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The host-pathogen interaction between barley and casual agent of spot blotch (*Bipolaris sorokiniana*) disease: a review

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ABSTRACT

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Understanding of the host-pathogen interaction is key to uncovering the defence mechanisms for being used in breeding programs and integrated disease management. Spot blotch is one of the most common foliar diseasesof barley worldwide. *Bipolaris sorokiniana* (Sacc.) Shoemaker is the causal agent of spot blotch and can cause other destructive diseases of barley such as common root rot, seedling blight and black point or smudge. Adequate knowledge about epidemiology and pathogenicity of the pathogen can provide great assistance to inhibit the outbreak of spot blotch. In this review, first the distribution and host range of the pathogen has been reviewed. Then, disease symptoms and yield loss caused by spot blotch in barley are reviewed. Subsequently, virulence diversity and other pathogenic aspects of *B. Sorokiniana*such as epidemiology, primary and secondary infections, survival and effect of environmental conditions on epidemic of the disease are discribed in detail. Later, different aspects of interaction between barley and *B. Sorokiniana* such as host response and genetics of resistance has been discussed. The importance of molecular markers for studying population structure of the pathogen and genetics of resistance in barley genotypes are also covered. Finally, different disease control measures have been presented and discussed.

Key words: Spot blotch, genetic diversity, host resistance, integrated disease management

INTRODUCTION

Bipolaris sorokiniana (Sacc.) Shoemaker [(teleomorph *Cochliobolus sativus,* (Itoand Kuribayshi) Drechs. ex Dastur. syn. *Helminthosporium sativum* Pamm. King and Bakke.] is the causal agent of spot blotch. It has been a serious problem of barley and wheat productionsince 1910 when it was first shown to be a plant pathogen (Pammel *et al*., 1910). The fungus is one of the most important constraints to the normal growth and development of the crops in warm and humid conditions, and usually the infection becomes severe after inflorescence emergence (Couture and Sutton, 1978c).

Distribution

The pathogen is found in nearly every region where barley is grown. *Bipolarissorokiniana* attacks a large number of species in the Gramineae family (Sprague, 1950)and a few dicotyledonous species (Spurr and Kiesling, 1961). Because of its extended host range, the pathogen has a wide spread distribution.

According to Commonwealth Mycological Institute (CMI) map, the evidence of disease has been reported from more than 90 countries spread over all continents (Anonymous, 1986). The occurrence of the pathogen in cooler areas of the world (East Europe, North-west China, North-WestAfrica, and North America) may be attributed to its ability to acclimatize to cold, which enables its inoculum survival even under freezing winter temperatures (Kumar*et al.*, 2002).

The host range

Bipolaris sorokiniana is known primarily as a cereal pathogen (Nelson and Kline,1961). It causes a major disease, spot blotch, of barley and wheat (*Triticum aestivum* L.) and also can attack oat (*Avena sativa* L.), rice (*Oriza sativa* L.), rye (*Secale cereale* L.),Triticale (*Triticale sp*.) and corn (*Zea mays* L.) (Bakonyi *et al*., 1997; Sivanesan, 1987).Zillinsky (1983) reported that rye is less susceptible and oat is

seldom infected. However, the pathogen has been collected from both these cereals in many countries (Bakonyi *et al*.1997; Sivanesan, 1987).

Yashwant *et al.* (2017) reported that some crops including *Avena sativa* (oats), *Brassica campestris* (mustard), *Glycine max* (soybean), *Lens culinaris* (lentil), *Pennisetum amaricanum* (millet), *Sorghum bicolor* (sorghum), *Vigna radiata* (green gram), *Vigna mungo* (black gram) and *Zea mays* (maize) can be parasitized by *B. sorokiniana* under in-vitro conditions.A wide variety of wild relatives of cultivated cereals and other grasses can be potential hosts for*B. sorokiniana* (Nelson and Kline, 1961, 1962 and 1963).

The pathogen may rarely attack dicotyledonous plants in the field. *Bipolaris sorokiniana* was isolated from leaf lesions in a field of Michelite beans (Spurr and Kiesling, 1961). In addition, Spurr and Kiesling (1961) found that bean, cowpea, cucurbits, pea, sunflower and tomato plants can be parasitized by *B. sorokiniana* in the greenhouse.

Spot blotch

Spot blotch caused by *Bipolaris sorokiniana*, is one of the most important foliar diseases of barley found in nearly every region in the world where the crop is grown (Mathre, 1997). However, it appears to cause significant losses only in areas with a warm and humid climate, and is rarely a problem for barley grown under semiarid conditions (Bonman *et al*., 2005).

Spot blotch can markedly reduce both grain weight in barley andcauses significant yield losses. Under favorable climatic conditions severe damage canoccur, and crop losses are proportional to the amount of leaf and sheath tissue destroyed (Bailey *et al*., 2003).

