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The effect of different pre-crops on *Rhizoctonia solani* complex in potato

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Abstract. Rhizoctonia disease in potato is widely distributed in Estonia. Field experiments with cv. 'Red Fantasy' were undertaken with seven pre-crop treatments at the Estonian Research Institute of Agriculture in 2009 and 2010. Monocropped potato, spring barley, spring barley underseeded with red clover, spring wheat, grain pea, spring oil seed rape and oil seed radish were involved in the study as pre-crops. Growing conditions on both years were rather optimal for potato growth but year 2010 was drier at early bulking stage. The effect of different pre-crops on *Rhizoctonia solani* complex was studied (*i.e.* incidence and severity of stem and stolon canker and black scurf) at 15, 30, 45, 60, 90 and 120 days after planting. Results indicated that pathogen-free seed tubers are of primary importance in the disease control and no pre-crop was suppressive to disease if seed tubers had sufficient amount of inoculum. However, to achieve consistent reduction in disease development, inoculum-free seed tubers and crop rotation with non-host crops should be considered.

Key words: potato, stem canker, stolon canker, black scurf, chemical control, pre-crops.

INTRODUCTION

The disease complex incited by *Rhizoctonia solani* Kühn causes remarkable losses in potato due to reductions in both total and marketable yield across the world (Tsror, 2010). The causal organism was first described by German scientist Julius Kühn in 1858 and the fungus has remained one of the most important and widely distributed fungal pathogens in the crop production as well as it has proved to be a popular fungal test organism in the scientific studies during the last century (Ogoshi, 1996; Menzies, 1970). For long time, no clear distinction between *R. solani* Kühn isolates was used until advent of numbered anastomosis groups (AGs) in the 1960s (Parmeter et al., 1969). Nowadays, at least 13 AGs have been identified, among which AG1 through AG5 affect potato crop as pathogens (Carling et al., 2002). In temperate and cool climates main damage in potato is caused by AG 3 (Carling et al., 1986; Campion et al., 2003; Woodhall et al., 2007, Lehtonen et al., 2008).

In potato, *Rhizoctonia solani* Kühn causes stem and stolon canker and black scurf that have also been recognised as main symptoms and signs of the disease. Later, several other symptoms have been found, *e.g.* skin blemishes (necrosis, cracks), tuber malformations, shifts in tubers size distribution (Ahvenniemi et al., 2006b; Lehtonen, 2009; Buskila et al., 2011 and Muzhinji et al., 2014). A disease cycle for AG 3 of *R. solani* consists infection of sprouts and stolons by hyphae growing from sclerotia or mycelium on seed tubers or in soil (*i.e.* tuber- and soilborne infections, respectively), damage in young, juvenile tubers and production of sclerotia on progeny tubers (*i. e.* typical 'black scurf' sign). The causal organism has also sexual stage *Thanatephorus cucumeris* (Frank) Donk that can be seen as whitish mat at the base of above-ground stems near soil surface (*i.e.* 'white collar') (Cubeta & Vilgalys, 1997; Woodhall et al., 2008). The plants showing this sign have usually severe stem canker below ground (Carling et al., 1989). The host range of AG 3 is confined to *Solanaceae,* among which potato is exceptional due to being clonally propagated (Lehtonen, 2009). Seed tubers provide an excellent route for the fungus to disseminate from field to field, continentwide as well as between continents (Tsror, 2010).

Comparing the effect of tuber- *vs*. soilborne inoculum, both are etiologically important in the disease cycle (Frank & Leach, 1980; Tsror (Lahkim) & Peretz-Alon, 2005). Tuberborne inoculum is close to emerging sprouts and stolons, however, it can be destroyed by dressing seed tubers with effective fungicides or biocontrol agents (Hide & Cayley, 1982; Weinhold et al., 1982; Carling et al., 1989; Wicks et al., 1995; Bains et al., 2002). In contrast, soil-borne inoculum is unevenly distributed in the field soil and control strategies are more limited (Gilligan et al., 1996).

Classical methods for reduction soilborne inoculum and infections include crop rotations with non-hosts and/or disease-suppressive effects (Larkin & Honeycutt, 2006), fumigation, solarisation, sterilisation by steam *etc* (Agrios, 2005). Proper cropping sequences and rotation crops often provide the best and most cost-effective strategy in the disease management as other methods may be costly to use and applicable only on a small scale (*e.g.* greenhouses, seed beds, orchard sites). Intercropping has proved to be a valuable tool in order to control soilborne pathogens. The crucifers (*Brassicaceae*) are among the most effective plants since these crops contain glycosinolates which hydrolyse to release fungistatic or fungicidal isothiocyanates after incorporation into soil (*i.e.'biofumigation'*) (Kirkegaard, 2009). Anyhow, AG 3 of *R. solani* has poor survival without its potato host and population declines rapidly after decomposing host tissues and sclerotia (Lehtonen, 2009; Ritchie et al., 2013). The biological fitness of AG 3 is to produce as many sclerotia as possible on progeny tubers and disseminate by seed tubers (Woodhall et al., 2008; Lehtonen et al., 2009).

