Journal of Natural Sciences Research www.iiste.org
ISSN 2224-3186 (Paper) ISSN 2225-0921 (Online)
Vol.8, No.22, 2018

Septoria Tritici Blotch (Septoria tritici) of Bread Wheat (Triticum aestivum L.): Effect and Management Options - A Review

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Summary
Septoria tritici blotch (STB), caused by the fungal pathogen *Zymoseptoria tritici*, anamorph: *Septoria tritici* Rob. ex Desm., is an important wheat (*Triticum aestivum* L.) pathogen worldwide, reported to be major wheat production threating factor, posing considerable yield loss every year. Different disease management options are being used to minimize its effect. Cultural disease management methods such as rotation with non-host crops, field sanitation, late planting and inter-cropping of wheat with other crops can reduce the incidence and severity of STB by reducing available inoculum to initiate infection. Biological control agents such as fungi belonging to *Trichoderma* spp. and bacteria such as *Bacillus megaterium, Pseudomonads* spp. and *Lactobacillus* strains have ability to inhibit growth of the causal pathogen. Application of fungicides, seed treatment or foliar sprays, is used to control STB, as a stop-gap measure or as an integral part of the crop management system. Planting of resistant cultivars is reported to be efficient, economical, environmentally friendly and simple approach for managing STB. Integrating two or more disease management strategies will result in better STB management due to complementary action of one for another.
Keywords: Bread wheat, Septoria tritici blotch, Effect, Management

Introduction

Wheat (*Triticum aestivum* L.) is the second important staple grain produced all over the world and occupies nearly 220.11 million hectares of land worldwide. In 2016, the worldwide wheat production was 749.46 million metric tons (Mt), which makes it the second important grain crop after maize. China is currently the world's leading wheat producer, accounting for approximately 14.95% of the world's total production. Other major wheat producing countries are India, Russia, USA, Canada, France, Ukraine, Pakistan, Germany and Australia. These 10 countries together produce about 59.04% of the world's total wheat. Ethiopia is the 2nd wheat producing country in Africa, after Egypt, and it is the largest wheat producing country in the Sub-Saharan Africa (FAOSTAT, 2018). In Ethiopia, wheat ranks fourth after *tef* (*Eragrostis tef* Zucc.), maize (*Zea mays* L.) and sorghum (*Sorghum bicolor* L.) in both area of production and total grain production (CSA, 2017).

Wheat production is constrained by biotic (such as diseases, insect pests and weeds) and abiotic (such as waterlogging of vertisol soils, frost, low rainfall and depleted soil fertility) factors and low potential of landraces. Diseases caused by fungal pathogens are among the most important biotic factors constraining wheat production. Septoria tritici blotch (S*eptoria tritici*; perfect (sexual) state: *Zymoseptoria tritici*, formerly called *Mycosphaerella graminicola*) is among the major fungal diseases which threat wheat production. The disease has a worldwide distribution and causes considerable yield loss every year (Eyal *et al.*, 1987; Kasa *et al.*, 2015).

Being an important disease, different management options are being used to minimize its effect. Cultural disease management methods such as rotation with non-host crops, field sanitation, late planting and inter-cropping of wheat with other crops can reduce the incidence and severity of STB, by reducing available inoculum to initiate infection. Enhancing soil nitrogen content through cultural practices is also helpful to reduce severity of STB. Several biological control agents such as fungi belonging to *Trichoderma* spp. and bacteria such as *Bacillus megaterium, Pseudomonads* spp. and *Lactobacillus* strains have ability to inhibit growth of the causal pathogen. Fungicide application, either seed treatment or foliar sprays, controls STB as either a stop-gap measure or as an integral part of the crop management system. Planting of resistant cultivars is reported to be efficient, economical, environmentally friendly and simple approach for managing STB. Septorias have been controlled as part of an integrated crop management system using resistant cultivars and chemical control to complement the action of one with another although the disease is still sever in many parts of the world.

This brief piece of work was, therefore, carried out with the objective to review the effect septoria tritici blotch disease has on bread wheat production and its management options.