The severity of disease on barley can vary greatly from year to year, because the pathogen is sensitive to environmental conditions (Bonman *et al*., 2005). Under experimental conditions using artificial inoculation, yield reductions of 11 to 30% have been reported for susceptible barley cultivars in Canada (Clark, 1979; Dostaler *et al*., 1987; Ghazvini and Tekauz, 2004, Ghazvini, 2012).

Bipolaris sorokiniana can also attack other organs of a barley plant causing destructive diseases of barley such as common root rot and seedling blight (Kumar *et al*., 2002). Infection of heads can result in dark seed discoloration, termedblack point or smudge (Bailey *et al*., 2003).

Disease symptoms

The symptoms of *B. sorokiniana* infection vary with the barley genotype and growth stage, the isolate of the pathogen, and the environmental conditions (Kiesling, 1985). Spots can develop on leaves and leaf sheaths at all stages of plant growth and development. Symptoms first appear as small brown spots on the leaves that enlarge into elliptical, uniformly dark brown blotches with distinct yellow halos, but may later coalesce into irregular dark brown necrotic areas (Dickson, 1956). The spots are usually restricted in width by leaf veins; however, in some cases, lesions may continue to enlarge to form blotches that cover larger areas of leaves (Mathre, 1997).

The most common characteristic of the symptoms is the production of a dark brown pigment in the lesions (Kiesling, 1985). Older spot blotch lesions often appear as olive black, due to sporulation of the fungus (Mathre, 1997). Lesions closely resemble the spotted form of net blotch. Lesions may extend in length on theleaf blade, but they do not become long, narrow streaks as in net blotch (Bailey *et al*., 2003).

Depending on host response (resistance or susceptibility), pathogen virulence and environmental factors, lesion size may vary from minute to small necrotic lesions (0.3-0.7 mm in length and 0.3- 0.5 mm in width) with no or very slight diffuse marginal chlorosis, indicative of low compatibility, to large necrotic lesions (4.0- 8.0 mm in lengthand 1.4-3.2 mm in width) with distinct chlorotic margins (ranging from 0.5 to 1.0 mm in width) indicative of high compatibility (Fetch and Steffenson, 1999).

Dark spots may also appear on the leaf sheaths, necks, and heads of the plants. Lesions on the stalk below the head, especially at the nodes, can result in ‘neck break’ (Bailey *et al*., 2003). Early floral infections cause aborted embryos or severely shriveled grains (Anderson and Banttari, 1976). The grain blight phase of the disease is referred to as ‘black point’ or ‘kernel blight’, and may develop if inoculum is abundant following heading, and environmental conditions are conducive to infection (Mathre,

1997). The dark brown areas that develop on lemmas of infected grains are usually found at the basal end (Anderson and Banttari, 1976).

Yield loss

Spot blotch caused average yield losses of 26% and 16% in 1976 and 1977, respectively, and a 10% reduction in grain weight, in Ottawa, ON (Clark, 1979). In North Dakota, Nutter *et al*. (1985) found that yield losses in six-rowed barley cultivars inoculated at specific growth stages with *B. sorokiniana*ranged from 4% to 20%. An annual grain yield loss of 5-10% was estimated for barley production in Manitoba when plants were damaged by the leaf spot complex of net blotch (*Pyrenophora teres*) and spot blotch (Tekauz *et al*., 2003). Results of two-year field trials with two barley cultivars naturally and artificially infected with *B. sorokiniana* showed that leaf inoculation resulted in significant reduction in thousand grain weight, grain size, and grain yield in two years, and control of *B.sorokiniana* spot blotch by fungicides led to 8% increase in grain yield in one year (Presser,1991).

Ghazvini (2012) also found that the average grain yield losses of barley cultivars caused by the high virulence and low virulence isolates were 11% and 6%, respectively. The effects of seed-borne infection of barley by *B. sorokiniana* on yield loss have also been investigated. Whittle and Richardson (1978) found that with no development of foliar disease, average losses from heavily infected seed stocks was 15%.

Physiological specialization and virulence diversity of *B. sorokiniana*

'Parasitic' specialization in *B. sorokiniana* was first described by Christensen (1922), who showed that isolates of the fungus varied considerably in their virulence on avariety of cereals and grasses. Further studies indicated that virulence diversity persistsamong isolates of the pathogen (Clark and Dickson, 1958; Gayed, 1962;Wood, 1962). Tinline (1960) reported pathogenic and cultural variation among populations of *B. sorokiniana*, but did not make any comments on differential expression of the isolates on wheat.

The first evidence of host-specific virulence in isolates of *B. sorokiniana* was reported by Levitin *et al*. (1985). Since then, several studies have reported on differential virulence versus continuous variation in aggressiveness of *B. sorokiniana* isolates. However, all of these studies, demonstrating the differential virulence of *B. sorokiniana* isolates, have been conducted using barley genotypes as differential lines.

To date, there is no report of differential virulence among pathogen isolates on wheat genotypes. Tinline (1988) mentioned that since most of the *B. sorokiniana* isolates were virulent, but demonstrated no differential virulence on wheat, that almost any isolates from the host could be used in screening wheat for resistance. Duveiller and Garcia Altamirano (2000) could not find any physiological specialization among isolates of the pathogen on wheat collected in Mexico. They suggested that the pathogen appeared as a continuum of isolates differing in aggressiveness.

