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A Comprehensive Overview on Black Scurf of Potato

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A B S T R A C T

Black scurf disease on potato is causing an economically losses to the potato crops which causes both quantitative and qualitative damage occurs in potato production areas all over the world. The fungus limits the growth by forming cankers on sprouts, underground stems, and stolons, and makes tubers ugly by forming black scurf (sclerotia) on tuber surfaces. Black scurf is caused by *Rhizoctonia solani* which is a complex species, at least 13 related but distinct genetically anastomosis groups (AGs). AG-3 is the main cause of black scurf, on the tubers there is brown to black sclerotia that develop late in the growing season. *Rhizoctonia solani* is a soil borne pathogen. So for effective management of this disease, it is requires implementation of an integrated disease management approach and knowledge of each of its stages. It is managed by the integrated approaches such as cultural control *viz;*disease free tubers, crop rotation, organic amendments, soil moisture and management, harvesting time, biological and chemical control.

Introduction

Rhizoctonia solani Kuhn (teleomorph: *Thanathephorus cucumeris* Frank Donk) causing black scurf and stem canker on potato crop (*Solanum tuberosum* L.) which is economically important diseases. The *Rhizoctonia* disease complex is common and occurs in potato production areas throughout the world (Banville 1989; Powelson *et al.*, 1993; Banville *et al.*, 1996; Jeger *et al.*, 1996; Banville and Carling 2001). The fungus limits the growth by forming cankers on sprouts, underground stems, and stolons, and makes tubers ugly by forming black scurf (sclerotia) on tuber surfaces. In few years the fungus causes significant yield reductions (upto 34%) and can cause a significant change in size distribution of tubers (too small or large). Like other seedborne pathogens (Slack 1993; Tsror (Lahkim) *et al.*, 1999), *R. solani* is transmitted by contaminated seed tubers, providing a mechanism for its long-distance dispersal. Once established in soil, the mycelium and sclerotia of the pathogen may then provide an additional source of primary inoculum. The recent species concept stipulates that isolates of *R. solani* possess characteristics such as some shade of brown hyphal pigmentation, branching near the distal septum of cells in young vegetative hyphae, constriction of hyphae and formation of septa a short distance from the point of

origin of hyphal branches, dolipore septa and multinucleate cells in young vegetative hyphae (Parmeter & Whitney, 1970).

The Disease: Infection Process, Disease Development and Symptoms in Potato

Rhizoctonia solani is a soil borne pathogen. *R. solani* colonises the below-ground potato plant surface in response to exudates release from root and shoot (Jeger *et al.,* 1996). It proliferates on the root/stolon system to form a broad network of anastomosing hyphae. During the colonising phase the host plant remains symptomless as long as infection structures are not formed.

The early steps of infection are initiated by successive branching of runner hyphae resulting in the formation of short swollen cells giving rise to infection cushions (Hofman & Jongebloed, 1988; Keijer, 1996). It is believed that infection cushions are prerequisite to inducing stem and stolon lesions (Keijer *et al.,* 1997) and serve as supplementary food base for further colonisation of the plant. AG-3 forms relative small infection cushions as condensed areas in a network of interconnecting hyphae (Keijer, 1996). The infection process is both mechanical and enzymatic, the enzymes involved being DNAse, RNAse, lipase, α-amylase, cellulose, chitinase, pectinase, pectin lyase, β-glucanase, protease and urease (Bertagnolli *et al.,* 1996). The symptoms of the disease are found on both above and below ground parts of the plant. There are two type of symptoms caused by *R. solani* on potato are presence of black colored sclerotia on tubers (black scurf) which is most obvious sign of *Rhizoctonia* disease and others occurring as brown, necrotic lesions on stems and stolons below the soil surface (stem canker). Hymenia of the teleomorph may form near the soil surface on aerial stems. The hymenia do not cause damage to the plant but basidiospores enclosed in them may serve as source of subsequent infections (Banville *et al.,* 1996). Other manifestations of infection include poor and uneven stands; premature dying; pruned stolons and sprouts; lesions on roots, stems and stolons; rosette appearance; girdled stems; necrosis in the stem-end of tubers; russeting of skin; and cracked and malformed tubers (Carling *et al.,* 1989; Hide *et al.,* 1992).