Geographical Distribution and Economic Importance of Septoria Tritici Blotch (STB)

Septoria tritici blotch is a widely distributed wheat pathogen that occurs throughout the globe. It is found in the

Mediterranean region, South Eastern and Eastern Africa (including Ethiopia), Western Europe, Australia and South and North Americas. In the USA, Brazil, the Netherlands, the United Kingdom and Australia, the sexual state (ascospores: *Mycosphaerella graminicala*) has been identified (Eyal *et al.*, 1987). STB occurs throughout the world in countries as diverse as Argentina, Ethiopia, Iran, the USA, the Netherlands, Russia, New Zealand, and Australia (Ponomarenko *et al.*, 2011).

STB is among fungal diseases which threat wheat production and it is reported to be major wheat production threating factor worldwide causing considerable yield loss every year (Eyal *et al.*, 1987; Kasa *et al.*, 2015). STB occurs in wheat producing areas of all continents and results in serious crop losses in many wheat-growing regions of the world with crop losses in some areas, such as North Africa and southern Brazil, being devastating (Zillinsky, 1983). The disease causes serious yield loss and losses attributed to heavy infestation in fields planted with wheat susceptible cultivars have been reported to range from 30% to 40% (Eyal and Wahl, 1975). Epidemics can be particularly devastating in developing countries, such as those in East Africa, and sever epidemics of STB can reduce wheat yields by 35 to 50% (Ponomarenko *et al.*, 2011).

In the USA, STB is second only to wheat rust in terms of importance, and it is the number one disease of wheat in Russia and many countries of Western Europe (Ponomarenko *et al.*, 2011). In the United Kingdom (UK), annual losses due to STB average around 20% of the country's total harvest (Fones and Gurr, 2015). In European Union (EU), STB affects each country's economy in two-fold: (i) the direct loss of the wheat harvest due to the disease and (ii) the cost of fungicide application. Five to ten percent (5-10%) losses in France and Germany give direct costs ranging between €120 and 700 million. Fungicide treatment additionally costs the farmers between €160 and 500 million across these three nations (Fones and Gurr, 2015). A report by Kettles and Kanyuka (2016) stated that in Europe, STB is the most economically damaging disease of wheat, with an estimated €1 billion per year in fungicide expenditure directed toward its control. Approximately 70% of the estimated volume of fungicide used on cereals in Europe is used to control STB (Ponomarenko *et al.*, 2011). In Iran, yield loss due to STB ranged from 30% to 50% in 2006-2007 and 5% to 20% in 2007-2008 (Mojerlou *et al.*, 2009). The average yearly losses in yield in the United States due to STB and septoria nodorum blotch were estimated between 1 and 7% annually (Eyal, 1981). Annual losses from STB in the USA are estimated to be more than $275 million dollars per year (Ponomarenko *et al.*, 2011).

In Ethiopia, STB resulted in up to 41% yield loss at Holeta Agricultural Research Center (Takele *et al.*, 2015) and 48% at Areka Agricultural Research Center (Said and Hussein, 2016). The disease is widely distributed all over wheat growing areas of Ethiopia and it is economically important disease (Said and Hussein, 2016). STB is the most destructive disease in West and South West Shewa zones and the overall distribution/prevalence of the disease reached 100% (Hailu and Woldeab, 2015). Data generated by 2014 *Belg*[1] season disease survey revealed that STB and leaf rust are the two important diseases constraining wheat production in Arsi and Bale areas of Ethiopia (Kasa *et al.*, 2015).

Economic losses due to STB infections can result not only from losses in grain yield, but also in quality such as under severe epidemics, the kernels of vulnerable wheat cultivars are shriveled and are not fit for milling (Eyal *et al.*, 1987; McKendry *et al.*, 1995).