Hetzler *et al*. (1991) reported that only 1-2% of the variance was attributed to host-pathogen interactions, when evaluating the infection responses induced by 206 *B. sorokiniana* isolates collected from 24 different countries on 12 wheat cultivars. Maraite *et al*. (1998) also pointed out that *B. sorokiniana* isolates, unlike rusts, do not exhibit clear virulence patterns and consist of a continuum of strains differing in aggressiveness.

On the contrary, differential virulence in *B. sorokiniana* isolates has been reported using barley genotypes. Fetch and Steffenson (1994) found *B. sorokiniana* isolates having differential virulence on several two-rowed barley genotypes from North Dakota. These types of isolates were further designated as ND pathotype '2' (Valjavec-Gratian and Steffenson, 1997a). Differential virulence among *B. sorokiniana* isolates was also detected by Meldrum *et al*. (2004) who identified six pathotypes of *B. sorokiniana* in Australia based on their differential virulence on 12 barley genotypes. Distinct differential virulence in *B. sorokiniana* isolates has also been reported in Uruguay (Gamba and Estramill, 2002) and Syria (Arabi and Jawhar, 2004).

Ghazvini and Tekauz (2007) evaluated the virulence diversity of 127 *B. sorokiniana* isolates collected from Canada and other parts of the world using 12 barley differential lines. They found different virulence patterns among *B. sorokiniana* isolates and classified 127 isolates into eight virulence groups. Their results indicated broad virulence diversity in population of the pathogen in prairie region of

Canada, especially in Manitoba.

For further evaluation of the *Hordeum vulgare-B. sorokiniana* interaction model, Ghazvini and Tekauz (2008) also used different quantitative approaches to analyze data. Analysis of the data based on infection response elicited on the barley differentials indicated that population of *B. sorokiniana* consisted of three distinct pathogenic groups (having low virulence, differential virulence, and virulence with varying levels of aggressiveness). The results of the various quantitative approaches indicated that complex interactions exist among barley genotypes and *B. sorokiniana* isolates of the third pathogenic group which cannot be easily analyzed using the classical method of pathotype identification. It also was inferred that the gene-for-gene model is not the principal system operating in the *H. vulgare-B. sorokiniana* pathosystem, although it plays a role in some interactions. This classification was confirmed by Knight *et al*. (2010).

Epidemiology

Primary infection

Bipolaris sorokiniana over winters on crop residue, in the soil, on wild grasses (many of which are hosts of the pathogen), or on the seed (Mathre, 1997). In the field, the pathogen sporulates on infected foliar and underground plant parts (Chinn, 1977; Raemaekers and Tinline, 1981). However, viable conidia in the soil typically are dormant until being stimulated to germinate by exogenous substances such as exudates from roots of the host plants (Tinline, 1988).

Initial foliar infections in the spring results from airborne conidia produced either from infected straw, wild grasses, infested soil, or myceliain infected straw and seed (Dickson, 1956; Mathre, 1997). The level of primary inoculum depends directly on the development of disease in the preceding crop and on environmental conditions for infection during seed formation (Shaner, 1981).

Spores of the fungus do not normally move long distances by wind and as such do not initiate wide spread epidemics in the same year; spot blotch is thus considered to be a localized disease whose development depends on indigenous primary inoculum as well as on local environmental conditions (Shaner,1981). The number of airborne conidia is primarily related to the extent of infection on the upper leaves. Infection may occur at any growth stage of the host plants. Root rot and seedling blight, spot blotch, and kernel blight are all caused by *B. sorokiniana* at various growth stages of barley (Bailey *et al*., 2003).

Secondary infection

Secondary inoculum is less important than primary inoculum because such conidia may be produced too late to cause significant additional foliar damage during the growing season. Daily spore counts expressed cumulatively to the time of harvest showed that few spores were dispersed sufficiently to serve as inoculum for epidemics early in the growing season (Couture and Sutton, 1978c).

The high spore populations of *B.sorokiniana* trapped after harvest indicated that pathogen sporulates heavily and for long periods of time on barley stubble and debris (Couture and Sutton, 1978c). The incubation period for the leaf blights caused by *Drechslera* and *Bipolaris* species is 6-8 days and tends to be shorter as temperature increases to the optimum in the range of 18-25 °C (Shaner, 1981). In contrast, the latent period in *B. sorokiniana* is relatively long because conidia do not form until the necrotic lesions are extensive, usually at later stages of plant growth; i.e. in crops that are nearly ripe or that have dead leaves (Chinn, 1965; Couture and Sutton, 1978c). However, spores produced during the growing season on diseased portions of lower leaves, even when small in proportion, can spread the disease to upper leaves and heads, and to the other plants in the field (Bailey *et al*., 2003).