Several biocontrol agents (BCA) have been tested for control of *R. solani* (Brewer, Larkin, 2005). In Finland, *Trichoderma harzianum* clearly reduced incidence of black scurf on progeny tubers, but the best control effect was achieved by combining BCA and seed dressing with fungicide (Wilson et al., 2008). Yet, integration of all methods available for disease management provides better and more consistent control for the Rhizoctonia disease complex than using each control measure separately.

The objectives of the study was to examine the effect of different pre-crops on the incidence and severity of stem and stolon canker and black scurf in potato.

MATERIAL AND METHODS

Field experiments with potato cultivar 'Red Fantasy' were undertaken at the Estonian Research Institute of Agriculture in 2009 and 2010. 1-year potato crop was grown prior to planting seven pre-crops, *i.e.* potato, spring barley, spring barley underseeded with red clover, spring wheat, grain pea, spring oilseed rape and oil seed radish in 2008 and 2009. Seed tubers were chitted in a natural light at 15–20 °C for 3–4 weeks. A day before planting, seed tubers of grade 35–55 mm were treated with fludioxonil (commercial formulations Maxim 025 FS and 100 FS, Syngenta Crop Protection AG, Switzerland) by immersion at rate 185 and 500 ppm for 3 minutes to kill mycelium and sclerotia on tuber surface. Higher concentration (500 ppm) was used in 2010 since 185 ppm was too dilute to destroy larger sclerotia. Tubers were allowed to dry and planted by machine in 70-cm rows at 33-cm apart. Compound fertiliser Kemira Gropcare 8-11-23 (Kemira GrowHow, Finland) at rate $N_{75}P_{47}K_{178}$ was in-furrow applied by machine at planting. Weed control performed by ridging and harrowing in 2009 and Titus herbicide supplemented cultural practises in 2010. Plants were sprayed with fungicides up to seven times during growing seasons to protect them from foliar late blight including fungicides such as Ridomil Gold, Ranman and Shirlan. Haulms were flailed 10–12 days before the final sampling (120 days after planting (DAP)) in order to encourage skin set and production of sclerotia on progeny tubers.

Soil has been described in the previous study (Simson et al., 2016). In short, soil type was *Endogleyic Cambisol (eutric)* (Deckers et al., 2002) and texture was a loamy sand. Soil contained 2.5–3.0% organic matter and pH was 5.5.

Experimental years were relatively optimal for potato growth and the mean temperatures were similar. July 2010 was warmer and drier than July 2009 that mainly affected tuber size but both years were favourable for development of *R. solani* (Tabel 1).

Table 1. Meteorological data in the experimental years

Month	Temperatures, °C		Precipitation, mm	
	2009	2010	2009	2010
May	10.8	11.4	20	52
June	13.5	13.5	93	42
July	16.5	21.0	149	78
August	15.5	17.1	89	48
September	12.9	10.9	39	63

The trials had three replicates for all treatments and sampling times. The sampling unit included 2x2 plants in square from two middle adjacent rows. The plants were sampled for stem canker at 15, 30, 45, 60, 90, 120 DAP in 2009 and at 15, 30, 45, 60, 90 DAP in 2010. Stolon canker incidence and severity were assessed at 60, 90, 120 DAP in 2009 and at 60, 90 DAP in 2010. Black scurf infestation on progeny tubers was examined at 120 DAP in both years. For stem and stolon canker, Rhizoctonia stem and stolon lesion index (RSI) were calculated. To find out the RSI, each stem and stolon was assessed separately and placed in one of the following classes: 0, 1–5, 6–25, 26–37, 38–50, 51–75 and 76–100% of belowground stem surface area covered by lesions. The number of stems in each class was multiplied by the midpoint of the class and the sum of all values was divided by the total number of stems to give the RSI for each sampled plant

(Weinhold et al., 1982). The maximum RSI is 88 that is the midpoint of the class 76–100. At the final sampling, progeny tubers of sampled plants were recovered and washed with a brush to remove adhering soil and debris. Tubers were then allowed to dry before the further evaluation. In order to estimate black scurf infestation on progeny tubers, the assessment key for the amount of sclerotia of *R. solani* was used consisting of five classes: free, very lightly, lightly, moderately and heavily covered by sclerotia. By counting tubers in each severity class, black scurf index (BI) was calculated using the formula (Dijst, 1985). The maximum value is 100.