Disease Cycle, Symptoms and Epidemiology of *Septoria tritici*

Disease Cycle

Infection by *Z. tritici* is initiated by air-borne ascospores and splash-dispersed conidia produced on residues of the previous season's crop. Primary infection occurs soon after seedlings emerge in fall (for winter wheat) or spring. Secondary spread within the crop occurs from rain splash by asexual fungal spores, conidia, produced on infected plants. Ascospore germ tubes are attracted to the stomata, through which they gain entry into the sub-stomatal cavity either directly or after production of an appressorium-like structure (infection cushion). For several days, the hyphae grow intercellularly with little increase in biomass. After the switch from biotrophic to necrotrophic growth, cells collapse, lesions form and are identified initially by small, yellow flecks or blotches. The lesions expand, primarily in the direction of the leaf veins, to form long, narrow, necrotic blotches. Pycnidia develop around stomata within the necrotic areas of the lesions and exude conidia in gelatinous, hygroscopic cirrhi. These spores are disseminated by rain splash to leaves of the same or nearby plants. The pathogen survives crop-free periods primarily as pseudothecia but also in pycnidia on crop debris. Autumn-sown crops and volunteer plants can aid survival over winter (Ponomarenko *et al.*, 2011).

[1] Belg is the cropping season which extends from March to June.

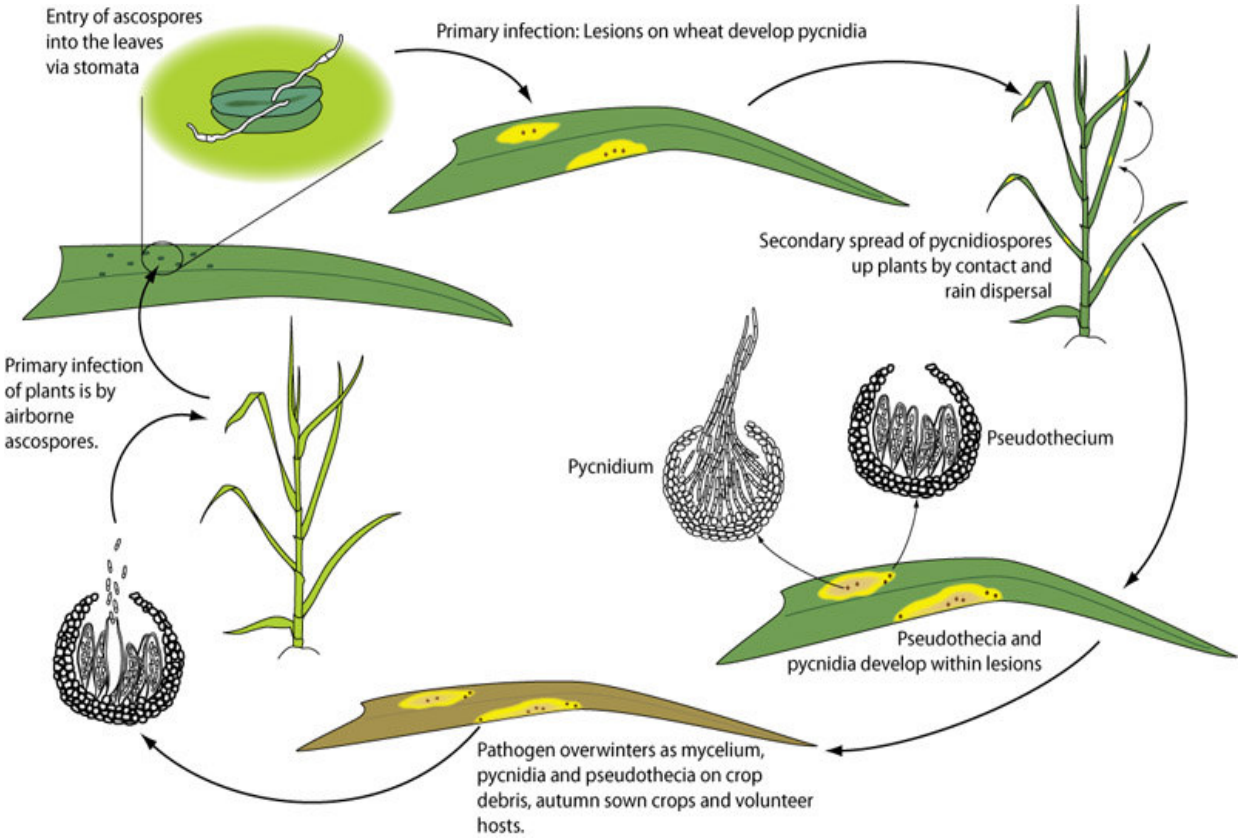

Figure 1: *Zymoseptoria tritici* (anamorph, *Septoria tritici*) disease cycle (Source: Ponomarenko *et al*., 2011)

