Understanding Yield Loss and Pathogen Biology to Improve Disease Management: *Septoria Nodorum* Blotch – A Case Study in Wheat

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The estimated potential yield losses caused by plant pathogens is up to 16% globally (Oerke 2006) and most research in plant pathology aims to reduce yield loss in our crops directly or indirectly. Yield losses caused by a certain disease depend not only on disease severity, but also on the weather factors, the pathogen's aggressiveness, and the ability of the crop to compensate for reduced photosynthetic area. The yield loss-disease relationship in a certain host-pathogen system might therefore change from year to year, making predictions for yield loss very difficult at the regional or even at the farmer's level. However, estimating yield losses is essential to determine disease management thresholds at which acute control measures such as fungicide applications, or strategic measures such as crop rotation or use of resistant cultivars are economically and environmentally sensible. Legislation in many countries enforces implementation of integrated pest management (IPM), based on economic thresholds at which the costs due to a disease justify the costs for its management. Without a better understanding of the relationship between disease epidemiology and yield loss, we remain insufficiently equipped to design adequate IPM strategies that will be widely adapted in agriculture. Crop loss studies are resource demanding and difficult to interpret for one particular disease, as crops are usually not invaded by only one pest or pathogen at a time. Combining our knowledge on disease epidemiology, crop physiology, yield development, damage mechanisms involved, and the effect of management practices can help us to increase our understanding of the disease-crop loss relationship. The main aim of this paper is to review and analyze the literature on a

representative host-pathogen relationship in an important staple food crop to identify knowledge gaps and research areas to better assess yield loss and design management strategies based on economic thresholds.

Wheat is one of the most important staple foods worldwide and is susceptible to several important plant diseases. In our article, we focus on *Septoria nodorum* blotch (SNB) or Glume blotch of wheat as an example for a stubble-borne, seed-transmitted disease with a worldwide distribution causing considerable and regular yield losses. In their review on yield losses due to wheat pathogens in Australia, Murray and Brennan (2009) estimated the current annual economic loss due to SNB as high as $\$108 \times 10^6$, with potential costs as high as $\$230 \times 10^6$. The causal fungus, *Parastagonospora nodorum*, is currently serving as a model organism for molecular studies of the intimate relationship between necrotic effector-producing fungal strains and their corresponding susceptibility genes present in wheat cultivars (Oliver et al. 2012). In this paper, we analyze the literature on the biology of this common wheat pathogen, the yield loss it reportedly has caused, and the effect of control strategies to reduce this loss. Based on this analysis, we will evaluate the use of common management practices to reduce disease-related yield loss and identify related research needs.

The Pathogen

Septoria nodorum blotch, also known as Glume blotch in wheat (*Triticum aestivum* L.) (Bergstrom 2010; Oliver et al. 2016), is caused by the heterothallic ascomycete *P. nodorum* (Berk.) Quaedvlieg, Verkley and Crous, comb. nov. (synonymous: *Phaeosphaeria nodorum* [E. Müller] Hedjaroude, *Leptosphaeria nodorum* E. Müller; anamorph: *Stagonospora nodorum* [Berk.] Castellani & Germano, synonym: *Septoria nodorum* Berk.) (Quaedvlieg et al. 2013). *Parastagonospora nodorum* was first described as a pathogen of wheat in 1845 and can attack all aerial parts of its host plant (Baker 1978). On leaves, it forms oval, irregular, light-brown lesions (Fig. 1A), which are easily

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confused with *Septoria tritici* blotch, caused by *Zymoseptoria tritici* (P. Crous; syn. *Mycosphaerella graminicola*, *Septoria tritici*) and tan spot caused by *Pyrenophora tritici-repentis* (Died) Shoem in the field. In fact, these three pathogens can co-occur in the same field and on the same leaf. SNB was the dominant leaf blotch disease on wheat in Europe until the 1980s, when *Z. tritici* started to dominate in most European wheat fields. A disease survey based on a questionnaire from 1990 showed SNB was reported from Canada, the U.S.A., Mexico, Central America, South America, China, Japan, Africa, Russia, Denmark, India, Australia, and New Zealand (Leath et al. 1993). In that study, SNB occurrence was positively correlated with latitude, soil phosphorus, and minimum tillage. A study based on PCR analysis of the Rothamsted Broadbalk experiment archives showed a sharp decline in *P. nodorum* after 1980 and the rise of *Z. tritici*, relating this change to SO₂ emissions (Shaw et al. 2008). Currently, SNB has been reported mostly from the U.S.A., Canada, Western Australia, southern Germany, Switzerland, Latvia, Finland, and Norway. As the disease symptoms can be easily confused between the three leaf blotch pathogens and all three can co-occur in the same field, comprehensive disease management needs to address all three pathogens to define economic thresholds and reduce yield loss in some areas.

Head infections by *P. nodorum* are often seen as dark brown-purple blotches on the glumes (Fig. 1B), thus the name 'glume blotch' (Weber 1922). Leaf and head infections appear to be significantly correlated, while the overall plant disease index (disease intensity of the whole plant) correlates negatively with yield (Nelson et al. 1976). Glume infections can lead to seed infections and subsequently seedling infections after sowing. Infected wheat debris are an important inoculum source for this disease as *P. nodorum* infects the plants by splash dispersal of conidia in rain droplets or through wind dispersed ascospores from fruiting bodies overwintering on the debris. Precipitation is an

important driver for SNB epidemics and the temperature for infection and spread ranges between 5 and 30°C (Rapilly and Skajennikoff 1974). A schematic presentation of the life cycle of *P. nodorum* is shown in Figure 2, while the role of the different spore types is summarized below. More detailed information about the sources of inoculum and how they relate to management practices is given in the later sections.