The Statistica version 11.0 (Statsoft Inc.) software package was used for all statistical analyses. Factorial analysis of variance (ANOVA) and one-way ANOVA were applied to test the results. The means are presented with their standard error. The level of statistical significance was set at $P < 0.05$, unless indicated otherwise.

RESULTS AND DISCUSSION

During the growing season, as a mean of all treatments, RSI_{stem} increased from 5.1 at 15 days after planting (DAP) to 23.8 at 90 DAP in 2009. The RSI_{stem} varied from 0.3 at 15 DAP to 11.3 at 90 DAP in 2010 (Fig. 1). Stolon lesions were first assessed at 60 DAP when the RSI_{stolon} was 13.4 in 2009 and 1.1 in 2010 (Fig. 2). At 90 DAP stolon damage was 8.4 in 2009 and 0.9 in 2010. At the final sampling (120 DAP), RSI_{stolon} reached 9.4 in 2009. In 2010, the last evaluation was omitted due to earlier senescense of stems that made it impossible to score disease severity properly.

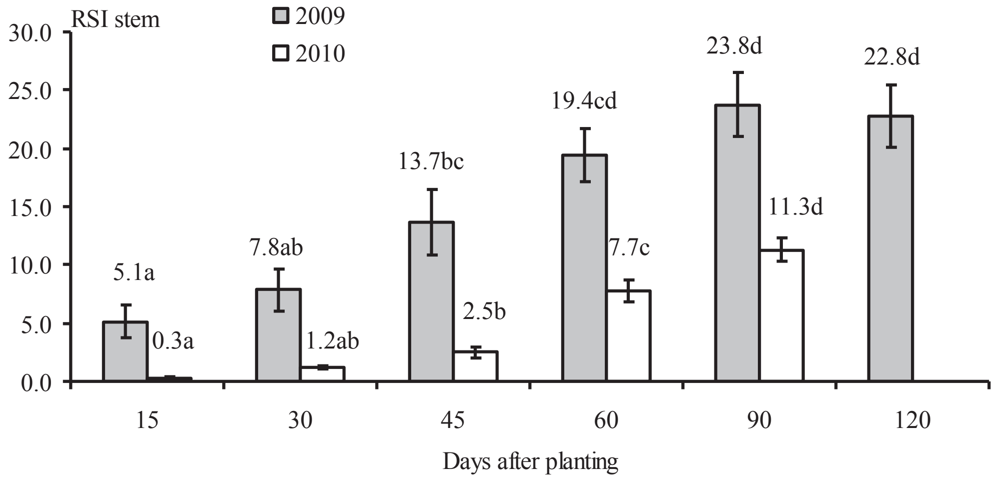

Figure 1. Disease development in belowground portions of the stems measured as RSI_{stem} as mean of the pre-crops. Vertical bars denote 0.95 standard error. Different letters indicate significant differences ($P < 0.05$) between days after planting.

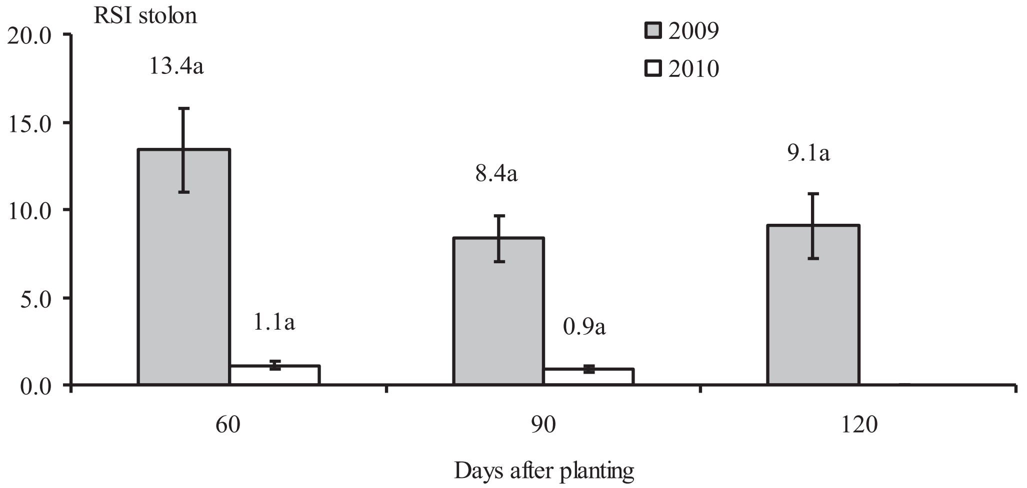

Figure 2. Disease development in stolons measured as RSI_{stolon} as mean of the treatments. Vertical bars denote 0.95 standard error. Different letters indicate significant differences ($P < 0.05$) between days after planting.