Symptoms

The initial symptoms of STB are small chlorotic spots on the leaves that appear soon after seedlings emerge in the fall or spring (or 5-6 days after artificial inoculation). However, the time of first expression is highly dependent on environmental conditions (such as moisture, temperature, and light), the cultivar, and the septoria isolate. Under usual field conditions, however, symptoms generally appear after 14-21 days. The necrotic lesions appear sunken and grayish-green at first. As they enlarge, the lesions become light tan and develop darker colored fruiting bodies. By holding the leaf up against sunlight, the beginning of pycnidia formation (when occurring) can often be seen, usually after 15 days. Lesions on mature leaves most often are long, narrow and delimited by leaf veins but also can be shaped irregularly or can be elliptical, particularly on seedlings or leaves that were young when infected. In high rainfall zones when conditions are favorable, lesions can spread across the leaf forming large blotches. Mature lesions contain black or brown fruiting structures, the asexual pycnidia or sexual pseudothecia. In more susceptible varieties, the lesions can appear silver-grey. The pycnidia or pseudothecia develop in the substomatal cavities of the host so are spaced regularly with in the lesions. The pycnidia are scattered within the lesion, and can be found on both the upper and lower surfaces of the leaf. The size of pycnidia may vary among cultivars and is also affected by the number of pycnidia present. As the number of pycnidia on the leaf increases, the pycnidia themselves may become smaller. The disease begins on the lower leaves and gradually progresses to the flag leaf. In wet years, the speckled leaf blotch fungus can move onto the heads and cause brown lesions on the glumes and awns, hence known as glume blotch. These lesions often become light tan as they age and the fungal fruiting bodies are often seen embedded in the lesions on the awns (Eyal and Brown, 1976; Eyal et al., 1987; Ponomarenko et al., 2011).

Epidemiology and Inoculum Dissemination & Survival

An epidemic of STB of wheat is associated with favorable weather conditions (frequent rains and moderate temperatures), specific cultural practices, availability of inoculum, and the presence of susceptible wheat cultivars. The splashing dispersal mechanism affected by rain limits distances to which pycnidiospores can be spread. The usual vertical progress of septoria from lower to upper leaves is affected by the distance between consecutive leaves - the "ladder effect". On tall varieties, the distance between each leaf is greater, especially towards the flag leaf. In dwarf cultivars (70-90 cm tall), the closeness of the upper leaves to the lower leaves facilitates contact between newly emerging leaves and splashed pycnidiospores. Movement of the pathogen from infected lower leaves is thereby made simpler. As a result, pycnidia often appear earlier on upper plant parts of

dwarf cultivars than they do on leaves of taller cultivars. Under severe epidemics, however, the differences in plant architecture and stature of susceptible cultivars are of no importance to the pathogen. Thus, both resistance- and morphology-related genetic factors influence disease spread and resulting severity (Eyal, 1971).

Increased severity of STB is therefore associated with factors such as early sowing of the crop, direct drilling into crop residues which may carry inoculums, and the use of dwarf verities which are more likely to get infection (Lucas, 1998). In wheat-growing regions where septoria pathogens are a potential danger, plant architecture, especially leaf placement, should be taken into account when new wheat cultivars are to be released. Long rainless intervals with high temperatures often interrupt STB progresses from lower infected leaves to upper plant part (Eyal *et al.*, 1987). Initial inoculum usually consists of airborne ascospores, which cause the primary infections on seedling leaves, but also can be from conidia. Primary infections from an ascospore shower will occur evenly over a crop and give rise to lesions that bear pycnidia, the asexual structures that allow for rapid dispersal of the secondary inoculum, conidia (Ponomarenko *et al*., 2011).