Conidia of SNB (asexual spores) can spread the infection from infected wheat debris vertically up the plant by splash dispersal, until it reaches the heads. Free water is essential for sporulation (Brennan et al. 1986), supporting the observation that release of conidia was associated with rainfall in the field (Jeger et al. 1981). *P. nodorum* has the potential to produce large amounts of conidia. When conidia were quantified on main tillers of three different wheat cultivars, production of conidia varied between 3×10^3 and 9×10^5 per tiller per year (Shearer and Wilcoxson 1980). The latent period of *P. nodorum* varied between 6 days at 22°C and 36 days at 4°C under water-saturated conditions (Shearer and Zadoks 1972). The latent period correlated negatively with temperature in a growth chamber experiment (Jeger et al. 1984). Nevertheless, latent period was affected by growth stage of wheat; the older the plant, the shorter the latent period, as Leonard (1988) demonstrated in a greenhouse study with four different cultivars, and Shearer and Wilcoxson (1980) showed in a 3-year field study with natural epidemics. Spore production appears to be higher on dead than on living tissue (Rapilly and Skajennikoff 1974), which might delay the epidemic at the start of the season when dead host tissue is limited in fields and accelerate it toward the end of the season, when senescing tissue is more abundant. This might be of significance if there is no local source of inoculum and the epidemic relies on in-flying ascospores. Scharen (1966) observed that *P. nodorum* conidia were produced repeatedly

Fig. 1. Typical symptoms of *Parastagonospora nodorum* on wheat leaves (A) and heads (B).

in the same pycnidia, and new conidia were still being discharged after 7 cycles of wetting, discharging, and drying. These studies suggest that conidia, with their repeated production in high numbers, constitute the driving factor of the epidemic once it is established, only limited by the lack of free water during the season and increasing as the average temperature and senescing tissue increase during the season.

P. nodorum requires two opposite mating types for sexual reproduction. Ascospores form in pseudothecia. These types of spores can be spread over long distances with air currents. Therefore, they can initiate SNB epidemics in areas without wheat debris or infected seeds present, thus reducing the effect of rotation, plowing, and seed treatment. In the event of early ascospore discharge and under conducive weather conditions, they can have a decisive role in the level of yield loss occurring. However, presence of the two mating types in a region does not mandate a significant role of ascospores as primary inoculum source (Bennett et al. 2007). Timing of ascospore arrival in previously disease-free fields can be critical for disease development and fungicide sprays. Seasonal patterns of ascospore production and release were observed in several areas (Mittelstädt and Fehrmann

1987), while other researchers captured ascospores of *P. nodorum* continuously during the season (Bathgate and Loughman 2001; McFadden and Harding 1989) or throughout the year when temperatures were above freezing (Arseniuk et al. 1998a, b). Rapilly et al. (1973) observed that ascospore release in the field followed rain events and continued 6 to 10 h after the rain, while others concluded that other forms of moisture such as mist and dew were sufficient to release the spores (Bathgate and Loughman 2001; Sanderson and Hampton 1978). In laboratory studies, pseudothecia development took 3 to 4 months (Halama and Lacoste 1992), but once pseudothecia had developed on wheat debris, ascospores were discharged for at least 8 months thereafter in the presence of free water (Sanderson and Hampton 1978). The regional differences in ascospore release patterns are important in evaluating the effectiveness of debris management and the need for additional fungicide applications and dosage. Studies from a 2-year field trial suggested that tissue type was influencing the degree of pseudothecia formation, as more pseudothecia were found on wheat spikes than on basal plant tissue (Cowger and Silva-Rojas 2006). If ascospores arrive continuously throughout the season, the epidemic is likely to have an early start even in the absence

Fig. 2. General life cycle of *Parastagonospora nodorum* (original drawing by Hermod Karlsen, from Brodal et al. [2009]).

of any wheat residues, while late arrival of them might reduce the need for fungicide application substantially, if no other sources of inoculum are present. The time and conditions needed for developing mature ascospores could play a critical role in starting the SNB epidemic in disease-free fields planted with healthy seeds and could be integrated in disease prediction and yield loss models.

Yield Loss in Wheat

How the SNB epidemic affects yield and to what extent is the driving question for disease management. Yield losses are mostly due to the reduction of the photosynthetic active leaf area, leading to reduced yield quantity and quality, which includes shriveled grains and reduced thousand kernel weight (TKW), but SNB can also affect the kernels directly as it attacks the glumes and awns. In order to estimate yield loss and establish an action threshold for disease management, we need to understand what parts of the crop contribute to yield over time, how SNB is affecting these plant parts during the season, and how the host is compensating for these effects.

Yield determinants. Photosynthetic active leaf area above the flag leaf node at flowering is supposed to relate directly to grain yield in wheat (Lupton 1972; Simpson 1968; Yoshida 1972). However, the contribution of different plant organs to total photosynthetic area changes over time and with wheat cultivar (Birecka 1966). The results from a climate chamber study with ^{14}C showed that the contribution from flag and secondary leaves to photosynthesis during the first 3 weeks after flowering was actually relatively small (5 to 10%), while photosynthetic activity of the stem was declining rapidly and photosynthesis of the heads was increasing (Fig. 3A) (Birecka and

Dakić-Włodkowska 1963). In a field study, Lupton (1972) also used ^{14}C to determine carbon uptake from heads and leaves (but not from stems) and showed that heads and flag leaves contributed most to grain yield after flowering (Fig. 3B). Even though contribution from the different plant parts varied significantly between the two studies, heads and flag leaves appear to be the most important parts contributing to grain yield during the grain-filling period, which starts after flowering. It appears obvious that a targeted fungicide application would need to protect heads, flag leaves, and possibly stems until the end of grain filling, pointing to the need for late sprays, especially when weather conditions during grain filling are likely to be conducive for further disease development.

