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REVIEW

An overview of the *Sclerotinia sclerotiorum* pathosystem in soybean: impact, fungal biology, and current management strategies

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Abstract

Sclerotinia stem rot (SSR), caused by *Sclerotinia sclerotiorum*, is one of the most important diseases of soybean. Disease management is complicated by the long-term survival of sclerotia in the soil and the absence of resistance in elite, commercial cultivars. Furthermore, the lifecycle of *S. sclerotiorum* in soybean fields is highly dependent on weather conditions, leading to a highly sporadic occurrence of the disease over seasons and an aggregated distribution within fields. Management relies on a multi-pronged approach of combining partially resistant cultivars with cultural practices, such as altering row spacing and planting population, along with chemical control. These control measures are constrained by economic trade-offs, incomplete efficacy of chemicals, and a lack of understanding of application timing for fungicides. Newer tools have been developed to improve management, such as disease prediction models that can assist farmers in making decisions about fungicide application. This review aims to introduce the *Sclerotinia* pathosystem in soybean, while covering the complicated biology of *S. sclerotiorum* that leads to the need for integrated management by soybean farmers.

Keywords *Glycine max* · Soybean · *Sclerotinia sclerotiorum* · White mold · Sclerotinia stem rot · Disease management

Introduction to *Sclerotinia sclerotiorum* (Lib.) de Bary

History and impact on soybean

Sclerotinia sclerotiorum (Lib.) de Bary, the soilborne causal agent of Sclerotinia stem rot (SSR), is a devastating fungal pathogen affecting more than 400 host species globally (Boland and Hall 1994). These species include important field, vegetable, fruit, ornamental, tree, shrub, and numerous weed crops (Saharan and Mehta 2008). While virtually all dicotyledonous crops may become infected, the fungus also infects some monocotyledonous hosts, such as onion and tulip (Bolton et al. 2006). The species was initially described as *Peziza sclerotiorum* by Madame M.A. Libert (1837), until Fuckel described the genus *Sclerotinia* and renamed the fungus *Sclerotinia libertiana* (Fuckel 1869). The genus *Whetzelinia* was also proposed by Korf and Dumont (1972), however, the current accepted *Sclerotinia sclerotiorum* binomial was eventually cited according to the International Rules of Botanical Nomenclature by de Bary (1887) and Massee (Purdy 1979; Saharan and Mehta 2008; Wakefield 1924). The distribution of *S. sclerotiorum* covers 95 countries and almost every continent, including Africa, Asia, Australia, Europe, North America, and South America (Saharan and Mehta 2008). The pathogen thrives particularly well in cooler moist regions (temperate areas) such as the Great Lakes Region of the USA. In the United States, *S. sclerotiorum* has been reported in 44 states throughout the northern, southern, central, eastern, and western regions (Saharan and Mehta 2008).

In soybean, SSR is a severely yield-limiting disease when favorable environmental conditions are met. Yield reductions are caused by reduced seed number and weight (Hoffman et al. 1998; Danielson et al. 2004) resulting from the girdling of stems and disruption of xylem and phloem. Additionally, the presence of sclerotia mixed in seed may result in lower grain prices, and SSR infection within seed pods may lower oil content and reduce seed germination (Hoffman et al. 1998; Mueller et al. 1999). In Argentina, the Northern Pampean region's cool, moist climate is ideal for SSR, and most yield losses are attributable to *S. sclerotiorum*. In 1998, yield loss

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from SSR was estimated at over 423 million kg, the second-greatest loss outside of the United States on record (Wrather et al. 2001). Additionally, in South America, Brazil faces production challenges caused by SSR in southern Paraná, Santa Catarina, Rio Grande do Sul, and the high plains of central Brazil (Wrather et al. 2001). Sclerotinia stem rot is a primary disease of *Brassica napus* in China (Ma et al. 2009) and causes losses on soybean in some regions of the country (Wrather et al. 2001).

