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Management of beet rust in accordance with IPM principles

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ABSTRACT

High disease pressure from beet rust (*Uromyces beticola*) has been registered in Denmark in recent years, increasing the need to manage this pathogen in accordance with principles of Integrated Pest Management (IPM). A revised disease cycle with all major steps has been proposed including spread of the disease by sea beets (*Beta vulgaris* subsp. *maritima*) acting as a green bridge. Locations with early onset of beet rust were investigated and indicated a connection between early rust occurrence and the presence of local wild sea beets. The impact of the disease was investigated through data from 15 years of testing. In field trials with significant infection, yield reductions up to 11% have been measured and two treatments with reduced doses of epoxiconazole + pyraclostrobin resulted in the highest economic returns on average. Fungicide efficacy and susceptibility of sugar beet cultivars were tested in greenhouse and under field conditions. Results indicated high sensitivity of the pathogen to major fungicides. In greenhouse trials high efficacy was obtained by all conventional fungicides (epoxiconazole, pyraclostrobin, epoxiconazole + pyraclostrobin, difenoconazole + propiconazole) both as preventive and curative treatments. Hydrogen peroxide was tested as control agent, but was not significantly different from the untreated control. The biological control agent *Bacillus subtilis* QST 713 provided 76% control of beet rust when used preventively (not significant). A ranking of relevant cultivars showed variation across years and differences in susceptibility where the least susceptible cultivar on average reduced disease severity to 66% of the most susceptible cultivar. Based on the relevant literature and the findings in this study an IPM management strategy for beet rust should include less susceptible cultivars, fungicides with different modes of action, appropriate doses and application should be carried out according to monitoring and predictions.

1. Introduction

Beet rust on sugar beet (*Beta vulgaris*) is caused by the pathogen *Uromyces beticola* (Boerema et al., 1987). In Denmark beet rust is one of the most prevalent diseases together with powdery mildew (*Erysiphe betae* (syn: *E. polygoni*)) (Hansen, 2015b).

Rust fungi are plant parasites and obligate biotrophs that depend on a living host to complete their life cycle (Voegele et al., 2009). *U. beticola* is an autoecious rust (Gäumann, 1959) and can complete its entire life cycle on the same host with no alternate host involved. Since sea beets, Swiss chard, beet root and mangolds also belong to the *Beta vulgaris* species they can all act as hosts for *U. beticola* (Punithalingam, 1968). *U. beticola* is distributed globally (Termorshuizen and Swertz, 2011; Punithalingam, 1968; Gäumann, 1959). The most important spore form that cause the spread and development of an epidemic is the rust-coloured urediospores that are visible during the growing season (McKay, 1952; Koike et al., 2007; Punithalingam, 1968). The urediospores can germinate at temperatures between 6 and 24 °C with an optimum of 17 °C (Pozhar and Assual, 1971). Germination of *U. beticola* is not light dependent according to Rasmussen (1992). Further development of the disease is favoured at 15–22 °C and supressed at temperatures above 26 °C (Newton and Peturson, 1943). Disease development is favoured by humid conditions (Hanson, 2009; Koike et al., 2007; Voegele et al., 2009). The incubation period can vary from 8 to 32 days depending on temperature and plant age (Pozhar and Assual, 1971).

Beet rust negatively affects root yield and sugar content of sugar beets (Punithalingam, 1968; O'Sullivan, 1996; McKay, 1952). Infections can result in decreased protein synthesis, increased transpiration and weakening of photosynthesis (Pozhar and Assual, 1971). In Denmark, beet rust caused losses of up to 15% in certain areas in 1989 (Nielsen, 1991) and up to 17% yield loss in trials in 1992 (Sørensen and

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Marcussen, 1996). In trials from Ireland conducted between 1988 and 1993 beet rust affected both sugar yield and economic return in susceptible cultivars that were harvested late (O'Sullivan, 1996). However there is little consistency in the literature regarding whether beet rust is an economically important disease in sugar beet production or not. Termorshuizen and Swertz (2011) estimate the disease to cause no significant damage and Wolf and Verreet (2002) state that no control measures are required. Previously *U. beticola* was described as only occasionally epidemic in Europe (McKay, 1952), and Koch (1986) describes it as mostly without economic significance. Both Duffus and Ruppel (1993) and Hanson (2009) state that sugar beet cultivars in the U.S. are resistant to beet rust but that fungicides are necessary for control in some parts of Europe. Fungicides were earlier considered unnecessary in sugar beets; in 1978 Cercospora leaf spot (*Cercospora beticola*) was the only disease known to require the use of fungicides in Europe (Byford, 1996). In Denmark, Cercospora is only a minor disease, however fungicides have been used to control powdery mildew since 1980 (Byford, 1996) initially using sulphur. Today fungicides are commonly used in sugar beets in Denmark and dominated by broad spectrum fungicides from the triazole and strobilurin groups (Hansen, 2015a). Triazoles are inhibitors of ergosterol biosynthesis (Copping and Hewitt, 1998) and belong to 14-demethylase inhibitors (DMI's) (Leroux et al., 2008). Strobilurins are QoI fungicides, and act by inhibiting fungal respiration. With the widespread application of these fungicide groups the risk for resistance development is an issue in disease management. DMI are classified fungicides as having 'medium risk' of resistance development and QoI fungicides as having 'high risk' of resistance development (FRAC, 2010). Resistance has not been found against either of the two groups in *U. beticola* (FRAC, 2013). In this study two alternatives to conventional fungicides have also been included; a biological control organism *Bacillus subtilis* and hydrogen peroxide. *Bacillus subtilis* is a rhizobacterium, that can produce several different antibiotics (Stein, 2005) and possibly induce resistance in the host plant Ongena et al. (2007) and stimulate plant growth (Santoyo et al., 2012). *Bacillus subtilis* QST 713 is registered for control of a number of fungal diseases of vegetable and fruit crops in greenhouses and as field crops in Denmark (Nielsen et al., 2016). Hydrogen peroxide has been of some interest in plant protection due to its antiseptic properties (Baldry, 1983) and as a possible resistance inducer by acting as a signalling molecule in response to biotic and abiotic stress (Neill et al., 2002; Orozco-Cárdenas et al., 2001).