Yield loss due to SNB. In agreement with the studies above, several SNB trials showed that TKW and yield quantity were strongly affected by leaf and head infections (Brönnimann 1969; Cooke and Jones 1970; Nelson et al. 1976; Scott and Benedikz 1977). Verreet et al. (1987) observed in their greenhouse study that SNB infections of the leaves actually had a stronger effect on yield than on head infections. However, all studies agree that SNB infections of heads and leaves affect grain yield to different extents depending on the isolates, wheat cultivars, and growth conditions.

The studies so far focused on the effect of SNB infection during grain filling; however, yield does not only depend on the filling of the grain after flowering, but also on the biomass produced before flowering and the number of tillers and heads developed. While several studies (Holmes and Colhoun 1974; Jones and Odebunmi 1971; Verreet et al. 1987) showed that yield loss peaked after infections between flag leaf emergence (GS 37) and booting (GS 50 to 59), Cooke

Fig. 3. Contribution of different plant parts to total photosynthesis in wheat over time after anthesis (**A**, data from Birecka and Dakić-Włodkowska [1963]; **B**, data from Lupton [1972]). Whole plants were exposed to $^{14}\text{CO}_2$ for 24 h in an airtight enclosure, before different plant parts were sampled to determine radioactivity as an indicator for photosynthetic activity.

and Fozzard (1973) observed a consistent increase in yield loss with inoculations after flag leaf emergence, while Pirson (1960) registered a continuous decline in yield loss with inoculations after booting (Fig. 4). These studies, based on SNB field trials, suggest the time for optimal fungicide application to be sometime between flag leaf emergence and booting or even flowering (GS 60 to 69). As the window of optimal application time based on these different studies is rather wide, we need to integrate the actual field-based infection risk during this time period with host resistance and cropping history to make meaningful recommendations for fungicide applications.

After considering the organs that need to be protected and the timing of possible fungicide applications, it would be good to define damage thresholds, the amount of disease or loss of green leaf tissue that would lead to a significant yield or grain loss that justifies fungicide applications. Studying two wheat cultivars, Trotter et al. (1975) showed that 20% of the flag leaf had to be necrotic due to SNB before TKW was significantly reduced. However, 20% SNB severity on the flag leaf seems to be a very high threshold for fungicide applications and this is likely to change with cultivar and with the pathogen population present. As a matter of fact, Krupinsky and Scharen (1973) showed that some isolates of *P. nodorum* affected apparent photosynthetic rate differently depending on the organs inoculated. Extension services have developed economic damage thresholds based on empirical and experimental data. Depending on the region, the U.S. extension services are currently recommending fungicide applications when 25 to 50% of leaves on a plant are infected with SNB, but first application is not recommended before the flag leaf stage. In Norway, fungicide application is recommended by the fungicide companies as soon as SNB symptoms become visible. In Germany, 12% incidence at GS 39 to 49 is considered the economic threshold for SNB management, while Switzerland recommends spraying when 20% of the upper four leaves are infected. The wheat disease management guide from the Agriculture and Horticulture Development Board (AHDB) for cereals and oilseeds 2016 in the U.K. recommended farmers to spray at GS 32 and at GS 39 to control SNB, but the risk for SNB infection in this region was described as low. It seems that the economic threshold and consequentially the action threshold is lower in Europe, with intensive wheat cultivation and high yields, than in the U.S., where fungicide applications are not recommended before a quarter to half of the leaves are infected.

As easy to apply as economic damage thresholds might seem, they are often based on a generalized assumption of how much yield loss is caused by how much disease and how much loss would justify a fungicide application in a certain region. This decision depends on the prices of produce and pesticides (Zadoks 1985), which can

change substantially over time and space, but also on the individual economic situation at the farm level. A farmer might prefer to make the decision on the economic injury threshold based on a reliable yield loss-disease prediction and his own economic assessments, than rely on generalized economic assumptions.

What we would need for yield loss prediction is a robust relationship between some measure of disease intensity and yield loss at a certain developmental stage that is general enough to be applied over several years, cultivars, and SNB isolates present.

Infection-yield loss relationship. Oliver et al. (2008) hypothesized that in contrast to biotrophic pathogens, the SNB-wheat pathosystem should show a very direct relationship among symptoms, biomass, and yield loss, because *P. nodorum* penetrates the epidermis rapidly, has a short secondary infection cycle, and lacks a quiescent phase. In their 1-year field trial, they found that the correlation between flag leaf symptoms and relative grain weight (RGW) improved from the first disease assessment at 150°C thermal days to the second assessment at 220°C thermal days. Estimation of *P. nodorum* biomass by quantitative PCR on infected leaves collected at those same two time points produced an even higher correlation with yield loss than the SNB score (Oliver et al. 2008). The highest correlation between disease severity and yield loss (TKW) in a 1-year trial at Cardiff, U.K., was seen when average SNB infection of the two last leaves were assessed at GS 69 and GS 71 to 75 (kernel watery to milky ripe) for two different cultivars (Cooke and Fozzard 1973). It would have been interesting to see if the relationship between SNB and yield loss was similar between different years.