Infected plants generally produce either a large number of small (<3 cm diameter) progeny tubers, or a few oversize tubers (Banville, 1989). Tubers can form in leaf axils of severely infected plants (Hartill, 1989). Severe stem and stolon attacks decrease fresh yield, dry matter yield and dry matter content of tubers and increase the number of deformed and small tubers, whereas the effect on haulm yield and stem number is comparatively small (Scholte, 1989). On the other hand, Gudmestad *et al.,* 1999 report indicate that moderate infection can improve yield and increase gross income per hectare, whereas tuber and soil inoculation with *R. solani* in the greenhouse has been shown to increase the yield of marketable tuber relative to the control (Stack *et al.,* 1999).

Anastomosis groups of *Rhizoctonia solani*

Rhizoctonia solani is a complex species, at least 13 related but distinct genetically anastomosis groups (AGs) (Carling *et al.,* 2002a). AGs have been further divided into subgroups, and subgroups into subsets, on the basis of culture morphology, pathogenicity, host range, nutritional requirements and/or biochemical and genetic properties (Stevens Johnk *et al.,* 1993; Kuninaga *et al.,* 1997, 2000b; Nicoletti *et al.,* 1999; Carling *et al.,* 2002b). Binucleate *Rhizoctonia* isolates are associated with diseased potato plants, but cause minimal damage when tested on potatoes (Tsror, 2010).

At present, 13 AGs (designated AG-1 through AG-13) and 21 subgroups (designated AGs 1-IA, 1-IB, 1-IC, 1- ID, 2-1, 2-2-IIIB, 2-2-IV, 2-2-LD, 2-3, 2-4, 2-BI, 3-IIA, 3-IIB, 3-IIC, 3-TB, 4-HG-I, 4-HG-II, 6- GV, 6-HG-I, 9-TX, 9-TP) are recognised (Ogoshi, 1987; Naito and Kanematsu, 1994; Carling, 1996; Hyakumachi *et al.*, 1998; Carling *et al.*, 1999, 2002a, b; Kuninaga *et al.*, 2000a; Priyatmojo *et al.*, 2001). Subgroups within AGs are partially based on differences in one or more biochemical, genetic, or pathogenic characteristic (Ogoshi, 1987).

Thirteen AGs of *R. solani* are currently known to exist (Carling *et al.*, 2002b). Each AG can vary in host range and often geographic locations. Table lists some of the main hosts associated with individual AGs.

AG-1 and AG-2 cause only least damage to sprouts (Carling & Leiner, 1990b). Isolates of AG-2 were collected from sclerotia on potato tubers and from hymenia and lesions on stems (Chand & Logan, 1984).

AG-3 is the main cause of black scurf, on the tubers there is brown to black sclerotia that develop late in the growing season (Bains & Bisht, 1995; Balali *et al.*, 1995; Campion *et al.*, 2003; Woodhall *et al.*, 2008; Fiers *et al.*, 2011). Sclerotia are most likely responsible for long distance dispersal of the pathogen (Ceresini *et al.*, 2003). Black scurf does not physically harm tubers but reduces their market value (Banville *et al.*, 1996). AG-3 also causes rhizoctonia stem canker (Woodhall *et al.*, 2008). AG-3 was originally designated as a homogeneous population, causing disease only on potato. Members of this group have since been shown to infect tomato and tobacco (Kodama *et al.*, 1982; Meyer *et al.*, 1990; Misawa & Kuninaga, 2010). Stevens Johnk *et al.*, (1993) differentiated AG-3 isolates from potato and tobacco on the basis of culture appearance, fatty acid profile and pathogenicity. Subsequently, AG-3 was divided into three subgroups: AG-3PT (potato type), AG-3TB (tobacco type) and AG-3TM (tomato type), according to the variation in nuclear ribosomal rDNA internal transcribed spacer (ITS) sequences (Kuninaga *et al.*, 2000a; Misawa & Kuninaga, 2010).

Isolates belonging to other AGs (AG-2, AG-4, AG-5, AG-7, AG-8 and AG-9) are associated with potato diseases in different parts of the world, but cause little damage in comparison to AG-3 (Carling *et al.*, 1987, 1998; Bains & Bisht, 1995; Woodhall *et al.*, 2007).