In the United States, SSR ranked in the top ten yield reducing diseases of soybeans in 2000, 2004, 2006, and 2009, and continues to significantly contribute to soybean yield reduction (Allen et al. 2017; Koenning and Wrather 2010; Wrather and Koenning 2009). From 2010 to 2014, SSR was responsible for almost 2.8 million metric tons of yield loss in soybeans, which cost growers $1.2 billion USD according to market prices (Allen et al. 2017; USDA-NASS 2017). In 2011 in the Great Lakes region, 94% of the yield losses due to SSR occurred in this region, according to the United Soybean Board; producers lost a corresponding ~$138 million according to 2011 market values (USDA-NASS 2017). The continued global impact of SSR underscores a dire need for advancements in management of the disease.

General biology and infection strategies of *Sclerotinia sclerotiorum*

Sclerotinia sclerotiorum taxonomy and biology

Sclerotinia sclerotiorum belongs to the kingdom Fungi, phylum Ascomycota, class Discomycetes, order Helotiales, family Sclerotiniaceae, and genus *Sclerotinia*, which has been defined to include only species which produce tuberoid sclerotia not incorporating host tissue, developing an apothecial ectal excipulum composed of globose cells, and not producing a disseminative conidial state (Kohn 1979; Saharan and Mehta 2008). In other words, *Sclerotinia* spp. have thick, tuber-like sclerotia (survival structures) which do not contain host tissue, produce apothecia (ascocarp) containing a cup-shaped layer of asci which is situated near the outer-surface, and do not produce conidia (asexual spores). Since no asexual conidia are produced, *S. sclerotiorum* is typically identified by its hyaline, septate, multinucleate hyphae and white to tan mycelium (Bolton et al. 2006). *S. sclerotiorum* does produce sexual ascospores through homothallic, or self-fertile, reproduction *via* a single *MAT* locus which contains both domains encoding the *MAT1–1* and *MAT1–2* mating type genes (Amselem et al. 2011).

The fungus persists in the soil as either mycelium or sclerotia, which are melanized survival structures composed of compact mycelia and may remain viable in the soil for up to 8 years (Adams and Ayers 1979; Willetts and Wong 1980). During this time a conditioning period (at least 8 weeks at 8–16 °C) is typically required prior to apothecial germination (Dillard et al. 1995; Phillips 1986). After appropriate conditioning, and given sufficient light, moisture (near saturation), and temperature (10–25 °C) stimuli (Abawi and Grogan 1979; Sun and Yang 2000), either myceliogenic or carpogenic germination can occur. Myceliogenic germination produces infective hyphae (Willetts and Wong 1980). Carpogenic germination results in the production of apothecia and asci containing hyaline ascospores (sexual spores). Millions of ascospores (covered in mucilage) are ejected and carried *via* the wind to infect susceptible plant tissue (Abawi and Grogan 1979; Bolton et al. 2006; Willetts and Wong 1980). Ascospores are forcibly discharged from apothecia beneath the soybean canopy, at a rate of 1600 spores/h under optimal conditions (Clarkson et al. 2003). Necrotic or senescing tissues, such as flowers, typically serve as a nutrient source to initiate ascospore germination (Abawi and Grogan 1979). Mycelium can then penetrate the cuticle using enzymes, mechanical force, or through stomata (Bolton et al. 2006; Lumsden and Dow 1973). As disease progresses, plants become reservoirs of newly formed sclerotia which become primary sources of inoculum in subsequent years.

Sclerotinia stem rot cycle on soybean

Sclerotinia sclerotiorum overwinters as sclerotia, which are dormant, soilborne survival structures that are resistant to prolonged periods of freezing and thawing (Grau and Hartman 2015). Sclerotia often require a conditioning period (at least 8 weeks at 8–16 °C) prior to apothecial germination (Dillard et al. 1995; Phillips 1986). Carpogenic germination of sclerotia also requires soil water potential of at least −100 kPa for one to 2 weeks, while temperatures of 15–25 °C, for 27–34 days, are optimal (Clarkson et al. 2004), and germination is enhanced by scarification (Garg et al. 2010). Sclerotia of isolates from temperate climates tend to germinate carpogenically more readily when formed at 10 °C than those from warm climates, but isolates from warmer regions may germinate more readily, after a period of cold conditioning, as observed with cultures formed at 25 °C (Huang et al. 1998). As such, adaptations in *S. sclerotiorum* have allowed for an expanded range of the pathogen. Once sclerotial conditioning is achieved, apothecia will germinate from sclerotia present in the top 2–3 cm of soil (Abawi and Grogan 1979). Apothecia produce ascospores which are ejected and carried *via* the wind to infect nearby flowers and pods. Ascospores produced from *S. sclerotiorum* apothecia are the primary source of inoculum for infection by this fungus in soybean (Abawi and Grogan 1974; Grau and Hartman 2015; Peltier et al. 2012; Saharan and Mehta 2008). Conditions of 15–25 °C and 2–4 h of leaf wetness allow for ascopsore germination (Young et al. 2004) and subsequent infection if these conditions coincide with flowering in soybean. Once ascospores germinate on the plant