The effect of different pre-crops was as follows. In 2009, the lowest RSI_{stem} was recorded in the potato plot after spring wheat pre-crop (12.9) and the highest one in barley with underseeded red clover treatment (18.7). There were no statistically significant differences among treatments. In 2010, RSI scores were lower. Mean RSI stem was 2.6 after spring wheat and 6.1 after barley with underseeded red clover. Stolon damage based RSI_{stolon} was rated 7.3 for pea plot and 15.2 on barley plot in 2009. In 2010, lowest severity was encountered in potato pre-crop plot (0.1) and the highest one in pea plot (2.0).

Table 2. The effect of different pre-crops on stem canker (RSI_{stem}) and stolon canker (RSI_{stolon})

Pre-crop	RSI_{stem}		RSI_{stolon}	
	2009	2010	2009	2010
Potato	15.1 ± 2.3a*	3.0 ± 0.6a	8.0 ± 2.2a	0.1 ± 0.0a
Barley	15.8 ± 2.8a	3.9 ± 0.8ab	15.2 ± 4.1a	0.2 ± 0.1a
Barley us red clover	18.7 ± 3.0a	6.1 ± 1.2b	12.6 ± 3.2a	2.0 ± 0.6b
Spring wheat	12.9 ± 2.4a	2.6 ± 0.6a	11.3 ± 2.7a	0.5 ± 0.3a
Spring rape	14.0 ± 2.6a	3.5 ± 0.8a	9.8 ± 2.9a	0.3 ± 0.1a
Pea	16.5 ± 3.1a	3.7 ± 0.8a	7.3 ± 2.7a	0.8 ± 0.3a
Oil seed radish	15.0 ± 2.3a	4.2 ± 0.9ab	7.6 ± 2.1a	0.7 ± 0.2a

Note. Within the same column, values with different letters are significantly different (ANOVA, Fisher LSD test); * – ± denote the standard error.

Black scurf index (BI) was 0.8 in potato plot and 15.5 in barley plot (Fig. 3). In other treatments, BI varied from 5.4 to 12.9 in 2009. The progeny tubers infested with sclerotia were found on 4–9 of 12 sampled plants in 2009 (data not shown). In 2010, BI reached 3.7 in potato plot. In addition, 7 out of 12 plants had the progeny tubers with sclerotia in this treatment (data not shown). In other plots, BI was 1.3 in barley with underseeded red clover pre-crop and 2.2 for pea pre-crop plot. No black scurf infestation was found on tubers from the plots after spring barley, spring wheat, spring oilseed rape and oil seed radish in 2010.

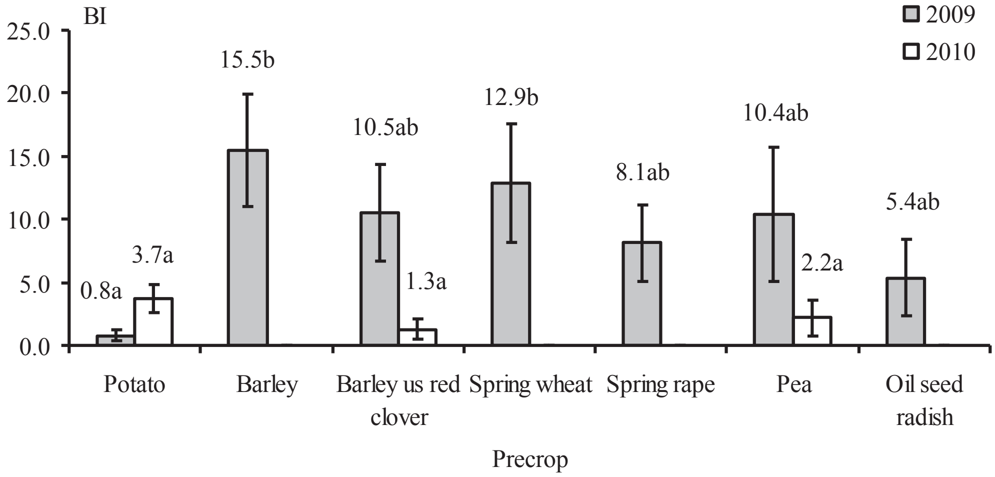

Figure 3. The effect of different pre-crops on black scurf infestation of the progeny tubers measured as BI. Vertical bars denote 0.95 standard error. Different letters indicate significant differences ($P < 0.05$) between pre-crops.