RFLP in the nuclear encoded ribosomal DNA repeat of *R. solani* revealed considerable molecular variation among and within subgroups that have been recognised previously on the basis of anastomosis, morphology and pathogenicity (Vilgalys & Gonzalez, 1990).

Other factors like environmental may influence AG distribution. Anguiz & Martin (1989) reported that AG-3 was more commonly associated with potato diseases at high altitudes and in cool environments, whereas AG-4 was more frequent at low altitudes and in warm climates. In contrast, Bains & Bisht (1995) did not observe specific AGs associated with particular climatic regions, and demonstrated that AG-3, AG-4 and AG-5 occurred on infected potato plants grown in a single potato field. Alternative hosts for *R. solani* may also affect the distribution of the fungus, with solanaceous weeds acting as possible reservoirs for the pathogen (Tsror, 2010).

AG-1 and AG-2 cause least damage to sprouts (Carling & Leiner, 1990a). Isolates of AG-2 were collected from sclerotia on potato tubers and from hymenia and lesions on stems (Chand & Logan, 1984).

AG-3 is by far the most aggressive AG on potato, and indiscriminately attacks roots, stolons and subterranean portions of the main stem (Carling & Leiner, 1990a; Bains & Bisht, 1995). Although AG-3 is virulent across a broad range of temperatures (5 to 25 °C), it is particularly aggressive at 10 to 15 °C, where other AGs generally become less damaging (Carling & Leiner, 1990a). On average, isolates from hymenia were significantly more virulent than isolates from lesions, but neither differed significantly in virulence from isolates obtained from sclerotia or soil (Carling & Leiner, 1990b; Hill & Anderson, 1989).

Hosts range of *R. solani*

It is believed that *Rhizoctonia* species other than *R. solani* have no or little role in causing disease on potato (Carling and Leiner, 1990b). Sturz *et al.,* (1995) survey on Prince Edward Island 80 species and found 56 that harboured *R. solani*. Further inoculation trials with 61 weed species showed that 28 could be infected by the anastomosis groups AG3 and AG5. AG5 has been isolated from winter wheat (Woodhall *et al.,* 2012) and couch grass in the UK (Woodhall and Lees, 2004). *R. solani* AG-3 has also been isolated from the roots and stems of many weeds present in Spanish potato fields (*Chenopodium album, Diplotaxis eurocoides, Solanum nigrum,* and *Sorghum halepense*) (El Bakali *et al.,* 2000). Bains *et al.,* (2002) found that neither *Beta vulgaris, Brassica campestris, Hordeum vulgare, Pisum sativum, Triticum aestivum* nor *Zea mays* were able to be infected by *Rhizoctonia*. However, other studies have isolated *R. solani* AG-3 from barley (Murray 1981) and sugar beet (Windels and Nabben 1989).

Eco-friendly management of black scurf

Since black scurf is soil and tuber borne disease. So for effective management of this disease, it is requires implementation of an integrated disease management approach and knowledge of each of its stages. Inoculum source and its impact on progeny tubers play an important part in strategies for controlling R. solani on potato. R. solani is a tuber- and soilborne pathogen (Frank and Leach 1980; Powelson *et al.,* 1993). The ability to detect the presence of the pathogen in the crop or the soil, to determine threshold levels of inoculums and to investigate the relative importance of seed and soil borne inoculum provides information on which disease management decisions can be based (Lees *et al.,* 2002; Brierley *et al.,* 2009). Although the most important measures are cultural, chemical controls can be utilized in some cases (Harrison *et al.,* 1970; Powelson *et al.,* 1993; Wicks *et al.,* 1995; Johnston 1995; Loria *et al.,* 1997).

Cultural Control

The value of cropping practices to control soil borne diseases has been recognized long before fungicides and fumigants were commonly available. Agronomic factors such as plant material, cultivar, crop rotation, soil management, tillage, irrigation, pesticide application, haulm destruction, harvesting, crop residues, volunteer plants and storage all have a profound influence on the incidence and severity of potato rhizoctoniasis (Jeger *et al.,* 1996).