Secondary spread of STB is by conidia, which form readily in high humidity, particularly if there is free water present on the leaves, but also can be by ascospores. Pycnidia with conidia are produced roughly 14 to 40 days after infection, depending on the host and seasonal conditions. These spores disperse through rain wash and splashing, causing local spread of the disease to uninfected leaves of the same and nearby plants. Production and dispersal of conidia occurs quite rapidly compared to pseudothecia with ascospores, which take several weeks until ripening. Thus both conidia and ascospores contribute to the epidemic but the asexual cycle seems to dominate during the growing season. Ascospores can be airborne over large distances, while conidia are unlikely to travel far from their site of origin by rain-splash dispersal. Conidia help to spread the disease upwards through the canopy. Infection of flag leaves (last leaf to emerge on a wheat stalk) is common and leads to greatly reduced yields and poor quality of harvested grain. Rain splash of conidia can lead to disease foci, which can give a patchy appearance to the overall disease distribution in a field. A more uniform appearance of the disease is typical when the airborne ascospores are plentiful during the initial infection (Ponomarenko *et al*., 2011).

Many cycles of sexual and asexual reproduction during the growing season allow epidemics to develop rapidly. Debris from heavily infected leaves and stems remains in fields after harvest to produce inoculum for the next growing season. Rain-splashed pycnidiospores are transported vertically upward from the base of the crop to the upper leaves where they germinate and penetrate through the stomata and courses infection (Ponomarenko *et al*., 2011). Infectious spores of septoria are readily dispersed within the crop canopy by wind and splashing rain. The fungi survive from year to year (overwinter) in diseased wheat straw of previous crops, volunteer wheat diseased seed, and other susceptible grasses. In seed, the fungi can remain viable for a year or more. In infested straw, the fungi can remain viable for as long as three years. Overwintered sources of the fungi provide spores (inoculum) for infection of the next wheat crop grown (Hershman, 2012).

Management of Septoria Tritici Blotch of Bread Wheat

Different management options are being used to minimize the effect of STB, although many have their own limitations so that the disease remained difficult until to date. Disease management, including cultivars with acceptable levels of resistance, crop rotation, seed treatments, different cropping and tillage systems and fungicides have been and are being used by growers.

Cultural Management

Cultural disease management methods such as rotation of non-host crops and destruction of residues from previous wheat crops can reduce the incidence and severity of STB. A one year rotation to non-host crops is less likely to be effective as the inoculum may survive viable in infested straw for more than a year and hence, a three- to five-year crop rotation is necessary to reduce the incidence of STB while even longer rotations of six to eight years were insufficient to eliminate the disease. Sanitation achieved by removal of crop debris and deep ploughing can decrease the amount of inoculum available to initiate a new disease cycle. However, this may be less effective due to long-distance dispersal of ascospores, but may be helpful if coordinated within a region. In addition, removal of crop residue is not practicable in light soil areas where stubble must be kept to prevent erosion. Late planting of winter wheat (e.g. mid-October versus late September) may be used as a strategy to moderate the amount of initial infection by avoiding ascospore flights in a newly planted wheat crop. Inter-cropping of wheat with other crops and growing a mixture of wheat cultivars can also be used to help reduce ease movement of inoculum within a wheat field thereby reducing severity of STB of wheat although cultivar mixtures can be difficult to implement on a large scale due to differences in agronomic and quality traits between the pure wheat lines (Eyal et al., 1987; Ponomarenko et al., 2011; Cuthbert, 2011). Krupinsky and Tanaka (2001) reported that enhancing soil nitrogen content through cultural practices is helpful to reduce severity of STB. This might be through producing vigorous seedlings that are likely to survive well under stress by the pathogen.

Biological Control

As Zymoseptoria tritici commonly undergoes sexual reproduction, local populations have extremely high levels of variability which provide the pathogen the ability to rapidly adapt to changing environments such as host resistance or fungicides. To counter these resistance developments, there is an increased need to develop alternative control strategies, including biological control. Safe and environmentally friendly products for plant protection represent an insignificant portion of the pesticide market, which remains dominated by synthetic chemicals (Lynch *et al.*, 2016).

Several biological controls have been and are being evaluated for STB, and some have shown promise. Fungi belonging to *Trichoderma* spp. have been used previously as biocontrol agents to protect wheat plants against STB in Argentina. Perelló *et al.* (1997) tested the biocontrol ability of *Trichoderma harzianum* and *Gliocladium roseum* against *Septoria tritici* under *in vitro* and greenhouse conditions. They observed a highly significant effect of *T. harzianum* and *G. roseum* on the development of *S. tritici* colonies in the *in vitro* tests. A complete growth over and coverage of *S. tritici* colony by both of the antagonists was noted; *T. harzianum* more efficiently inhibited the pathogen than *G. roseum* did. However, the antagonists failed to show their inhibitory capacity in the *in vivo* assays although the percentage of necrotic area with pycnidial coverage reduced when compared with the control.