The amount of dead leaf tissue determines the potential amount of spore production during the growing season but the final outcome depends considerably on the environmental conditions (Couture and Sutton, 1978c; Shaner, 1981). Secondary inoculum likely is more important for the damage (discolouration) caused on the developing seeds in spikes (i.e. kernel blight phase of pathogen) than is to the destruction of additional foliar tissue.

Survival

The fungus can be long-lived, and this can be attributed to the thick-walled structure of both conidia (Kiesling, 1985) and mycelia (Mead, 1942). In soil, conidia form thickened inner walls and chlamydospores; conidia

without chlamydospores do not germinate (Meronuck and Pepper, 1968). *Bipolaris sorokiniana* is the only known leafblight pathogen that has conidia which can survive, between cropping seasons, in the soil(Hampton, 1979). Mycelial infection of the wheat pericarp by *B. sorokiniana* can persistmore than one year (Shaner, 1981). Boosalis (1962) reported that 65% of the conidia kept inoutdoors'unamended soil remained viable for 490 days.

Effect of environmental factors in spread of the disease

Temperature, moisture, and light are the important environmental factors influencing the development and spread of plant pathogens (Agrios, 1997). *Bipolarissorokiniana* has a world wide distribution, but it damages crops, particularly, in regions that are warm and humid (Kiesling, 1985; Mathre, 1997). Extended periods of light (i.e. longer than 16 h) of warm (above 20ºC), moist weather are conducive to epidemic development (Mathre,1997). Conidial germ tubes of *B. sorokiniana* penetrate into leaf tissue over a temperature range of 6 to 39ºC, with an optimum of 12 to 34 ºC. Maximum infection rates have been found to occur between 22 and 30 ºC (Dosdall, 1923). Clark and Dickson (1958) found that development of maximum foliar disease occurs at 28 ºC. In atwo-year field trial, foliar damage caused by *B. sorokiniana* in barley was more severe inthe warmer year in which mean daily maximum temperatures were 27-30 °C, when compared to the year having more normal temperatures of 21-30 °C (Hampton, 1979).

Couture and Sutton (1978c) reported that airborne spores were especially numerous on days that followed a period of persistent leaf surface wetness of 24-72 h, high relative humidity (RH > 95%), and warm temperature (15-25 ºC). However, a season in which temperatures reach 28-30 °C during the day, but do not persist for long periods, also may be quite favorable for disease (Shaner, 1981). The latent period becomes shorter at 28 °C compared to that at lower temperatures, and more leaf tissues become necrotic sooner following infection (Shaner, 1981). Therefore, even though 28 ºC is not the most favorable temperature for spore production directly, but leads to a greater potential substrate for spore production (Shaner, 1981).

The persistence of moisture on the leaf surface is a significant factor in plant infection. Moisture is also necessary for sporulation, spore dispersal, and infection. Relative humidity near 100% is essential for formation of conidiophores and conidia in fungal leaf blights (Shaner, 1981). Although prolonged (24-72 h) periods of leaf-surface wetness favour sporulation, moist periods only at night seems to be sufficient for copious spore production (Shaner, 1981). A change in leaf spot predominance from tan spot to spot blotch between 1990 and 1991 in Nepal was attributed to foggy weather and higher than normal night temperatures (Gilbert *et al*., 1998).

Light has also an important role in formation of conidiophores and conidia. *Bipolaris sorokiniana* has a diurnal periodicity for spore production and spore release (Shaner, 1981; Sheehy and Huguelet, 1967). Light promotes conidiophores production and no conidiophore forms in darkness (Shaner, 1981). In contrast, only few conidia form in light, and most of these form in darkness once conidiophores have developed (Shaner,1981). Spurr and Kiesling (1961) found that *B.sorokiniana* sporulated more heavily on barley straw when this was located mainly on areas exposed to direct sunlight. Arabi and Jawhar (2003) reported that exposure of the fungal colonies to ultraviolet-C radiation (254 nm) increased mycelial growth and sporulation of *B. sorokiniana* as well as its virulence on barley sub-crown internodes.

Effect of environmental factors on spore release and dispersal

Conidia produced on barley debris in spring are dispersed by wind and rain splash to new crops and may initiate epidemics. Wind and rapidly declining relative humidity increases spore release (Kiesling, 1985). Low relative humidity favours spore release, especially at low wind speeds (Shaner, 1981). Although wet conditions are necessary for spore production, dry conditions favor aerial dispersal (Shaner, 1981).

The daily peak of *B. sorokiniana* spores in the air above a wheat field canopy occurred between 13:00 h and 16:00 h and the majority of daily spore peaks occurred after 10 or more hours of sunshine (Sheehy and Huguelet, 1967). Circumstantial evidence indicated that spore production and dispersal were prompted by persistent leaf-surface wetness, high relative

humidity and temperatures (> 15 °C) alternating with dry and windy periods (Couture and Sutton, 1978c).