Sugar beet growers are currently under increasing pressure to ensure economic returns. In 2017, quotas and guaranteed prices were discontinued in the EU, and terms for sugar beet production are now determined through negotiations between growers and factories (Nocentini, 2011; NaturErhvervstyrelsen, 2011). The new economic situation is an incentive to reassess the control strategy. Furthermore growing concerns from policy makers about intensive pesticide use, resistance development and the limited availability of agrochemicals will tend to keep future chemical control of pests at a minimum (Hillocks, 2012; Jess et al., 2014). In 2009 integrated pest management (IPM) became the official EU policy (The European Union, 2009) aiming to prevent, monitor, forecast and make economic thresholds for pests and diseases. Leaf disease monitoring and warning systems is an important tool in an IPM program in order to determine whether and when to apply direct control measures. In sugar beets, warning systems have been developed mainly for Cercospora (Vereijssen et al., 2007; Wolf et al., 2001) in which meteorological data has been integrated Racca and Jörg (2007). The basis for warning systems that include rust and powdery mildew have been developed (Kleinhenz et al., 2010).

The aim of this study is to elucidate the impacts of infection with *U. beticola* in sugar beet and assess whether beet rust should gain more attention in sugar beet production. There is a need for summing up and completing the knowledge about the spread and survival of the disease to provide an integrated control strategy. The impact on yield and economic return has been assessed to achieve up-to-date information on potential loss due to beet rust. Different control means have been investigated including choice of cultivar and selected biological and chemical control options of different modes of action.

2. Disease cycle and epidemiology

An illustration of the disease cycle of beet rust can be found in Fig. 1. During the growing season urediospores infect plants as the primary source of inoculum (Koike et al., 2007). When a urediospore lands on a susceptible plant, it infects by germinating through the stomata (Littlefield, 1981). The urediospores constitute the repeating and epidemic stage of the disease as they can infect new tissue and produce new uredia and urediospores. Wind and rain are reported to spread the spores (Asher and Hanson, 2006), but the distance they can travel has not been measured. At the end of the growing season, when temperature and humidity decrease, telia with teliospores develop (Pozhar and Assual, 1971). Teliospores are the main overwintering structure (Littlefield, 1981; Pozhar and Assual, 1971). If the winter is mild, the disease might be entirely continued by means of urediospores (Duffus and Ruppel, 1993) or probably also by perennial aecidial mycelia (Gäumann, 1959). The sexual stage of the fungus is initiated when the diploid teliospores germinate in spring with a metabasidium, from which haploid basidiospores are formed (Pozhar and Assual, 1971). Teliospores cannot infect new tissue, but the basidiospores released can germinate and infect young leaves directly through the cuticula and epidermis (Littlefield, 1981; Asher and Hanson, 2006). From the basidiospores, a mycelium is formed and pycnia containing haploid pycniospores are formed. The pycniospores fuse with receptive hypha and initiate the dikaryotic phase. Spores from different pycnia mate when they are transferred by splashing raindrops, by the rubbing of leaves together in the wind, or by insects attracted to the honeydew covering the pycniospores (Littlefield, 1981; Pozhar and Assual, 1971). From the fusion of pycniospores and receptive hypha, an aecium is established, and the aeciospores produced can infect new tissue, typically through the stomata (Littlefield, 1981). From this infection, uredia with urediospores are produced, and the disease cycle is complete. Although *U. beticola* is autoecious and has no alternate host, many of the stages might in practice rarely be present on sugar beet. Sugar beet is a biennial plant grown as an annual spring crop to avoid bolting during vernalisation during winter (Elliot and Weston, 1993). More stages might be completed on overwintering seed beets and sea beets.

The survival of the pathogen during winter in the absence of a host is mainly attributed to teliospores, which are covered by a thick epidermis (Koch, 1986). According to Pozhar and Assual (1971) teliospores of *U. beticola* can survive on the soil surface for a year. Apart from being a medium for overwintering, teliospores can transmit the disease in commercial sugar beet production through infected seeds. The pathogen was earlier described to survive on beets stored in the ground and overwintering seed beets (Punithalingam, 1968). Today sugar beets are no longer stored in the ground in Denmark, and crops for seed production have been moved to Southern Europe. The disease is not seed-borne in a strict sense as it is not carried in the embryo (McKay, 1952), but teliospores on seed clusters can be carried with the seed lot and continue the disease or spread it to new areas (Emdal and Foldoe, 1979). Infected seeds from Europe are considered responsible for the spread of the disease to Canada (Newton and Peturson, 1943) and India (Agarwal et al., 2006). The teliospores can remain viable for two years under store house conditions (Agarkov and Assual, 1963).

There is yet no knowledge of genetic variation within *U. beticola* and possible differences in virulence. Because the teliospores are described as the primary overwintering structure, it could be speculated that genetic recombination occurs regularly; however because urediospores can also overwinter, the disease is able to continue without genetic recombination. Wilson and Henderson (1966) furthermore described that aecia are uncommon in Europe but they have been observed in Denmark in recent years on overwintering beets (Lisa Munk, 2017;