Perelló *et al.* (2009) evaluated four *Trichoderma harzianum* strains and one *T. koningii* strain, applied as spore suspension and coated-seed technique, under field conditions and reported that STB severity was effectively reduced, compared with untreated control, when applied at tillering (Zadoks growth stage 23; Zadoks *et al.*, 1974). Their finding revealed that one of the strains of *Trichoderma harzianum*, applied as seed-coating, reduced the incidence of STB to 40% and the severity to 70% of that of the control, similarly to the tebuconazole fungicide applied.

Nolan and Cooke (2000) examined the effect of a wheat non-host fungal pathogen *Drechslera teres*, a barley pathogen which causes net blotch disease, for its biocontrol potential against septoria tritici blotch of bread wheat under field conditions. They reported that flag leaf inoculation of wheat plants with *D. teres* prevented *Septoria tritici* symptom expressions and there was significant reduction in disease caused by *S. tritici* when wheat plants were pre-treated with *D. teres* prior to inoculation with the pathogen, *S. tritici*, resulting in a significant increase in grain yield.

Pseudomonas bacteria have been tested for their biocontrol activity against *S. tritici*. In 1990, Flaishman *et al.* identified an isolate *Pseudomonas aeruginosa* strain LEC 1 from soils of Israel central coastal plain and tested for its *S. tritici* suppression capacity *in vitro* and on seedling plants. They noted a growth suppression ability of *Pseudomonas aeruginosa* strain LEC 1 on *S. tritici* and reported the probable mechanism of action to be antibiosis. They also found a marked suppression of pycnidial production (90% of the control) on seedling tests. According to Levy *et al.* (1992), the growth of *S. tritici* was inhibited by *Pseudomonas fluoreseens* strain PFM2 *in vitro,* with a gradually increased inhibition zone when grown in paired culture. The ability of PFM2 to lyse developed conidia indicated its fungicidal activity against *S. tritici.* The antibiotics the strain produced also inhibited the growth of *S. tritici*.

Kildea et al. (2008) screened a collection of bacteria originating from barley leaves and grain, oat chaff and wheat rhizosphere and leaves for their ability to control STB. A total of seven bacteria (unidentified isolates MKB5, MKB86, MKB137, MKB163, two *Pseudomonas fluorescens* isolates MKB21 and MKB91 and a *Bacillus megaterium* strain MKB135) inhibited STB development (significantly reduced the percentage of diseased leaf area covered with pycnidia) by up to 92%, relative to the positive control plants, under controlled environmental conditions. When three of these bacteria (two *Pseudomonas fluorescens* isolates MKB21 and MKB91 and a *Bacillus megaterium* strain MKB135) were assessed for their abilities to control STB on adult wheat plants in small-scale field trials, only *B. megaterium* strain MKB135 consistently retarded STB development (resulted in a significant decrease in AUDPC by up to 80%).

Lynch *et al.* (2016) explored effective biological control agent against fungicide-resistant *Zymoseptoria tritici* in Ireland and found that two *Lactobacillus* strains (*Lactobacillus brevis* JJ2P and *Lactobacillus reuteri* R2) were effective against the pathogen. Under controlled conditions, these two strains significantly reduced the development of STB on wheat seedlings resulting from infection with three different *Z. tritici* strains. Although several biological control agents seem to be promising, none is available yet for commercial production.

Using Resistant Varieties

Planting of resistant cultivars is an efficient, economical, environmentally friendly and simple approach for managing STB. Resistance to *Z. tritici* can be qualitative or quantitative and is more common among winter wheat than in spring types. To date, many qualitative (major) genes conferring resistance to STB have been named, mapped and published. In the field, some septoria tritici blotch resistance genes (*Stb* genes) have been quite durable (long lasting) while others have failed due to rapid genetic change in the pathogen population. For example, *Stb1* has remained effective in Indiana for more than 25 years, while *Stb4* was effective in California

for 14 years before it failed, but only lasted one or two years in Oregon. Often wheat cultivars reported as resistant in one region have been found to be susceptible in another. This may be connected to the genetic composition of the local pathogen population, which can be affected by cultivars grown, the suitability of the environment for infection, and the relative importance of the sexual stage in the disease cycle (Ponomarenko *et al.,* 2011).