Infection of the flag or penultimate leaf appeared also to be a good predictor of yield loss in field trials conducted in Western Australia (Bhathal et al. 2003). Wheat plants were inoculated with *P. nodorum* spore suspensions after crop establishment and assessed six or seven times after GS 31 (first node visible). SNB severity on either flag or penultimate leaf during GS 73 to 77 (kernel at milk stage) explained 36% of yield variation (TKW) in one year, while it explained 71.4% of variation in yield the next (Bhathal et al. 2003), showing the great variation between years and the importance of the seasonal weather factors on the relationship.

Gilbert and Tekauz (1993) studied the relationship between disease severity and yield components also in a 2-year field trial, using 12 different wheat cultivars in Canada and assessing at GS 65 to 75 in one and at GS 75 to 85 in the other year. While no effect on kernel number was observed, TKW was reduced significantly for all wheat cultivars inoculated with SNB in the second year. There was no consistent relationship between leaf necrosis on the flag and penultimate leaves during GS 75 to 85 and yield variation (Gilbert and Tekauz 1993).

Nelson et al. (1976) developed a septoria disease index (SDI) by assessing all leaves on the plant, as well as heads at GS 75 based on 30 to 33 soft winter wheat cultivars over 2 years in the U.S. They found a simple negative relationship of SDI to yield of 0.61, which meant that with every increasing unit of SDI, yield was reduced by 0.61% (Nelson et al. 1976). However, it is not clear from their results if the relationship was similar in both years or if it was simply the average of the two trial years. In Finland, Karjalainen et al. (1983) reported a negative correlation of 0.26 to 0.34 between yield and SNB severity on flag leaves at least 2 weeks after anthesis. In Great Britain, King et al. (1983) found a direct relationship between SNB severity on the flag leaf at GS 75 and yield loss to be approximately 1:1, showing that 1% SNB severity on the flag leaf at GS75 lead to 1% yield loss. In Norway, the relationship between severity of SNB on the last three leaves at GS 70 to 85 and yield loss varied between 0.5 and 1.2 in a 3-year trial using one spring wheat variety at one location (Ficke et al. 2016).

The studies above showed that infection of the upper leaves after flowering does affect yield, but also that the relationship is highly variable between locations/environments and cultivars. Despite the efforts taken to relate yield loss to SNB severity or fungal biomass at a certain time, a robust relationship between disease intensity and potential yield loss that could be used to establish a fungicide application threshold remains elusive at this stage. Alternatively, mathematical models could be used to account for some of the variation to predict yield losses based on different factors including disease intensity and its damage profile.

Fig. 4. Relationship between yield loss and inoculation at different developmental stages (GS) of wheat based on different studies published in the literature.

Yield loss models. Mathematical models to predict yield losses based on disease intensity can be divided into three groups: the critical-point model, which is based on one assessment during the growing season; multiple-point models, which are based on multiple assessments during the season; and the area-under-the-curve model (James 1974). James (1974) reviewed attempts to relate disease intensities at a given time point to yield loss and argued that the critical-point model neglects the importance of the varying infection rate over time and the shape of the disease progress curve in determining yield loss. The multiple-point model, on the other hand, accounts for the disease dynamic and varying impacts on yield. This model would allow yield loss estimates based on disease assessments at any given time during the season (James 1974). The area-under-the-curve model distinguishes between different epidemics, which have the same disease intensity at a given point in time, but it cannot apply a different weighing to disease relative to the growth stage of the crop. The different weighing of disease is important as yield is a product of biological processes that are affected differently by diseases at different times during the growing season (James 1974).

Waggoner and Berger (1987) claimed that yield was not correlated with disease intensity, but with the integral over days of the healthy leaf area index; the healthy area duration (HAD) or the healthy area absorption (HAA). They defined yield loss in terms of leaf area duration (LAD) of a healthy standard and the HAD of the infected crop (Waggoner and Berger 1987). Bryson et al. (1997) showed that HAD of wheat varied strongly between years and suggested the use of the healthy area absorption (HAA) index to more accurately estimate yield. However, HAD and HAA both relate to leaf area duration and do not include photosynthetic active area of the stem or the flower head, which according to Lupton (1972) and Birecka and Dakić-Włodkowska (1963), also play an important role in yield development.

A more general approach was undertaken by Savary and Willocquet (2014) in their crop loss model for wheat (WHEATPEST), where diseases, insects, and weeds (pests) were considered to affect the crop through generalized damage mechanisms simultaneously under different production situations. In this model, the attainable growth of the crop depended on the production situation and was linked to the injury patterns (such as “light stealer,” “assimilate sapper,” “photosynthetic rate reducer,” and “tissue consumer”) from different pests, including SNB. The difference between the attainable yield and the actual yield (Fig. 5) could then be considered under different management regimes to evaluate appropriate strategies to reduce crop loss under a certain production system (Savary and Willocquet 2014). The simulation model was not intended to determine disease severity thresholds or timing of fungicide applications, but to evaluate different crop protection strategies. The last paragraphs have focused on which plant part was important for grain yield development and how the disease might affect the different stages of crop development. This rather neglected area is critical to determine disease management thresholds for the farmer to avoid his or her potential yield and consequentially economic loss. However, deciding on when and what to choose from the disease management toolbox does not only depend on the cost of yield loss itself, but also on the actual tools available, their costs, and their effectiveness to control SNB. Today, only a few decision support systems include crop loss cost estimation based on disease development, making it difficult to give farmers economic advice for their crop on the field level. The following paragraphs summarize the most common management strategies used in controlling SNB of wheat and the use of disease prediction models for SNB.