The results obtained in the study were consistent with voluminous data gathered from many experiments across the world. Tuberborne inoculum was recognised more important in causing all symptoms and signs in the present experiments since disease progression began early after planting and it was visible as stem and stolon damage. It allows to suppose that such infections were caused by tuberborne rather than by soilborne inoculum. The higher RSI_{stem}, the higher RSI_{stolon} was. If diseased plants had severe stem and stolon canker, the progeny tubers had also considerable level of black scurf. As confirmed by Hide & Cayley (1982), plants grown from seed tubers infested by black scurf (*i. e.* sclerotia) had more stem canker, stolon damage, hymenium at base of the stems and more black scurf on progeny tubers than plants grown from uninfested seed tubers. There is also a close correlation between stem and stolon canker (Lootsma & Scholte, 1996; Weinhold et al., 1982). Likewise, planting sclerotia-infested seed tubers increased number of progeny tubers with black scurf (Banville,1989; Carling et al., 1989; Brierley et al., 2016). These findings suggest that there might be links between all phases in the disease cycle but the correlations and relative importance of each phase can vary with different infestation level of seed tubers and cropping practises. Soilborne inoculum is also relevant in the disease cycle but its role is less remarkable when proper crop rotations are used and break crops between potatoes are grown. The effect of rotation crops on Rhizoctonia disease was small. In general, the influence of crop rotation on the disease ensues from period between two consecutive potato crops, *i.e.* cropping frequency (Scholte, 1992). Since AG3 has higly specilized on potato and survival without potato host is limited (Lehtonen, 2009), growing non-host rotation crops normally provides sufficient level of control. When 2 years separate potato crops, the inoculum and infections coming from soil is considerably reduced (Gilligan et al., 1996; Ahvenniemi et al., 2006a; Ritchie et al., 2013). This time is needed for breakdown of host tissues and sclerotia of *R. solani* AG3. Tuberborne inoculum can be effectively reduced or eliminated by treating seed tubers with effective fungicide. Fludioxonil (Maxim 025 FS and Maxim 100 FS) used in this study has also been evaluated for control of Rhizoctonia disease in Canada where it proved to be an effective control agent for disease management (Bains et al., 2002). Fungicides applied for seed tuber treatment have an effect on reduction of initial stages of disease (mainly stem and stolon canker) but later these have no suppressive effect on disease development if soilborne inoculum is present (Wilson et al., 2008). Treated seed tubers can be used to evaluate soil inoculum and infections affecting potatoes but disease-free mini-tubers may be better choice. Non-host rotation crops do not usually become infected by potato isolates of *R. solani* (Bains et al., 2002) but leguminous pre-crops (clover and soybean) have sometimes increased disease incidence and severity in potato (Larkin & Honeycutt, 2006). In the latter case, crop debris may serve as nutritious substrate rich in nitrogen for the fungus. Because of patchy distribution of soil inoculum it it complicated to evaluate the influence of these propagules on Rhizoctonia disease. The sufficient soil inoculum load may be achieved by growing potatoes from infested seed tubers for more than 1 year and/or using artificial inoculation of test plots. The effect of soil inoculum is highest when amount of tuber inoculum is lowest, however, it is important to take into account total inoculum load from either source (Atkinson et al., 2010). Several biocontrol agents (BCA) have been tested for control of *R. solani* (Brewer, Larkin, 2005). In Finland, *Trichoderma harzianum* clearly reduced incidence of black scurf on progeny tubers, but the best control effect was achieved by combining BCA and seed dressing with fungicide (Wilson et al., 2008). Yet, integration of all methods available for disease management provides most reliable and consistent control for the Rhizoctonia disease complex.

CONCLUSION

Results demonstrated that it was difficult to quantify soilborne infections in potato caused by *Rhizoctonia solani* if tuberborne inoculum was not completely destroyed. In 2009, fungicide solution was too dilute to kill all the sclerotia, and even in case of 500 ppm fludioxonil in 2010, laboratory test confirmed that the largest sclerotia survived and these might able to cause infections in potato crop. In this study, the influence of soilborne inoculum and infections might be confounded by tuber inoculum, however, its effect seemed to be small. Planting different non-host rotation crops (or break crops) could not reduce disease incidence and severity if tuberborne inoculum was not effectively controlled. The effective management of Rhizoctonia disease should be based on planting inoculum-free seed tubers and application cropping sequences with non-hosts.

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