Spot blotch evaluation

Assessment of host response to isolates differing in virulence is essential to evaluate host-pathogen interaction. Many terms have been used to describe disease measurements (Chester, 1950). However, disease incidence and disease severity (James, 1974) defined as the number of plant units affected (e.g. percentage of diseased plants or leaves) and the area of plant tissue infected by disease (e.g. percentage of lesions covering the total area of a leaf), respectively, have been widely accepted in plant pathology. While disease assessment methods that estimate incidence have been applied uniformly by different researchers, the number of methods have been developed to estimate disease severity, indicating the diverse interest of specialists in studying disease lossappraisal, epidemiology, or disease resistance (James, 1974).

Based on lesion size and the presence or absence of a chlorotic halo around the necrotic lesions, Cook and Timian (1962) developed a rating scale with five classes to differentiate spot blotch severity on barley plants. Fetch and Steffenson (1999) devised a more comprehensive rating scale for assessing infection responses of barley to *B.sorokiniana*. They developed an illustrated 1-9 scale for infection responses at the seedling stage, and an illustrated descriptive scale consisting of four classes (resistant, moderately resistant, moderately susceptible and susceptible) at the adult plant stage.

Although these scales were well-defined and clearly illustrated, the seedling stage rating scale only describes individual lesion size (including both necrotic and chlorotic components) and does not measure the total leaf area affected by disease. Adlakha *et al*. (1984) described a 0-5 spot blotch rating scale for wheat that includes the percentage of leaf area infected as a component of disease measurement. However, this rating scale does not define the lesions size for each class as precise as the scale developed by Fetch and Steffenson (1999).

Duveiller and Garcia Altamirano (2000) used the rating scale developed by Adlakha *et al*. (1984), but also measured average leaf lesion density (number of lesions cm^{-2}) to evaluate infection responses of the wheat cultivar to *B. sorokiniana* isolates. James (1974) stated that measuring the total leaf area infected i.e. pustules or lesions, including any accompanying chlorosis, necrosis, or defoliation, is likely to be better correlated with disease damage than only measuring individual pustule or lesion area alone.

Although it does not cover all aspects of disease assessment, the spot blotch rating scale of Fetch and Steffenson (1999) has been used extensively by other researchers because of its ease of use (Arabi and Jawhar, 2004; Gamba and Estramill, 2002; Ghazvini and Tekauz, 2007 and 2008; Meldrum *et al*., 2004). The number of lesions and the total leaf area infected can be considered as complementary components of spot blotch assessment when using this scale.

Interaction between barley and *B. sorokiniana*

Interaction is a general term that describes the relationship between two species such as those between a pathogen and its corresponding host plant. Different aspects of the host-pathogen interactions have been evaluated in barley × *B. sorokiniana* pathosystem.

Host response to spot blotch disease

Cell wall defense mechanisms similar to those against biotrophic fungi have been detected and implicated in preinfectional defense of barley (Schäfer *et al*., 2004) and wheat (Ibeagha *et al*., 2005) cultivars against *B. sorokiniana* penetration. This includes formation of papilla-like structures beneath sites of fungal attack, the hypersensitive reaction, or both, resulting in failure of epidermis cell penetration, and the local generation of Hydrogen peroxide (H2O2) in cell wall appositions or in whole cells in both barley and wheat genotypes (Ibeagha *et al*., 2005; Kumar *et al*., 2001; Schäfer *et al.*, 2004).

Formation of pathogen-induced cell wall appositions (papilla-like structures) is considered to be an effective defense mechanism against *B. sorokiniana* (Kumar *et al*.,2001). A post-penetration hypersensitive reaction which restricts fungal growth after penetration into an individual epidermal cell, and is followed by its death, has been also reported to occur in barley (Schäfer *et al*.,

2004).

Ibeagha *et al*. (2005) found that fungal penetration into the epidermal layer failed primarily as a result of a cell wall-associated defense mechanism. They demonstrated that the biotrophic growth phase of *B. sorokiniana* on wheat is confined primarily to a single epidermal cell invaded by infection hyphae, whereas the necrotrophic growth phase is initiated by invasion of the mesophyll tissue and is followed by host cell death which appears to be a consequence of toxin secretion. Resistance of genotypes may be combination of defense responses both at the cell wall level and in mesophyll tissue. Ibeagha *et al.* (2005) pointed out that invasion by itself (biotrophic phase) as well as spread after invasion (necrotrophic phase) were subject to distinct defense reactions exerted in more resistant wheat genotypes. They reported that in wheat cultivars in which the fungus successfully overcame the initial epidermal defense, its spread within the mesophyll tissue (necrotrophic phase) was restricted in the more resistant genotypes compared to susceptible genotypes.

Al-Daoude *et al*. (2019) evaluated changes of different defense mechanisms involved in salicylic acid–mediated defense signaling networks in compatible/incompatible barley-*B. sorokiniana* interactions and found that resistant and susceptible cultivars indicated a reduction in salicylic acid levels 72 hours after inoculation. They suggested that this signaling pathways could have facilitated spot blotch resistance.