surface, *S. sclerotiorum* takes advantage of its large lytic repertoire and uses mechanical force *via* compound appressoria and infection cushions (Davar et al. 2012; Lumsden and Dow 1973; Lumsden 1979) to gain entry into the host pant, or it enters directly through stomatal openings. During this process, the virulence factor, oxalic acid (OA), contributes to the pathogenicity of this fungus by thwarting defenses and manipulating the host redox environment, leading to cell death and disease establishment (Kabbage et al. 2013, 2015; Kim et al. 2008; Williams et al. 2011). Oxalate also leads to stomatal opening during infection which increases the transpiration rate, decreases biomass, and, thus, contributes to wilting (Guimaraes and Stotz 2004). Stem colonization and maceration also cause wilting and death of the plant and, eventually, formation of sclerotia within the colonized stem tissues and on the host surface (Guimaraes and Stotz 2004). Significant stem colonization can drastically reduce yield-potential (135–336 kg ha^{-1} for every 10% increase in disease severity) (Grau and Hartman 2015). Typical symptoms are water-soaked tan or brown lesions and wilting, with obvious signs of *S. sclerotiorum* including presence of fluffy, white mycelia and black sclerotia on or in plant tissue (Bolton et al. 2006). Disease development is favored by cool (temperatures below 28 °C) and moist conditions (continuous surface wetness for 40–112 h) (Boland and Hall 1988a). Conducive conditions, however, must coincide with apothecial germination and soybean flowering for SSR development.

Sclerotinia sclerotiorum population structure

While overwintering sclerotia may myceliogenically germinate in the soil, the millions of ascospores generated from the apothecial sexual fruiting body are typically the primary source of SSR inoculum (Peltier et al. 2012; Willetts and Wong 1980). The limited dispersal of ascospores (Ben-Yephet and Bitton 1985) and homothallic life cycle of *S. sclerotiorum* (Willetts and Wong 1980) support regional clonality. Individual clones, however, may be more widely distributed by seed-borne transmission of sclerotia (Hartman et al. 1998). As a result, *S. sclerotiorum* clones are considered highly dispersive, and agricultural populations may contain a conglomeration of different clones (Anderson and Kohn 1995).

Predictably, diverse isolate populations have been documented in US soybean fields (Aldrich-Wolfe et al. 2015; Koga et al. 2014; Kull et al. 2004; Petrofeza and Nasser 2012). These findings suggest that soybean genotypes should be selected for resistance using appropriately representative isolates. In Brazil, the importance of characterizing *S. sclerotiorum* isolate diversity has been investigated for dry bean resistance evaluations; however, little or no regional variation in aggressiveness was found and/or no interaction between isolate diversity and cultivars was observed (Koga et al. 2014; Lehner et al. 2016a, b; Zancan et al. 2015). As such, resistance evaluations, should account for the regional variation within a pathogen population to ensure the development and release of a durable SSR-resistant varieties.

Diversity of *Sclerotinia sclerotiorum*

S. sclerotiorum isolate diversity has been investigated using growth characteristics, aggressiveness properties, mycelial compatibility groups (MCGs), and the production of the key pathogenicity factor oxalic acid (OA) (Koga et al. 2014; Kohn et al. 1991; Kull et al. 2004). *In vitro* mycelial compatibility is used as an indication of isolate homogeneity and may be used to detect variation within a fungal population (Kohn et al. 1990). Multiple MCGs have been detected in North America and South America (Kohn et al. 1991; Kull et al. 2004). Isolate genotype, however, is not necessarily associated with isolate aggressiveness at the regional level (Kull et al. 2004; Lehner et al. 2016a, b).