Disease Management Tools

Several complementary practices can reduce pathogen infections, such as the use of healthy seeds and/or applying fungicide seed treatments, wheat residue management, planting moderately resistant cultivars, and applying effective fungicides. Removing alternative hosts has been considered, since many weeds can serve as alternative hosts for *P. nodorum* within and outside the wheat fields (Obst 1971; Weber 1922; Williams and Jones 1973). However, its potential contribution to an epidemic and consequently to yield loss has not been

established. Together, these approaches are targeting different inoculum sources and stages in the epidemic and have shown different effectiveness in reducing yield loss from SNB.

Seed treatment. Seedborne inoculum can introduce the pathogen into uninfected fields, contribute to an even distribution of disease foci throughout a field (Shah and Bergstrom 2000; Shah et al. 1995), increase the risk for an early start of an epidemic (Cunfer 1978; Shah and Bergstrom 2002), and transmit *P. nodorum* to the next generation of seed (Shah et al. 1995). The frequency and level of seed infection detected in laboratory tests and that of diseased seedlings after sowing in the field were positively correlated in some studies (Babadoost and Hebert 1984; Hewett 1975; Jenkyn and King 1977; Luke et al. 1985, 1986; Shah et al. 1995), but not in others (Bateman 1977; Hewett 1975). Shah et al. (1995) concluded that even low seed infection rates (<1% and 0.5%) could initiate SNB epidemics, and Griffiths and Ao (1976) hypothesized that fewer than 1,000 infected plants per hectare were sufficient to generate an epidemic, while Luke et al. (1986) reported that about 10% seed infection were needed to cause a severe SNB epidemic. In Norway and Denmark, for example, chemical seed treatment is recommended when the infection level of *P. nodorum* in wheat seed is $\geq 5\%$ to reduce the risk of SNB infection (Brodal 1993).

Even though healthy and/or fungicide-treated seed will reduce the risk for SNB infections in fields free of wheat debris, it is difficult to estimate the remaining risk of crop infections from alternative sources (Bennett et al. 2007). Luke et al. (1983) studied the effect of seedborne inoculum on SNB epidemics in wheat after 0, 1, and 2 years of rotation in fields with *P. nodorum*-infected wheat debris. They showed that seed treatment significantly reduced SNB severity when no crop debris remained in the field due to a 2-year rotation scheme, but not when rotation was absent or only one year elapsed between wheat crops (Luke et al. 1983).

Residue management. Wheat residue management by conventional tillage or by rotation with nonhost crops is widely used to reduce the risk of diseases transmitted by wheat debris, but the extent to which these measures reduce actual yield loss has not often been studied. Mehra et al. (2015) examined the relationship between infected wheat residue, SNB epidemics, and yield reduction in winter wheat. They found a correlation between percentages of residue coverage on the soil surface and SNB severity, although severity leveled off above 30% residue coverage. Grain yield and TKW were generally lower in plots with infected wheat debris than without, but the effect of wheat residue level on yield quality (test weight, determined by how many kg or pounds of grain fit in a certain volume) and quantity was only significant in one year. That year, weather conditions were also more conducive for disease development than in the other

* YL= Yield loss due to weeds, pathogens and insect pests

Fig. 5. Graphical illustration of potential, attainable, and actual yield and yield loss caused by pests and diseases. Modified after van Ittersum and Rabbinge (1997) by E. Fløistad.

(Mehra et al. 2015). Sutton and Vyn (1990) showed that wheat yield was significantly affected by crop rotation, but not by tillage regime, while Bailey et al. (2001) found that even a 2-year rotation had only limited effect on SNB, and increased yield in only 2 out of 7 years. Surprisingly, conventional tillage did not increase grain yield in any of the years (Bailey et al. 2001). Likewise, when Gilbert and Woods (2001) monitored 248 conventional and conservation till fields of wheat over 5 years, they isolated significantly more *P. nodorum* and *Z. tritici* in conventional than in conservation till fields. Milus and Chalkley (1997) concluded that a 4-year rotation was necessary to reduce SNB severity on the leaf and to increase yield in untreated field plots when seeds with low SNB incidence were used. This indicated that crop rotation is only effective if the host is absent from the cropping cycles for an extended time.

The limited effect of tillage regime on SNB severity and yield loss is surprising, as one assumes that conventional tillage leads to a significant reduction in wheat debris. Generally, necrotrophic pathogens that survive on plant debris are well managed by plowing, but in those cases plowing really needs to be deep enough to reduce plant debris sufficiently. The effect of deep plowing might be eroded after ascospore arrival or a low degree of seed infections by highly conducive conditions for SNB epidemics during the season. Late in the season, when SNB is usually assessed, the delayed start of the epidemic due to plowing could be well compensated by rapid disease development during the season. This hypothesis is supported by a study from Stover et al. (1996), where the effect of chisel and moldboard plowing on early and late wheat diseases was compared in a 3-year study.

“Conventional tillage” includes a variation in plowing depth and frequency of harrowing between the cropping seasons, which most likely have led to varying amounts of infected plant debris on the soil surface between the different studies. Quantifying plant debris after conventional, reduced, and zero tillage, and relating those numbers to SNB severity and yield loss would help us to choose a tillage regime that actually reduces inoculum sources and limits or delays a severe SNB epidemic. Rotation with nonhosts over several years appears to be an effective measure to naturally reduce plant debris and thus limit the amount of local inoculum available.