Quantitative resistance also is known and may occur commonly in wheat cultivars. However, unlike quantitative resistance against many other pathogens, that against *Z. tritici* may be isolate specific so could be circumvented more easily. Whenever possible, the use of qualitative *Stb* genes should be combined with quantitative resistance to help ensure its stability. Molecular markers have been linked to many of the *Stb* genes, which through marker-assisted selection will aid the creation of effective gene combinations in new wheat cultivars (Ponomarenko *et al*., 2011).

About 21 major genes conferring qualitative resistance to STB have been identified and mapped to date (Table 1). But, most are effective only against avirulent genotypes of *S. tritici* as resistance can be overcome through the evolution of pathogen virulence (Brown *et al.*, 2015). Eyal *et al.* (1987) also stated that most of the high-yielding wheat cultivars are susceptible to STB. A study by Abebe *et al.* (2015) in Tigray, Ethiopia, showed that, among 200 genotypes evaluated for their response to the prevailing *Septoria tritici* population under field conditions, none of the genotypes were resistance to STB and the majority were susceptible to highly susceptible. Therefore, resistance is a high-priority breeding goal since host resistance is "the main pillar of defense against disease" and for narrowing the potential and actual yields gaps (Browning, 1979; Scharen and Krupinsky, 1978).

Chemical Control

Fungicide protection has been used either as a stop-gap measure, or as an integral part of the crop management system. Its purpose has been to secure the high yields of susceptible cultivars (Cooke and Jones, 1970). Fungicides are only recommended when they would be of economic benefit. Factors to consider include the projected yield and loss from STB and whether the cost of fungicide will justify the expected benefit. The susceptibility of the wheat cultivar and amount of disease, in particular, influence this decision. Timing sprays to periods when the pathogen is most likely to be active will yield the greatest economic return on effort (Ponomarenko *et al*., 2011).

The design of an economical chemical control program for protection from the septoria pathogens of wheat depends upon several crop management considerations. Prior to applying chemicals, wheat growers and/or researchers must decide whether to resort to chemical control of the specific wheat field is necessary. The considerations include: early assessment of yield potential and economics of the specific wheat field; vulnerability of the wheat cultivar to septoria; history of wheat cropping and septoria epidemics in the specific field; disease levels in the specific field; cultural practices undertaken before sowing (burying of refuse, deep plowing, etc.) that might reduce the amount of primary inoculum; early detection of the diseases and assessment of its progress; the prevailing weather conditions; cost to be incurred for fungicide protection; and projected yields and losses (Eyal *et al*., 1987).

Several fungicides are currently used to control STB. A seed dressing of a fungicide, such a triticonazole, can suppress early infection and so can be used in regions where the seedling stage can be affected by the pathogen. Foliar sprays, however, are the most common type of fungicide treatment (Ponomarenko *et al*., 2011). As preventive foliar fungicides, Dithiocarbamates (maneb, manzate, mancozeb, zineb) have proved effective in controlling septoria diseases. However, these protectant fungicides require repeated application at 10- to 14-day intervals. A chemical control program of 3-4 maneb applications, where the upper plant parts responsible for grain filling are protected, can be effective in reducing the impact of the pathogens. It is also economically justified when yield potential is high. When using a foliar fungicide, it is important to protect the last two leaves since these leaves provide most of the energy needed to produce the grain. Protection of these leaves is best achieved by applying the fungicide between emergence of the flag leaf and the beginning of flowering (Eyal *et al*., 1987).