The non-specific key pathogenicity factor, OA, largely contributes to the extraordinarily broad host range of *S. sclerotiorum* (Godoy et al. 1990; Maxwell and Lumsden 1970; Noyes and Hancock 1981). The roles of OA in *S. sclerotiorum* infection include pH-dependent regulation of cell wall degrading enzymes (Bateman and Beer 1965), stomatal regulation (Stotz and Guimaraes 2004), and suppression of host defenses (Cessna et al. 2000; Williams et al. 2011; Kabbage et al. 2013). OA production may, therefore, more accurately describe isolate aggressiveness. Indeed, OA production by *S. sclerotiorum* isolates has been previously described, and was found to explain isolate aggressiveness in populations recovered from European red clover (Vleugels et al. 2013).

Current management strategies for Sclerotinia stem rot of soybean

Integrated management

The integrated management of SSR utilizes a combination of cultural, chemical, and biological control practices (Peltier et al. 2012). Some practices may include, crop rotation using non-host crops (Gracia-Garza et al. 2002; Mueller et al. 2002a, 2002b; Rousseau et al. 2007), practicing reduced tillage (Gracia-Garza et al. 2002; Kurle et al. 2001; Mueller et al. 2002a, b), using resistant cultivars (Grau et al. 1982; Hoffman et al. 2002; Kurle et al. 2001), modifying the soybean canopy through seeding rate and row spacing (Jaccoud-Filho et al. 2016; Kurle et al. 2001; Lee et al. 2005), and applying in-season chemical control (Mueller et al. 2004; Peltier et al. 2012; Sumida et al. 2015; Saharan and Mehta 2008) (Fig. 1). Many of these practices manipulate the host environment to be unfavorable for diseases development, such as increasing air flow through the canopy or reducing inoculum development in the field.

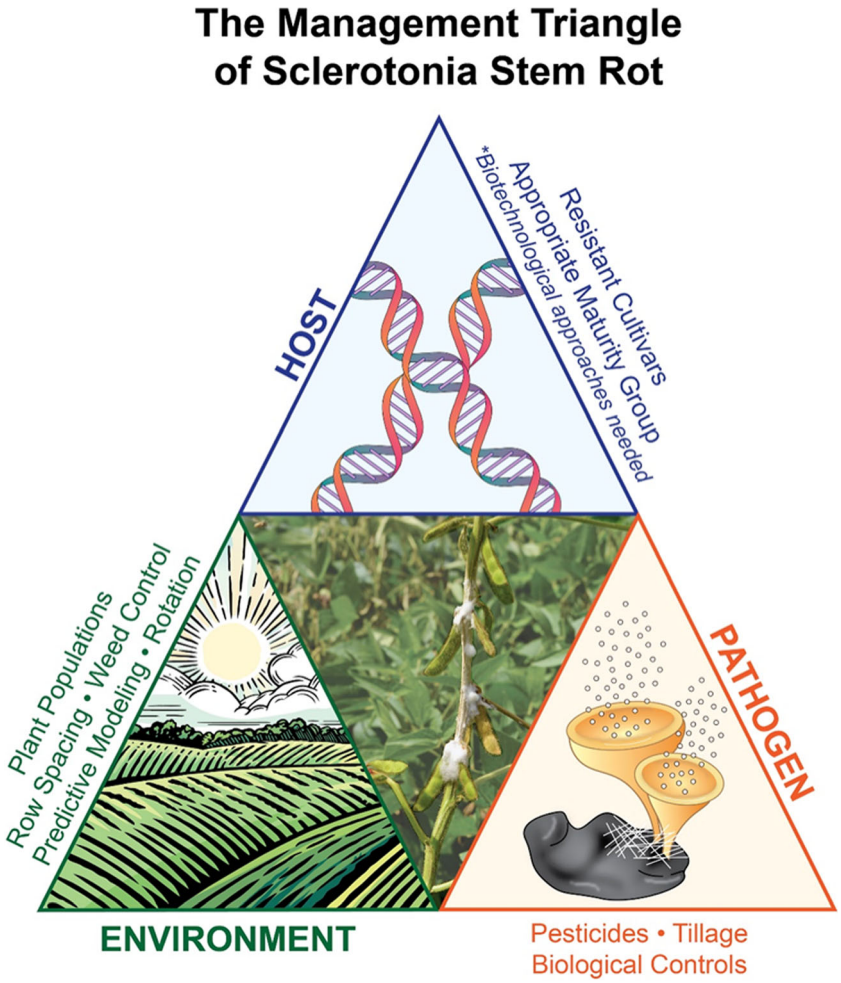

Fig. 1 Management strategies target various facets of the Sclerotinia stem rot disease triangle and may overlap in the part of the triangle they interrupt (Ex: Tillage). Management strategies related to the environment (green), the soybean host (blue), and the pathogen (orange) are illustrated