Host resistance. The use of cultivars resistant to SNB is recommended, if available in combination with other desirable traits, such as high yield and earliness. How much SNB resistance contributes to yield losses depends on the mechanism of resistance and the yield potential of the pedigree. There appears to be no complete resistance against SNB in the current wheat gene pool (Aguilar et al. 2005). Host resistance is mostly expressed quantitatively (Eyal et al. 1987). In recent years, insensitivity for necrotrophic effectors (NEs) or host selective toxins produced by *P. nodorum* have become a means to breed for reduced susceptibility to SNB (Oliver et al. 2012). This trait cosegregates with major disease resistance QTLs, but is mostly involved in seedling resistance (Solomon et al. 2006). A study by Ruud et al. (2017) has now shown that the interaction between an NE and its corresponding susceptibility gene in a recombinant inbred population explained 24% of phenotypic variation also at the adult plant stage in the field. Along with wheat residue, degree of cultivar resistance has been found to be one of the factors most closely associated with SNB severity (Mehra et al. 2016), and host resistance was shown to be an effective means of reducing SNB to acceptable levels (Cowger and Murphy 2007). However, resistance to SNB development in non-spike plant parts (leaves and stems) is under separate genetic control from SNB resistance in spikes (Fried and Meister 1987), making breeding for SNB resistance in the whole plant even more complex.

There is sparse information on the effects of host SNB resistance on yield. Australian investigators quantified SNB levels and reduction of grain weight in both diseased and fungicide-protected plots for a set of wheat lines with varying degrees of quantitative resistance to SNB (Loughman et al. 1999). In this 2-year field experiment, wheat lines were evaluated for their response to *P. nodorum* (Loughman et al. 1999). Disease levels and different yield parameters in infected fields were expressed as percentage of those in fungicide-protected plots. SNB severity on the flag or flag and penultimate leaf ranged

between 30 and 41% for moderate to resistant lines, while average RGW was between 81 and 94%. For susceptible lines, SNB severity ranged between 63 and 82%, which translated to an RGW of 57 and 67% of the control plots, respectively. Cultivar resistance here accounted for a 25% increase in RGW as compared with susceptible cultivars (Loughman et al. 1999). In the U.S., there has been screening of multiple regional nurseries of advanced experimental wheat lines, and the data have revealed significant differences in quantitative SNB resistance, both in leaves and heads.

In another study, Oliver et al. (2008) sought to determine the relationship among disease scores, fungal biomass as quantified by qPCR, and yield. They found that fungal biomass in samples collected at 220°C thermal days was predictive of the final loss in grain weight and that resistance levels were positively correlated with RGW when the most resistant cultivar had the highest RGW score and the lowest disease score. In other words, even in an environment where heavy losses to the disease were observed, it was possible to obtain an approximately 80% increase in RGW by using cultivar resistance, and each increase of one unit in disease score was worth a roughly 5% increase in RGW (Oliver et al. 2008).

In the Australian Grains Research and Development Corporation (GRDC) report, Murray and Brennan (2009) stated that breeding contributed 100% to SNB management in 1998, but only 30% in 2008, when fungicides contributed 29% and cultural control 42% to control of SNB. The Australian department of agriculture and food has rated Australian wheat cultivars for SNB, but found no fully resistant cultivar. As described in their 2017 report, SNB susceptibility ranged between medium resistant to susceptible.

In 2007, 44 European winter wheat cultivars were assessed for their susceptibility to SNB. Twelve of these were scored as having very good resistance to SNB (Tommasini et al. 2007). The study aimed to map resistance QTLs and determine predictive markers for SNB resistance using association mapping, and no yield data were given to assess the effect of SNB resistance on yield gain. SNB resistance in commercial cultivars and advanced breeding lines in Europe is evaluated continuously in field trials in Norway, Finland, and other countries with regular SNB epidemics to select for better and durable SNB resistance under field conditions.

The precise mechanisms of host resistance to SNB are not well understood. Possible traits include limiting initial infection, limiting lesion expansion, reducing sporulation in lesions, insensitivity to NEs, and lengthening the latent period. It is also possible that some cultivars simply compensate for diseased leaf and head area better than others. As a quantitative trait, scoring SNB resistance in the field can be challenging, especially if multiple stress factors, such as other leaf blotch diseases or drought, make assessment difficult.

Chemical control. Reducing potential yield losses and redundant fungicide application is the major goal in IPM. Determining the time of fungicide spray does not only depend on the stage of the epidemic, but also on the need to protect certain plant organs to reduce yield loss and the effectiveness of the product used. Currently, quinone outside inhibitors (QoIs) like azoxystrobin and trifloxystrobin, demethylation inhibitors (DMIs) such as epiconazole, tebuconazole, propiconazole and prothioconazole, and succinate dehydrogenases (SDHIs) such as bixafen are recommended to control SNB in Europe.

However, Blixt et al. (2009) showed that SNB populations in Sweden displayed a reduced sensitivity to QoIs, a trend that was also observed in Norway in the last 5 years. In their 2016 study of 321 isolates from 11 countries, Pereira et al. (2017) found mutations in the *CYP51* gene, which conferred reduced DMI sensitivity in China, Denmark, Sweden, and Switzerland. No mutations were found in isolates collected from North America, Australia, or Africa (Pereira et al. 2017). It is possible that the more high yielding European countries and China have a lower action threshold for fungicide applications than the lower yielding areas in North America, Australia, and Africa. Based on the 2014 data of the crop yield by country published by FAO (<http://www.fao.org/faostat/en/#data>), Iran, Australia, the U.S.A., and South Africa are producing less than 4 t/ha, while Sweden, Switzerland, Denmark, and China produced over 5t/ha. However, the tested isolates were sampled between 1991 and 2010 and it is not

clear if the reduced sensitivity in some of the populations reflected increased cultivation and fungicide use at or before the time of collection.