Disease free tubers of potato

Since infected seed tubers form the main source of inoculum, black scurf disease can be managed to a large extent through the use of certified seed free of sclerotia. Planting clean seed tubers or tubers treated with antagonists or fungicides can be helpful in few conditions. Therefore, keep an eye on black scurf incidence on seed tubers can be the first stage in preventing the disease. A common recommendation is to plant disease-

free tubers, although this is not always possible. In addition, each country defines regulations differently.

Crop rotation

Monoculture should be avoided, because of it is well recognized for further inoculums source for soil borne diseases. Rotations of 3-5 years are often necessary to effectively reduce losses caused by *R. solani*. The frequency with which potatoes are cultivated has a greater effect on black scurf incidence than crop rotation as such. An increased number of potato cropping cycles enhanced the incidence and severity of stem canker due to the increase in soilborne inoculums density (Scholte 1992; Honeycutt *et al.,* 1996). Ideally, three-year rotations or longer are often required to reduce damage caused by *R. solani* (Banville *et al.,* 1996). Despite the fact that crop rotation is practiced to manage Rhizoctonia diseases, an increase in the disease has been observed in potato fields (Celetti *et al.,* 1990; Errampalli *et al.,* 1999). In different 3-year cropping systems (soybean-canola, soybean-barley, sweet corn-canola, sweet corn-soybean, green bean-sweet corn, canolasweet corn, barley-clover) followed by potato, compared with continuous potato growing, both rotation and cropping sequence were important in the microbial characteristics, soilborne disease and tuber quality (Larkin and Honeycutt 2006). Rhizoctonia disease incidence and severity were reduced in most rotations, compared with the continuous potato, where canola, barley or sweet corn prior to potato had the lowest levels of Rhizoctonia disease and the best tuber quality (Larkin and Honeycutt 2006). Rhizoctonia disease was aggravated by rotation with certain legumes, sugar beet and broccoli (Baker and Martinson 1970). Results from different crop-rotation programs vary greatly with respect to their effect on Rhizoctonia incidence (Carter *et al.,* 2003; Peters *et al.,* 2003). Economical rotation systems should be developed according to particular growing conditions.

Table.1 Anastomosis groups of *Rhizoctonia solani*, host, associated valid names and persons credited with discovery of the AG (adapted from Roberts, 1999)

AG	Typical hosts	Discovery credited to:	†
1	Rice, corn, bean	Parmeter *et al.,* (1969)*	*T. sasakii* (AG1-IA) *T. microsclerotia* (AG1-IB)
2	Crucifers, sugar beet, carrot	Parmeter *et al.,* (1969)*	
3	Potato, tobacco	Parmeter *et al.,* (1969)*	
4	Bean, cereals, root rots	Parmeter *et al.,* (1969)*	*T. praticola*
5	Potato, turf grass, root rots	Ogoshi, (1987)	
6	Orchid mycorrhizal	Kuninaga *et al.,* (1978)	
7	Carnation, radish, soybean, saprophyte	Homma *et al.,* (1983)	
8	Cereals	Natio *et al.,* (1985)	
9	Crucifers, potatoes, saprophyte	Carling *et al.,* (1987)	
10	Wheat, barley	Ogoshi *et al.,* (1990)	
11	Lupin, wheat	Carling *et al.,* (1996)	
12	Orchid mycorrhizal	Carling *et al.,* (1999)	
13	Cotton	Carling *et al.,* (1990b)	

*Presence of four anastomosis groups before Parmeter *et al.,* (1969), who is credited with designation of AG1 to AG4.

†In some classifications, individual AGs have been classed as species and given specific names but this has not generally been accepted.

Soil moisture and the management

Practices that favour rapid emergence like shallow planting or using greened seed tubers seem to restrict stem canker infection (Carling & Leiner, 1990b; Jeger *et al.,* 1996) owing to the greater resistance to infection of mature than immature tissue such as emerging spouts and stolons. Firman & Allen (1995) showed that an increase in plant density resulted in an increased severity of black scurf on progeny tubers. Irrigation has variable effects on soil borne diseases. The timing and frequency of irrigation in relation to tuber initiation and tuber disease onset appears to be one of the factors determining tuber disease development of potato tuber diseases. It was reported that lower temperatures and increased soil moisture are favorable for stem canker infection (Hide and Firmager, 1989). Excessive soil moisture may affect potato tubers and lead to swollen lenticels and increased susceptibility to tuber borne infections (Adams and Stevenson, 1990). Moderate soil moisture was also to be conducive for black dot infection and disease development (Read and Hide, 1988).