Genetics of resistance to *B. sorokiniana* in barley

The use of resistant cultivars is the most economical and environmentally sound means for controlling spot blotch disease in barley. Spot blotch resistance in barley is inherited by both monogenic and oligogenic, as well as polygenic resistance. A single recessive gene that controls the resistance to isolate ND90Pr (ND pathotype '2') in line ND 5883 was reported by Valjavec-Gratian and Steffenson (1997b). Likewise, Bilgic *et al*. (2006) identified a single gene (designated as *Rcs6*) on chromosome 1H of line Calicuchima-sib using a 'Calicuchima-sib/Bowman-BC' DH population which confers resistance to isolate 'ND90Pr' at the seedling and adult plant stages.

Gonzalez Ceniceros (1990) identified two resistance genes to isolate ND85F (ND pathotype '1') in the cv. 'Bowman'. In contrast, Kutcher *et al*. (1996) found RAPD markers that were associated with common root rot and spot blotch resistance, but with relatively small phenotypic effects, supporting the quantitative mode for inheritance of resistance. Other studies have confirmed that resistance to more virulent isolates of *B. sorokiniana* may be exerted through complex inheritance mechanisms in barley cultivars with durable resistance.

Several genes and QTLs located on different chromosomes of the barley cv.'Morex' were found to be associated with spot blotch resistance at the seedling and adult plant stages against isolate ND85F, a virulent isolate of *B. sorokiniana* (Bilgic *et al*., 2005; Steffenson *et al*., 1996; Steffenson and Smith, 2004). The presence of these genes/QTLs in the genome of cv. 'Morex' may elucidate the polygenic inheritance of spot blotch resistance against more virulent isolates. Ghazvini (2014) reported that four putative loci on chromosomes 1H, 3H, 5H, and 7H were associated with spot blotch resistance in line TR 251, a Canadian barley breeding line with a high level of spot blotch resistance. Two loci located on chromosomes 1H and 5H had not been reported in previous studies.

Molecular markers as a powerful tool to assess spot blotch resistance in barley

Molecular markers have been employed in several studies to identify the chromosome location of genes/QTLs associated with spot blotch resistance in barley genotypes. Kutcher *et al*. (1996) used RAPD markers to identify common root rot and spot blotch resistance genes in barley genotypes. Steffenson *et al*. (1996) applied a combination of different molecular markers including RFLPs, RAPDs, single amino acid polymorphisms (SAPs), isozymes, telomeres, centromeres, and some morphological markers to evaluate spot blotch and net blotch resistance loci in a 'Steptoe/Morex'population. Bilgic *et al*. (2005) used the same marker systems to find QTLs associated with spot blotch resistance at the seedling and adult plant stages in several doubled haploid (DH) populations of barley. Recent studies, however, indicated that the use of SSR markers in molecular mapping of QTLs

conferring resistance to *B. sorokiniana*is becoming more common (Bilgic *et al*., 2006; Yun *et al*., 2005 and 2006). Using a combination of SSR and AFLP markers, Ghazvini (2014) reported that four putative loci on chromosomes 1H, 3H, 5H, and 7H were associated with spot blotch resistance in line TR 251, a Canadian breeding line with a high level of spot blotch resistance.

Molecular markers have been also used in several studies to elucidate molecular variability in *B. sorokiniana* populations. Molecular variability in *B. sorokiniana* population(s) was first examined by evaluating isozyme polymorphism among isolates (Terekhova and Rochev, 1989; Valim-Labres *et al*., 1997). Nakada*et al*. (1994) used RFLP markers to discriminate between *Bipolaris* and *Curvularia* species. Since the advent of PCR and PCR-based assays, molecular markers such as RAPDs and its derivatives have been exploited in several studies to elucidate the genetic variability within the *B. sorokiniana* populations (Bulat and Mironenko, 1993;de Oliveira *et al*., 2002; Mironenko and Bulat, 2001).

Zhong and Steffenson (2001) applied an AFLP assay to determine the molecular diversity in *B.sorokiniana* isolates from North Dakota and some other regions/countries. In another study, Zhong and Steffenson (2002) identified AFLP markers which were associated with a locus conferring virulence of *B. sorokiniana* isolates on barley cv. 'Bowman'. Zhong *et al*. (2002) also constructed a molecular genetic map and electrophoretic karyotype of *B. sorokiniana* consisting of 97 AFLPs, 31 RFLPs, two polymerase chain reaction amplified markers, the mating type locus (*CsMAT*), and a gene (*VHv1*) conditioning high virulence on the barley cv. 'Bowman'.

Leisova-Svobodova *et al*. (2012) used AFLP markers and found a high level of genetic diversity among *B. sorokiniana* isolates collected from different regions of the Czech Republic. Ghazvini and Tekauz (2012) used AFLP markers with eight primer combinations and stated that *B. sorokiniana* isolates possessing low virulence and differential virulence on barley genotypes were clearly discernible from other pathogenic isolates. Gene-specific primers were designed from the whole genome for being used in the Ecotilling assays on 50 isolates of *B. sorokiniana* collected from Syria (Jawhar *et al*., 2017). Gene-specific primers in this study were designed based on enzymatic mismatch cleavage and polymorphism discovery in *GlutSynth*, *Carp1* and *XYL2* genes.