Specifically, practices such as rotations with non-host plants, tillage, weed control, and cover crops (Peltier et al. 2012) can serve to reduce inoculum. Cover crops, such as small grains (oats, wheat, or barley) function by promoting apothecial germination during the growth of non-hosts, while tillage prevents germination *via* deeply burying sclerotia at a depth that prevents emergence, while no-till strategies avoid pulling inoculum to the surface of soil. Decreased incidence of SSR in no-till fields may also be related to a less dense canopy and exposure to environmental factors such as dry soil (Workneh and Yang 2000).

Environmental conditions that favor apothecial germination can be altered to manage SSR. This includes slowing the closure of soybean canopies, which when closed, facilitate the dark, moist conditions that result in SSR development. Alignment of environmental conditions favoring SSR development with canopy closure and flowering time, can be manipulated with cultivar selection of less bushy, earlier maturity group varieties and later plantings (Kim and Diers 2000). Additionally, plant populations can be decreased below densities that facilitate conditions for SSR development (Kurle et al. 2001 and Lee et al. 2005; < 432,100 soybean ha^{-1}), particularly in fields with a history of SSR. Wider row spacing (≥ 51 cm) can reduce SSR incidence due to longer timing to complete canopy closure (Grau and Radke 1984). Maximum yields in a study in Iowa, were associated with populations of 462,200 plants ha^{-1}, over the course of 3 years, but >95% of the maximum yield was achieved at populations as low as 258, 600 plants ha^{-1} (De Bruin and Pedersen 2008). Therefore, it will be necessary to assess trade-offs between SSR severity and yield when decreasing populations to control SSR.

Cultivar resistance in integrated management strategies

Cultivar selection is one of the most important considerations for SSR management. Disease control is limited by the lack of complete resistance to SSR, however, several partially resistant soybean genotypes have been identified (Boland and Hall 1987; Grau et al. 1982; Kim and Diers 2000). The use of resistant cultivars, particularly in fields with a history of SSR, is a powerful strategy to reduce the incidence and severity of SSR. Integrated management strategies that combine resistance with management practices to reduce inoculum

and avoid conditions favored by SSR development, can help to reduce infection and aid in the consistency of resistance. As demonstrated in variety trials in Wisconsin, resistance rankings vary between the Northern and Southern portion of the state with one cultivar having a mid-ranked 10% incidence in the southern part of the state, but the highest incidence at 68% in the northern portion of the state (Conley et al. 2017). Chemical control plans may also be altered with resistant varieties. Huzar-Novakowiski et al. (2017) found that chemical treatments had a greater impact on disease reduction and yield increase when moderately susceptible *versus* moderately resistant cultivars were planted. In the same study, SSR was reduced where applications of boscalid were used, but yield benefits were inconsistent.

Chemical control

As no complete resistance is available in commercial cultivars, in-season management relies heavily on chemical control targeted at protecting the flowers from *S. sclerotiorum* ascospore infection (Peltier et al. 2012). As the primary infectious unit, ascospores land on senescing flowers and infections most frequently initiate on the first one to five nodes of the main stem (Boland and Hall 1988a, b). Therefore, spray regimes are most effective when targeting the flowering window, particularly at the R1 (beginning bloom) growth stage (Mueller et al. 2004). In greenhouse studies, benomyl, thiophanate methyl, and vinclozolin have all demonstrated suppression of *S. sclerotiorum* signs and symptoms on leaves (Mueller et al. 2002a, b). Most fungicides used in SSR control are classified as methyl benzimidazole carbamates (MBC) (*e.g.* thiophanate-methyl), succinate dehydrogenase inhibitors (SDHI) (*e.g.* boscalid), demethylation inhibitors (DMI) (*e.g.* flutriafol, prothioconazole, tetraconazole), and quinone outside inhibitors (QoI) (*e.g.* fluoxastrobin, picoxystrobin, trifloxystrobin) (Armando et al. 2015; Di et al. 2016; Huzar-Novakowiski et al. 2017; Liang et al. 2015; Peltier et al. 2012). Fluazinam, an uncoupler of oxidative phosphorylation (uncouplers), has also been found to effectively inhibit *S. sclerotiorum* (Liang et al. 2015).