As shown above, protecting the green plant parts during the grain-filling period by spraying at flowering appears most critical for yield. Depending on national guidelines for the use of certain plant protection products, some fungicides cannot be applied later than 5 weeks before harvest, which often restricts the last fungicide application to late flowering. Alternatively, one can consider to spray preventively and halt the advancement of the spore spread vertically up the plant, before flowering and before the epidemic gains momentum.

A variety of fungicide application times have been tested to determine the timing that would reduce yield loss the most. In a 3-year fungicide trial in southeast England, Cook (1977) showed that only fungicide applications between flag leaf stage (GS 39) and heading (GS 58) had a significant effect on yield in one field (4.8 to 5.0 t/ha), while no significant effect on yield was registered when fungicides were applied at the dough stage (GS 70 to 75). The effect was only significant when fungicides were applied immediately after infection

periods (Cook 1977). Even though application timing depends on dose, growth stage of the crop, and disease pressure, Jørgensen et al. (1999) found the most profitable time for fungicide application between flag leaf stage and mid heading (GS 55). In a 3-year fungicide trial with winter wheat, Willyerd et al. (2015) showed that the highest yield response resulted from fungicide treatments between flag leaf emergence (GS 37) and mid boot stage (GS 43). Both studies suggest that fungicide applications before flowering (GS 37 to 59) can prevent infections of the yield determining plant organs. However, infection patterns are strongly dependent on local weather factors and in some regions, flag leaf development and grain filling periods might or might not coincide with precipitation patterns favorable to SNB infection, leading to very different results.

Disease prediction models. Some disease prediction models have been developed to better anticipate SNB epidemics in the field in order to optimize fungicide application timings; others have been developed to better understand the SNB epidemic and identify knowledge gaps. In the first mechanistic simulation model for SNB, called Episept, Rapilly and Jolivet (1976) considered each phase of the

Fig. 6. Graphical representation of the role of different inoculum sources (local and regional) to lower canopy infections, flag leaf and head infections, and the subsequent effect on yield. Red indicates the relationship is well supported by literature; yellow indicates there have been contradictory reports on the relationship between a particular factor and yield loss and quality. The question marks show the uncertainty about the specific quantitative relationship.

Fig. 7. Graphical representation of the effect of different management strategies on local and regional inoculum sources in relation to yield. Red indicates a confirmed relationship to yield loss, orange indicates controversy about the role of a specific factor in actual yield loss.

epidemic (liberation and dissemination of the conidia, infection, and latent period) in order to follow SNB development over time. This detailed parameterized model identifies the critical role of temperature and precipitation in the SNB epidemic. Djurle and Yuen (1991) designed a simulation model for *P. nodorum* by coupling a model simulating SNB development to a wheat growth model. The simulation model followed the life cycle of *P. nodorum*, starting with conidia produced in pycnidia from local inoculum sources. Running the model with doubled initial inoculum led to a 20% increase in spore production, infection rate, and lesion growth (Djurle and Yuen 1991). The authors concluded that spore dispersal parameters (weather factors) and threshold criteria for dispersal and infection were more important for disease development than the amount of initial inoculum (Djurle and Yuen 1991). Similarly, Bhathal et al. (2003) found it was not possible to predict the effect of early-season infections on later disease development in Western Australia.

Analyzing the data from the National Winter Wheat Leaf Disease Survey in England and Wales from 1970 to 1975, Tyldesley and Thompson (1980) developed a set of SNB prediction rules based mostly on the number of wet days (precipitation of 1 mm or more) during the second half of May (first forecast) and the first half of June (final forecast). The authors divided the potential SNB epidemics into three classes (slight, moderate, and severe) depending on the numbers of wet days during the two periods. Following these rules, they correctly predicted SNB epidemics in 30 out of 48 trials. In order to refine the forecast, Tyldesley and Thompson (1980) suggested to include severe infections the previous year, amount of sunshine, previous crop, cultivar resistance, and heavy rainfall (e.g., thunderstorms) as predictive factors.

Shah and Bergstrom (2002) described a regional rain-driven model to predict SNB seed infections. As the sexual stage had not been found and common rotation practices reduced local inoculum sources in New York State, seedborne infections rather than ascospores or debris-derived conidia are considered the primary inoculum sources for SNB epidemics. This model identified rainy periods during stem elongation and seed development as critical for predicting SNB epidemics. The predicted incidence of seed infection from this model did not differ significantly from the actual incidence in the field (Shah and Bergstrom 2002). The model also identified the importance of accurate predictors for splash height distribution, as rainfall with similar characteristics can lead to a range of different splash heights (Shaw 1991), complicating predictions of vertical movement of infection.

In the first pre-planting SNB risk-forecasting model, Mehra et al. (2016) compared multiple regression and three machine-learning models for their ability to accurately predict SNB severity using a dataset of 431 unique combinations of putatively influential pre-planting variables. They identified cultivar resistance, amount of wheat residue, and location in North Carolina (longitude and latitude) as significant predictors of SNB severity (Mehra et al. 2016). After validation with the use of additional data, the model could help facilitate such decisions as rotation adjustment and the relative importance of choosing SNB-resistant cultivars in an overall crop management plan.