At light irrigation and heavy irrigation (at 15-20 day), the % disease incidence was 16.7 % and 36.3 % whereas disease intensity was 2.8 and 3.7 respectively. The soil pulverization during summer (April-June) at 20 days intervals was found very effective to minimizing the *R. solani* population up to 1x102 propagules/g soil (Singh *et al., 2005).*

High levels of nitrogen and phosphorus in soil enhance sclerotium formation and disease severity, probably due to more nutritious tuber exudates (Allington, 1936; Papavizas & Davey, 1961; Scholte, 1992). It therefore stands to reason that disease can be reduced by fertilization, although the survival of *R. solani* in artificially infested soil was shown to be little affected by soil fertility.

Compost-amended soil has been found to be suppressive against nematodes, bacteria and soilborne fungi in various cropping systems (Hoitink & Fahy, 1986), although an increase of disease due to compost application has also been demonstrated (Nelson *et al.,* 1983; Tuitert *et al.,* 1998).

The variation in suppressiveness to *R. solani* was ascribed to compost maturity, with immature compost generally being condusive (Nelson *et al.,* 1983; Tuitert *et al.,* 1998). Antagonist enrichment of composts increases the reliability of disease suppressiveness of the composts towards *R. solani* (Postma *et al.,* 2003). Organic amendment *viz.* Vermicompost, Neem Cake and FYM (Farm Yard Manure) showed the inhibition effect on the growth of *R. solani* (Rahul *et al.,* 2014).

Harvesting time and dehaulming

Harvesting methods that are used in potato production can affect the level of black scurf (Dijst *et al.,* 1986). Dijst (1985) suggested that early haulm killing promotes development of sclerotia. Advancing the killing of vines does not lead to a rapid disintegration of the roots. The initially fully functional root system continues to function as water pump for about a week (Dijst, 1985). As evaporation through the foliage ceases, tubers serve as a sink for the water surplus, consequently increasing in mass and commencing leakage. However, the use of herbicides and other chemicals to kill potato shoots just before harvest time can also lead to increased incidence and severity of black scurf on potato tubers (Mulder *et al.,* 1992). Green-crop-harvesting (harvesting the immature crop mechanically and returning the tubers to the soil for curing before final harvesting two to four weeks later) and immature-crop-harvesting (pulling haulms and collecting the tubers by hand) often result in low levels of black scurf (Mulder *et al.,*

1992; Lootsma & Scholte, 1996). Green-crop harvesting has the additional advantage of allowing the application of chemicals or antagonists with the first lifting of the tubers, resulting in increased control of black scurf (Mulder *et al.,* 1992).

Biological control

Biological management of pathogenic organisms is considered to be a potential tool. A variety of bioagents have been used to control the pathogen, *R.solani,* and *Trichoderma* species is the most exploited one (Beagle *et al.,* 1985). Both *Trichoderma harzianum* and *Trichoderma viride* significantly suppress the mycelial growth of R. solani isolates (Hussain *et al.,* 2014). However, *Trichoderma harzianum* is more effective as compared to *T.viride*. *Trichoderma harzianum* produces protease enzyme that is capable of degrading the pathogen cell and reduce the capacity pathogen to grow or infect the plant (Elad *et.al.,* 1980). Suppression of R. solani has been achieved with various fungi and bacteria (actinomycetes, *Bacillus*, fluorescent *Pseudomonas*), as well as nematodes (Escande and Echandi 1991; Tuitert *et al.,* 1998). Arora (2008) reported the treatment of *Trichoderma viride* after seed dressing with boric acid (1.5%) significantly minimized the black scurf disease on potato tubers.

A hypovirulent R. solani isolate significantly reduced (by 56%) the area of infected potato stem tissue under controlled conditions by inoculation with both virulent and hypovirulent isolates (Bandy and Tavantzis 1990). Plants inoculated with the hypovirulent isolate had a significantly higher stolon and stem dry weight (4-fold and 1.7-fold increase, respectively). However, the same hypovirulent isolate failed to reduce disease severity or to stimulate plant growth when was applied to naturally contaminated seed tubers (Bandy and Tavantzis 1990).