These fungicide classes inhibit *S. sclerotiorum* growth and development in a variety of ways. The MBC fungicides inhibit fungal cell division whereas SDHI, QoI, and uncoupler fungicides interfere with the electron transport chain inhibiting cellular respiration and energy production (Peltier et al. 2012). DMI fungicides, on the other hand, inhibit sterol biosynthesis which results in abnormal fungal cell wall development (Peltier et al. 2012). Fungicides inhibiting energy production will effectively inhibit spore germination whereas those targeting cellular structure or growth, simply slow fungal growth. In addition to these fungicides, the herbicide lactofen has also been identified for SSR management; herbicides impact canopy development and promote systemic resistance, both of which inhibit infection by *S. sclerotiorum* and subsequent development of SSR (Peltier et al. 2012; Dann et al. 1999).

Chemical sprays may be ineffective and inconsistent when the incidence of SSR is high. The effectiveness of fungicides differs based on the chemical used and application timing in north-central regional studies (Byrne and Chilvers 2016; Huzar-Novakowiski et al. 2017; Mueller et al. 2016; Smith et al. 2015). Furthermore, field trials demonstrate effective control against *S. sclerotiorum* by several pesticides, but they do not provide complete control, and incidence after chemical sprays can range from 0 to 60% in plot trials (Mueller et al. 2002a, b, 2004). Application coverage is also important, with flat-fan spray nozzles with high-fine to mid-medium droplets (200–400 μm) being the most effective. Poor coverage, fungicide rate, mixing, sprayer calibration, and environmental conditions can all effect fungicide efficacy. Coverage is influenced by the density of the canopy, droplet size, and spray volume (Derksen et al. 2008). Additionally, the lactofen formulation used in Dann et al. (1999) had phytotoxic effects that resulted in a 10% yield decrease in the absence of SSR. Lactofen can also cause phenotypic effects such as stunting and discolored, malformed leaves (Huzar-Novakowiski et al. 2017).

Research avenues persist for enhanced biological controls

Biological control can be used as part of an *S. sclerotiorum* management plan for soybeans. *Coniothyrium minitans* is a known fungal pathogen of *S. sclerotiorum,* (Huang and Hoes 1976) that parasitizes hyphae and sclerotia, and it is the most widely deployed biological control organism for the control of SSR. *Coniothyrium minitans* can decrease sclerotia up to 95%, and Zeng et al. (2012) observed greater SSR DSI reductions in soybean using *C. minitans* (68%) compared to *Streptomyces lydicus* (43.1%) or *Trichoderma harzianum* (35%); although, all have been shown to effectively reduce SSR severity and increase seed yield (Sesan and Csep 1995). Field trials using biological control agents have been limited, and various strains of *Trichoderma spp*. have been found to have differential abilities to parasitize *S. sclerotiorum* (Haddad et al. 2017). More studies are needed to measure the efficacy and consistency of biological control bacterium, fungi, and potentially other micro-organisms to control SSR in soybean.

Epidemiological modeling to improve management strategies

Soybean flowering, apothecial germination, and conducive weather conditions must occur simultaneously for SSR development. Due to this complex array of factors, fungicide applications are often ineffective, and even unnecessary. *Sclerotinia* forecasting models have been developed for several crop systems, such as peanut, carrot, lettuce, and canola to

asssit in making the decision to spray fungicide. Under controlled environment conditions, ascospore density (>87 spores cm^{-2}), temperature (21.7 °C optimal), and relative humidity (80–100%) were used to predict *S. sclerotiorum* infection of lettuce (Clarkson et al. 2014). In lettuce, 30–50 days at temperatures of 18–20 °C was optimal for conditioning sclerotia for apothecial germination (Clarkson et al. 2007). These findings suggest that temperature and moisture over a period of 30–50 days influences apothecial development.