Disease management

Spot blotch can be controlled by several management strategies. Chemical seed treatment, foliar fungicide sprays, cultural practices such as crop rotations are used to reduce inoculum sources, where host resistance is considered as the most environmentally sound and safe method to reduce damage caused by *B. sorokiniana* (Bailey *et al*., 2003; Kiesling, 1985; Mathre,1997). Other management strategies such as biological control, induced resistance and integrated disease management have also been investigated (Kumar *et al*., 2002), and may be promising alternatives for spot blotch control in the future.

Chemical control

Seed treatment

B. sorokiniana can be seed-borne, therefore, the use of pathogen-free or fungicide treated seed may be useful to reduce infection levels (Mathre, 1997). Seedling blight of barley can be effectively controlled by seed treatment with fungicides (Clark, 1971; Millsand Wallace, 1969). Systemic fungicide such as Captan, Mancozeb, Maneb, Thiram, Carboxin, Guazatine plus Iprodione and Triadimefon have became available to eradicate seed-borne pathogens in cereals (Sharma-Poudyal *et al*., 2005; Stack and McMullen, 1991). Richardson (1972) found that *B. sorokiniana* colonies could not develop on barley seed treated with Carboxin. Seed treatment by Carboxin plus Thiram significantly reduced the intensity of disease development on subcrown internodes of certain barley genotypes (Hampton, 1979).

Narimol and Fenapanil, as seed dressings, have also been reported as effective systemic fungicides to control seed-borne infection of barley seedling by *B. sorokiniana* (Luz and Vieira, 1982). Seed treatment with Imazalil can significantly reduce root rot at the seedling and adult plant stages (Verma *et al*., 1981), lower disease severity in the sub-crown internode, and increase grain yield and test weight compared to the control treatment (Herrman *et al*., 1990).

Control of seed-borne pathogens is the main reason for seed treatment, it is suggested that decisions on application of seed treatments should be determined based on seed health test results (McGee, 1995).

Foliar sprays

Foliar applications of fungicides can significantly reduce the level of infection caused by *B. sorokiniana* in barley and increase yields (Mathre, 1997). Couture and Sutton (1978b) reported that spot blotch severity was significantly reduced by 63, 68, 68,77, 82 and 88% in barley, compared to controls, with application of foliar fungicides Mancozeb, RH-2161, Chlorothalonil, Fentin hydroxide, Triadimefon and Anilazine, respectively.

All fungicides except Chlorothalonil significantly increased the 1000-grain weight of treated plants by 11-15%. Triadimefon could markedly suppress the progress of spot blotch and delay leaf senescence in barley (Couture and Sutton, 1978a). Propiconazole can also reduce spot blotch severity. Foliar treatment of barley cultivars with Propiconazole resulted in increased yield and yield-related traits in both hulled and hulless barley genotypes (Lee *et al*., 1997). Based on a cost-benefit analysis of four foliar fungicides applied to wheat, Sharma-Poudyal *et al*. (2005) demonstrated that a single spray could be cost-effective when a susceptible cultivar is planted under high '*Helminthosporium* leaf blight' severity. However, several applications of fungicides are sometimes necessary to achieve adequate control (Mathre, 1997).

Rotation and crop management

Primary inoculum in crop residue can be reduced by rotation with non-host crops or by tillage practices either through burying or otherwise facilitating the rapid breakdown of stubble, grass weeds and volunteer cereals (Mathre, 1997). Leaf spot disease severity can be greater in monoculture compared to alternating with a non-host crop. Crop rotations take advantage of the fact that plant pathogens important on one crop may be non-pathogenic on another crop. Crop diversification can improve the management of plant diseases through crop selection and interruption of disease cycles through crop rotation (Krupinsky *et al*., 2004).

Increasing crop diversity in crop rotations was found to reduce populations of *B. sorokiniana* and some other pathogens in wheat leaves and roots (Bailey *et al*., 2001). The advantages of crop rotation in controlling conidia populations of leaf pathogens have been reported in several studies (Bailey *et al*., 2000; Chinn, 1976; Duczek *et al*., 1999). Leaf spot disease severity on barley was found to be lower following wheat, mustard, canola and dry pea compared with the barley-after barley rotation (Krupinsky *et al*., 2004). Rotations of two or more years to flax (*Linumusitatissimum* L.) as a break crop, when growing wheat or barley, reduced the amount of viable inoculum of *B. sorokiniana* in the soil (Conner *et al*., 1996).

The survival of *B. sorokiniana* on crop residue is an important means of carryover from one year to another (Duczek *et al*., 1999). The inoculum density of the pathogen in the soil is directly associated to the amount of fungal sporulation occurring on crop residues (Reis and Wünsche, 1984). Reis (1984) found that sporulation was higher on the aboveground plant debris than underground parts. Thus, clearing or ploughing in the stubble, weeds and volunteer plants should be useful in reducing inoculum density.

