 2009. Field resistance to potato stem colonization by the black dot pathogen, *Colletotrichum coccodes*. *Plant Disease* 93: 1116–1122.

Nitzan, N., M. Hazanovsky, M. Tal, and L. Tsror Lahkim. 2002. Vegetative compatibility groups in *Colletotrichum coccodes*, the causal agent of black dot on potato. *Phytopathology* 92: 827–832.

Nitzan, N., B.S. Lucas, and B.J. Christ. 2006b. Colonization of rotation crops and weeds by the potato back dot pathogen *Colletotrichum coccodes*. *American Journal of Potato Research* 83: 503–507.

Nitzan, N., and L. Tsror Lahkim. 2003. Effect of temperature and pH on in vitro growth rate and sclerotial density of *Colletotrichum coccodes* isolates from different VCGs. *American Journal of Potato Research* 80: 335–339.

Nitzan, N., L. Tsror Lahkim, and D.A. Johnson. 2006c. Vegetative compatibility groups and aggressiveness of North American isolates of *Colletotrichum coccodes*, the causal agent of potato black dot. *Plant Disease* 90: 1287–1292.

Otazu, V., N.C. Gudmestad, and R.T. Zink. 1978. The role of *Colletotrichum atramentarium* in the potato wilt complex in North Dakota. *Plant Disease Reporter* 62: 847–851.

Pasche, J.S., R.J. Taylor, and N.C. Gudmestad. 2010. Colonization of potato by *Colletotrichum coccodes*: Effect of soil infestation and seed tuber and foliar inoculation. *Plant Disease* 94: 905–914.

Pasche, J.S., C. Wharam, and N. Gudmestad. 2004. Shift in sensitivity of *Alternaria solani* in response to QoI fungicides. *Plant Disease* 88: 181–187.

Peters, J.C., G. Harper, J.L. Brierley, A.K. Lees, S.J. Wale, A.J. Hilton, P. Gladders, N. Boonham, and A.C. Cunnington. 2016. The effect of post-harvest storage conditions on the development of black dot (*Colletotrichum coccodes*) on potato in crops grown from different durations. *Plant Pathology* 65: 1484–1491.

Raid, R.N., and S.P. Pennypacker. 1987. Weeds as hosts of *Colletotrichum coccodes*. *Plant Disease* 71: 643–646.

Read, P.J., and G.A. Hide. 1988. Effects of inoculum source and irrigation on black dot disease of potatoes (*Colletotrichum coccodes* (Wallr.) Hughes) and its development during storage. *Potato Research* 31: 493–500.

Read, P.J., and G.A. Hide. 1995. Development of black dot disease (*Colletotrichum coccodes* (Wallr.) Hughes) and its effects on the growth and yield of potato plants. *Annals of Applied Biology* 127: 57–72.

Rivera, Y., J. Stommel, J. Damm, A. Ismaiel, C.A. Wyenandt, and J.A. Crouch. 2016. First report of *Colletotrichum nigrum* causing anthracnose disease on tomato frit in New Jersey. *Plant Disease* 100: 2162.

Rowe, R.C., and M.L. Powelson. 2008. Potato health management: A holistic approach. In *Potato health management, Second edition*, ed. D.A. Johnson. St. Paul: American Phytopathological Society.

Scholte, K., J.W. Veenbaas-Rijks, and R.E. Labruyere. 1985. Potato growing in short rotations and the effect of *Streptomyces* spp., *Colletotrichum coccodes*, *Fusarium tabacinum* and *Verticillium dahliae* on plant growth and tuber yield. *Potato Research* 28: 331–348.

Shcolnick, S., A. Dinoor, and L. Tsror Lahkim. 2007. Additional vegetative compatibility groups in *Colletotrichum coccodes* subpopulations from Europe and Israel. *Plant Disease* 91: 805–808.

Stevenson, W.R., R.J. Green, and G.B. Bergesen. 1976. Occurrence and control of potato black dot root rot in Indiana. *Plant Disease Reporter* 60: 248–251.

Sumner, D.R., B. Doupnik Jr., and M.G. Boosalis. 1981. Effects of reduced tillage and multiple cropping on plant diseases. *Annual Review of Phytopathology* 19: 167–187.

Tardi-Ovadia, R., R. Linker, and L. Tsror Lahkim. 2016. Direct estimation of local pH change at infection sites of fungi in potato tubers. *Phytopathology* 107: 132–137.

Thirumalachar, M.J. 1967. Pathogenicity of *Colletotrichum atramentarium* on some potato varieties. *American Potato Journal* 44: 241–244.

Thurston, H.D. 2001. Introduction – origin, history, and importance of the potato. In *Compendium of potato diseases, Second edition*, ed. W.R. Stevenson, R. Loria, G.D. Franc, and D.P. Weingartner. St. Paul: American Phytopathological Society.

Tsror Lahkim, L. 2004. Effect of light duration on severity of black dot caused by *Colletotrichum coccodes* on potato. *Plant Pathology* 53: 288–293.

Tsror Lahkim, L., M. Aharon, and O. Erlich. 1999a. Survey of bacterial and fungal seedborne diseases in imported and domestic potato seed tubers. *Phytoparasitica* 27: 1215–1226.

Tsror Lahkim, L., O. Erlich, and M. Hazanovsky. 1999b. Effect of *Colletotrichum coccodes* on potato yield, tuber quality and stem colonization during spring and autumn. *Plant Disease* 83: 561–565 1999.

Tsror Lahkim, L., O. Erlich, M. Hazanovsky, and I. Peretz. 1994. *Colletotrichum* on potato in Israel, is it a new disease? *Phytoparasitica* 22: 88.

Tsror Lahkim, L., and M. Hazanovsky. 2001. Effect of coinfecion by *Verticillium dahliae* and *Colletotrichum coccodes* on disease symptoms and fungal colonization in four potato cultivars. *Plant Pathology* 50: 483–488.

Tsror Lahkim, L., and D.A. Johnson. 2000. *Colletotrichum coccodes* on potato. In *Colletotrichum: Host specificity, pathology, and host-pathogen interaction*, ed. D. Prusky, S. Freeman, and M.B. Dickman, 362–373. St. Paul: American Phytopathological Society.

Yarwood, C.E. 1959. Predisposition. In *Plant pathology - an advance treatise. The Diseased Plant*, ed. I.J.G. Horsfall and A.E. Dimond. New York: Academic.

Zaitlin, B., E.I. Zehr, and R.A. Dean. 2000. Latent infection of peach caused by *Colletotrichum gloeosporioides* and *Colletotrichum acutatum*. *Canadian Journal of Plant Pathology* 22: 224–228.