Models incorporating soil temperature (maximum of 24 °C) and moisture (≥20 kPa) were also shown to predict the development of apothecia and ascospores of Sclerotinia rot in carrot (SRC) (Foster et al. 2011). These data were incorporated into a model which also accounted for field history, canopy closure (>95%), and senescing leaves (on 70–80% of plants) to better predict the fungicide applications necessary for disease control. In a two-year field validation study, the model reduced the total number of fungicide applications and achieved equivalent control to a typical calendar-based spray regime (Foster et al. 2011).

Studies in China have investigated correlations between the numbers of apothecia present during the blossom stage, disease incidence, and yield loss (Pan et al. 2001; Saharan and Mehta 2008). Both number of apothecia and disease severity were negatively correlated with yield loss, and a control threshold of three to four apothecia per 9.75 m^2 was established. Apothecial size (approximately 0.5–2.0 mm in diameter) (Grau and Hartman 2015) and aggregated distribution (Boland and Hall 1988b), however, make this model difficult to implement for farmers. A study conducted in the North-Central region of the US used logistic regression to model the prevalence of soybean SSR (Mila et al. 2004). Using total precipitation and air temperature in either April or July combined with regional tillage practices, disease prevalence was modeled at a regional level. This model, however, was not accurate at the field level.

Historically, *S. sclerotiorum* apothecia and SSR incidence were both spatially aggregated and correlated within sectors of soybean fields (Boland and Hall 1988b). More recently, the distribution of SSR has been correlated with apothecia in both canola (Qandah and del Rio Mendoza 2012) and soybean (Wegulo et al. 2000). In both studies, disease incidence decreased as distance from apothecial inoculum sources increased. Furthermore, ascospores were deposited near the apothecia within soybean fields (Wegulo et al. 2000), which supports the relationship between apothecia and disease. Sclerotial load, determined by intensive soil sampling, was not found to describe white mold incidence in bean fields (McDonald and Boland 2004). Apothecial presence, therefore, is a promising candidate to use for SSR risk assessment in soybean fields. In the Great Lakes region, Willbur et al. (2018a) developed SSR risk models using environmental parameters including maximum temperature, mean relative humidity, and maximum wind speed to predict apothecial presence. Models were used in a set of subsequent field validation experiments to test accuracy of prediction of end-of-season disease levels. In those validation efforts in Wisconsin, Iowa, and Michigan models predicted SSR over 80% of the time (Willbur et al. 2018b). Furthermore, sources of weather data were tested, including data from an open-source weather provider, darksky.net. Weather or climate data can be difficult to acquire for site-specific predictions unless an on-site scientific weather station is feasible. Simulated virtual weather data, however, is readily available for most air temperature and moisture variables. Models generated using these publicly available data allow the model to be accessible and functional in virtually any growing location (Magarey et al. 2001). Willbur et al. (2018b) found that data from darksky.net were nearly as accurate as weather from on-site weather stations, however bias was detected in the three weather variables of interest. When bias corrections were included, darksky.net weather data were considered a suitable source to drive SSR prediction models (Willbur et al. 2018b). Plant phenology information and canopy and row-spacing parameters (Fall et al, 2018) have subsequently been combined with these prediction models into a smartphone application that can be used predict the risk of apothecial presence during the soybean bloom period. Thus, timely fungicide applications can be made if weather is conducive or fungicide sprays can be saved if favorable conditions do not exist during bloom. The smartphone application is available on the Android (https://play.google.com/store/apps/details?id=ipcm.soybeandiseasecalculator) and iPhone (https://itunes.apple.com/us/app/sporecaster/id1379793823?mt=8) platforms and called Sporecaster.

Conclusions

Sclerotinia stem rot is a significant disease of soybean in many parts of the world. It is difficult to control due to the complicated and intricate biology of *S. sclerotiorum*. Incomplete management options do exist for soybean farmers, such as adjusting row spacing and decreasing plant population densities. Knowledge about the resistance or susceptibility of cultivars planted and the use of predictive models that consider SSR environmental variables can further enhance SSR management by allowing farmers to better understand when chemical sprays can be used at optimal economic timing. As an effective form of SSR control, resistant cultivars should also be an important consideration in integrated management plans. However, the need for advanced biotechnological approaches is apparent, as complete resistance in soybean would be desirable and eliminate the need for chemical controls or complicated management plans.

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