In 1994, Hansen et al. (1994) developed a simple predictive regression model to determine fungicide application thresholds for *Z. tritici* and *P. nodorum* in winter wheat based on historical disease and yield data from 1980 to 1989 in Denmark. The frequency of precipitation events necessary for disease severity above 5% on July 15th, was above eight for susceptible cultivars and above nine for resistant cultivars. The economic threshold was set at 0.6 t/ha yield increase compared with control plots, which justified fungicide applications, based on the costs for two fungicide applications in Denmark at the time of study. The model was tested in a 2-year fungicide trial and appeared to predict outbreaks of septoria epidemics well, even in fairly dry years (Jørgensen 1992; Secher and Murali 1992). Sources and amount of primary inoculum were not considered in this statistical model, but were assumed to be sufficient to start an epidemic. The Danish model was adapted by Elen (2007) as part of a Norwegian forecasting model for leaf blotch diseases in wheat. However, Elen (2007) noted that the model needed adjustment to better predict disease development during heading and consequently yield loss.

In a biologically oriented threshold decision model, Verreet and Hoffmann (1990) inspected leaves at different developmental stages for pycnidia development. They took indicator leaf samples from 30 to 40 plants at different intervals to determine the start of the epidemic. Fungicide applications were recommended when five pycnidia had developed on average per leaf. Spray applications following this threshold model almost doubled yield (0.85 t/ha) when compared with one standard application at early flowering (GS 60) (0.47 t/ha). However, application of the five pycnidia per leaf rule would require regular validation by sampling leaves, as the rule might change with different isolates, cultivars, seasonal climate, and locations.

Several models to predict SNB have been developed over the years integrating our current understanding of SNB epidemics and correlating determining factors with SNB severity. However, few of these models relate the predicted disease to the actual yield loss in order to help the farmer estimate the economic benefit for any pest management taken on the farm level.

Discussion

To date, we have a general understanding of the importance of seed transmission, wheat residue, and flag leaf and head infections in the SNB epidemic, but we lack an in-depth knowledge of how our current management tactics affect the primary inoculum on a field level and which plant organs to protect at what time to avoid how much yield loss. Most of the reviewed literature suggests that yield loss is highest when wheat is infected with SNB after flag leaf emergence (GS 39). However, since the effect of spike infections on yield loss has not been considered and most studies have been based on whole-plant inoculation with high spore concentrations instead of more natural straw-based inoculation, the robustness of this conclusion is somewhat limited. It might also be worth mentioning that correct field evaluations of SNB can often be hampered by infections from other leaf blotch pathogens, such as *Z. tritici* and *P. tritici repentis* that produce very similar symptoms.

The importance of the different inoculum sources and types of SNB in reducing yield at the end of the season is not known. If we focus management efforts on the time period between flag leaf emergence and leaf senescence, we need to reduce inoculum that can arrive at the upper plant parts from infected lower leaves or from airborne ascospores produced from neighboring fields and plants. Presence of ascospores depends on ascospore maturation time in different regions, and spore survival during long-distance transport and deposition. The role of ascospores in the epidemic will therefore vary with region, season, and the local presence of other primary inoculum. Ascospore discharge events were reported to occur mostly in the middle and the end of the season, which might be too late for flag leaf infection, but important for glume infection after the last fungicide can be applied. Local inoculum can originate directly from wheat debris, seed, or possibly alternate hosts. In areas where crop rotation and tillage are common, the use of healthy and/or fungicide-treated seeds are even more critical to delay the epidemic and reduce the risk of yield loss than in the presence of wheat residues or in an area where ascospore production is common.

Under conducive weather, a severe SNB epidemic can develop rapidly from relatively low concentration of initial inoculum due to the high production rate of conidia that can be spread locally up and between plants. Any measures that can delay the onset of the SNB epidemic, such as healthy seeds, rotation with nonhosts, and deep plowing, should be combined with slowing the epidemic down by the use of varietal host resistance and fungicide application if needed. The role of different inoculum sources on canopy infections and their possible impact on yield is shown in Figure 6, while the different management strategies affecting local and regional inoculum sources as they are related to yield is represented in Figure 7. As shown in Figure 7, none of the commonly used management options target inoculum sources beyond the farm level, as they would require a combined regional effort and agreement across a large area that stretches beyond single farmers or even country boundaries.

Considerably more work on SNB management thresholds remains to be done. The disease threshold at which a fungicide application

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becomes profitable will depend on local conditions, presence of other diseases, yield potential, host resistance, wheat prices, and markets. As shown with the yield models reviewed here, healthy area absorption (HAA) index might need to be considered to predict actual yield and yield losses more accurately and should not only include the leaves, but also the photosynthetic active area of the glume. Estimates of thresholds to direct fungicide applications will be most useful if they take into account not only yield on diseased versus protected crops, but also the costs of fungicide inputs as a determinant of partial profit given local markets (e.g., Cowger et al. 2016).

Our analysis of the literature shows that the complexity of the SNB-yield loss relationship requires an equally complex toolbox of knowledge and experimental approaches, drawing from multiple disciplines such as crop physiology, agronomy, aerobiology, meteorology, economics, plant genetics, and plant pathology. Additional research should investigate the yield benefits of the different management options under varying weather conditions and over several years to test their robustness

and predictability of their performance. Knowing how the fungus survives and spreads in the field, how it interacts with other necrotrophic and biotrophic leaf pathogens, and how infected wheat debris can be reduced quickly will allow us to optimize the already applied control measure and open ways to develop new ones.

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