Chemical control

A number of fungicides have been evaluated as seed or soil treatment against black scurf and stem canker, e.g. azoxystrobin, benomyl, carbendazim, fluazinam, fludioxonil, imazalil, iprodione, mepronil, pencycuron, propiconazole, quintozene (PCNB), thiabendazole, thiram and tolclofos-methyl (Davis *et al.,* 1971; Chand & Logan, 1982; Leach & Murdoch, 1985; Sumner, 1987; Jager *et al.,* 1991; Olaya *et al.,* 1994; Wicks *et al.,* 1995; Virgen-Calleros *et al.,* 2000), but mostly provided varying and inconsistent control. *R. solani* has acquired resistance to both protectant organic fungicides such as captan, dichlone, maneb, quintozene, thiram and tolclofos-methyl (Shatla & Sinclair, 1963; Elsaid & Sinclair, 1964; Meyer & Parmeter, 1968; Van Bruggen & Arneson, 1984) and to systemic fungicides such as benomyl, carboxin, dichlozoline, oxycarboxin, thiophanate-methyl and 2-(thiocyano-mathylthio)-benzothiazole (TCMTB) (Martin *et al.,* 1984). In most cases the resistance was temporary and possibly due to enzymatic adaptation (Elsaid & Sinclair, 1964). For quintozene and TCMTB, however, the resistance remained stable (Shatla & Sinclair, 1963) and ensued as a consequence of genetic changes. Control of *R. solani* in infested field soil with methyl bromide fumigation is highly effective but tends to aggravate disease derived from infected tubers. Alternatives to methyl bromide, such as methyl iodide, metam-sodium, dichloropropene, chloropicrin, 1,2-dibromo-3-chloropropane and dazomet have been evaluated and found to be equally effective (Ohr *et al.,* 1996; Csinos *et al.,* 1997). Boric acid and pencycuron are the two chemicals that are frequently used by Indian farmers to control black scurf (Khurana *et al.,* 2001). Application of boric acid to seed tubers is

recommended before sprouting usually prior to cold storage (Singh *et al.,* 2002; Arora *et al.,* 2006) whereas pencycuron can be applied to the sprouted tubers at planting (Thind *et al.,* 2002). Bioefficacy of different fungicides [penflufen 240 FS (0.042%), penflufen 240 FS (0.062%), penflufen 240 FS (0.083%), Monceren 250 EC (0.25%), carbendazim 50 WP (0.3%) and Emisan 6 FS (0.25%)] was evaluated against black scurf of potato by dip treatment on Kufri Bahar cultivar. Penflufen (0.062, 0.083%) dip treatment of scurf infected tubers for 10 minutes provided more than 97% disease control (Anil Kumar and Kushal Raj, 2016).

Black scurf of Potato is very complex disease to manage. Black scurf is caused by *Rhizoctonia solani* which is soil and tuber borne disease. Multiple inoculum sources such as soil, tubers, crop residues, raw organic manure, alternative hosts), the genetic and pathogenic variability of the pathogen, the long crop cycle including multiple stages in the tuber handling chain, from seed to the shelf, and limited practical control measures where limited use of chemical is a main goal in the trading markets, all contribute to the difficulty to adequately control the disease in a market with a very low tolerance for visible blemish on harvested tubers. Monitoring disease levels in seed tuber lots enables selection of suitable lots for seed production, thus avoiding the disease as a major proven approach applied that has been used to control other seedborne and soilborne pathogens. In addition, using advanced molecular techniques for the detection of R. solani inoculum in the soil will allow researchers to develop a decision support system to support growers in the selection of seed lots and fields to be cropped with potato. The additional information about epidemiology of the disease is an essential part of integrated disease management. The sustainable management of such a disease apparently requires integrated solutions, combining cultural control practices which reduce inoculum levels in the soil and tuber exposure to soil infestation. To manage Rhizoctonia diseases consequently remains a challenge, despite the current useful knowledge gathered on the biology and epidemiology of the pathogen.

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