Host resistance

Barley germplasm with a high level of resistance to spot blotch has been identified. Six-row malting barley cultivars grown in North Dakota, USA have remained resistant to *B. sorokiniana* for more than 40 years. Spot blotch resistance in this North Dakota barley germplasm appears to be derived from three main sources: 'Manchuria', 'Oderbrucker', and CI 7117-77 (Gonzalez Ceniceros, 1990). Line CI 7117-77 was crossed to cv. 'Kindred' which in turn was derived from cv. 'Oderbrucker' with some level of spot blotch resistance, and from this cross, line ND B112 and several resistance sibs were selected (Gonzalez Ceniceros, 1990). Line ND B112 is one of the most stable sources of spot blotch resistance, and has been used widely in barley breeding programs in North America.

The durable resistance found in six-row malting barley cultivars developed in North Dakota is mostly derived from line ND B112 (Valjavec-Gratian and Steffenson, 1997a). Cultivars such as 'Cree', Manker', 'Morex', 'Robust',with high level of spot blotch

resistance were all derived from ND B112 (Wilcoxson *et al*., 1990). Additional sources of spot blotch resistance, which are not genetically related to ND B112, have been identified among *H. vulgare* experimental lines or accessions such as 'Jet' (CI 967), Wisc 691-1, CI 1227, CI 6311, and CI 9584 and also among *H. agriocrithon* accessions (Mumford, 1966; Wilcoxson *et al*., 1990).

Line ND 7556, a two-row experimental line, with a good level of spot blotch resistance at the seedling and adult plant stages, is an additional source of resistance. This line was selected from the cross of 'Norbert//ND4856/M37', where 'Norbert', ND4856, and M37 in turn were selected from the crosses '7118-703-13/Klages', 'Klages/ND1244', and 'Manker/karl//M-18', respectively (Gonzalez Ceniceros, 1990). ND1244, 'Manker', and M-18 are the presumed sources of resistance in ND 7556, because all these six-row barley lines have ND B112 in their pedigrees (Gonzalez Ceniceros, 1990).

Ghazvini and Tekauz (2007 and 2008) found that barley line 'TR 251'wasa good source of resistance to *B. Sorokiniana* isolates especially to the highly virulent pathotypes emerged in western Canada. Al-Sadi (2016) reported that barley cultivars 'Omani' and 'Beecher' were resistant to *B. sorokiniana*. Leng *et al*., (2016) found that barley accessions PI 235186, PI 592275, and PI 643242 collected from the USDA Small Grains Collection were resistant to isolates of pathotypes 1 and 2 as well as newly identified pathotype ND4008 in North Dakota. Singh *et al*. (2017) screened infection response of 342 barley genotypes against spot blotch under natural infection conditions and stated that none of genotypes were immune and only genotype "6B89.2027/5/ATACO/BERMEJO/ /2HIGO/3/CLN-B/80.5138//GLORIA - BAR/COPAL/4/CHER VON-BAR/6/LEGACY" was resistant.

Other control measures

Warm and hot water treatments (Winter *et al*., 1996) and dry heat treatments (Couture and Sutton, 1980) of seed can be used as alternatives to chemical dressings when applied to seeds of barley cultivars susceptible to *B. sorokiniana*. Another alternative strategy for disease control, albeit in its infancy stage, is induced resistance. There is evidence for both biological and chemical induction of resistance to *B. sorokiniana*. Pre-treatment of wheat leaves with *Bipolaris oryzae* (inducer organism) reduced number and size of spot blotch lesions produced by *B. sorokiniana* on the same leaves (Sarhan *etal*., 1991). Chemical induction of resistance to *B. sorokiniana* in barley by pre-treatment with the resistance inducers 2, 6-Dichloroisonicotinic Acid (DCINA), Benzo (1,2,3) Thiadiazole-7-Carbothioic Acid-S-Methylester (BTH) or Jasmonates also resulted in symptom reduction by 10–20% (Kumar *et al*. 2002).

Among control measures other than the application of chemicals, agronomic practices and breeding for resistance, biological control of *B. sorokiniana* have received most attention. Biological protection of barley against *B. sorokiniana* has been studied extensively. Several fungal, bacterial or yeast species have been found that possess antagonist effects against *B. Sorokiniana* (Duczek and White, 1986; Biles and Hill, 1988; Patil *et al*., 1987; Prasad *et al*., 1978; Fokkema*et al*., 1979). Adding extracts of *Dittrichia viscosa* (previouslyknown as *Inula viscose* Aiton), a weed plant from the Asteraceae family, into the growing media inhibited growth of *B. sorokiniana* (Qasem *et al*., 1995). This indicates that antifungal product against this pathogen can be found even in plant species. To date, the efficacy of these antagonistic interactions in real-world farming situations is uncertain, but biological control of *B. sorokiniana* may be applicable to on-farm situations in future. However, integrated disease management is an environmentally and economically sound strategy for controlling the damages of spot blotch disease.

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