As an alternative to foliar applications, seed treatment has been investigated. Using disease free seed that has been treated with a recommended seed protectant fungicide is important. Seed treatment with systemic fungicides reduced pycnidiospores production in Victoria, Australia, for up to 3 months after sowing, though without a measurable increase in yield. The most effective chemicals for seed treatment were: thiabendazole (1.5 g/kg seed), triadimenol (0.3 g/kg seed), and nuarimol (0.2 g/kg seed), which reduced the number of plants infected with *S. tritici* by 62, 52, and 36%, respectively, but without improving yield (Eyal *et al*., 1987)

Fungicide seed treatments or early applications of foliar fungicides may offer some early season protection from STB, but will most likely not provide enough protection of the upper canopy during grain fill when the plants are most vulnerable to disease (Eyal and Wahl, 1975). Systemic fungicides with curative properties and longer protective action may be more beneficial than protectants. This is especially true when the action threshold is misjudged or the chemical protection program improperly executed. The systemic fungicides

benomyl (Benlate), prochloraz (Sportak), triadimefon (Bayleton), and propiconazole (Tilt) have proved effective in controlling STB in several countries. Other systemic fungicides, such as HWG 1608, fenpropimorph (Corbel), and myclobutanil (RH 3866), have also been found to be effective. Combining protectant and systemic fungicides to control septoria disease might provide an alternative route, since the systemic fungicides can lengthen the protection effect, counteracting outbreaks and timing difficulties, while the protectant fungicide reduces the selection pressure on the pathogen exerted by the systemic fungicides and expands the control spectrum and longevity of the control program (Eyal *et al*., 1987).

The rapid evolution of *Z. tritici* populations and their resistance development to fungicides, especially to the strobilurin class of chemicals, however, remained a major problem for chemical control. The first resistance of *Z. tritici* to these compounds was detected in 2002, and populations of the fungus in many regions are now nearly all resistant. Analysis of DNA sequence variation within populations showed that the strobilurin resistance was acquired independently through at least four recurrent mutations of the mitochondrial cytrochrome b gene. Hence, strobilurins are only being used in areas where resistance did not (abundantly) develop yet as these compounds also contribute to a longer green life of flag leaves and therefore to yield. However, the commonest fungicides currently being applied are azoles. The efficacy of these compounds is also decreasing due to mutations in the *cyp51* gene, although other factors also may be involved. Alternating fungicides with different modes of action helps mitigate the development of resistance (Ponomarenko *et al*., 2011). Among what makes the use of fungicides for the control of STB difficult is that under conditions favorable for disease, 2-12 fungicide applications are required to control STB (Burke and Dunne, 2008), which is actually costly for the poor.

Success in decreasing the impact of *S. tritici* on the expression of yield potential depends on integrating all components-cultural practices, epidemiology, genetic protection, chemical control, biological control, and extension services- into a disease management scheme that is part of the crop management system (Eyal, 1981). Integrated disease management, including cultivars with acceptable levels of resistance, crop rotation, seed treatments, different cropping and tillage systems and fungicides has been used by growers in Argentina (Simón *et al.*, 2013).

References

Abebe, T., Mehari, M. & Legesse, M. (2015). Field Response of Wheat Genotypes to Septoria Tritici Blotch in Tigray, Ethiopia. *Journal of Natural Sciences Research,* 5(1), 146-152.

Adhikari, T.B., Anderson, J.M. & Goodwin, S.B. (2003). Identification and molecular mapping of a gene in wheat conferring resistance to *Mycosphaerella graminicola*. *Phytopathology,* 93, 1158-1164.

Adhikari, T.B., Cavaletto, J.R., Dubcovsky, J., Gieco, J.O., Schlatter, A.R. & Goodwin, S.B. (2004b). Molecular mapping of the *Stb*4 gene for resistance to Septoria tritici blotch in wheat. *Phytopathology,* 4, 1198-1206.

Adhikari, T.B., Yang, X., Cavaletto, J.R., Hu, X., Buechley, G., Ohm, H.W., Shaner, G. & Goodwin, S.B. (2004a). Molecular mapping of *Stb*1, a potentially durable gene for resistance to Septoria tritici blotch in wheat. *Theoretical and Applied Genetics,* 109, 944-953.

Arraiano, L.S., Chartrain, L., Bossolini, E., Slatter, H.N., Keller, B. & Brown, J.K.M. (2007). A gene in European wheat cultivars for resistance to an African isolate of *Mycosphaerella graminicola*. *Plant Pathology,* 56, 73-78.

Arraiano, L.S., Worland, A.J., Ellerbrook, C. & Brown, J.K.M. (2001). Chromosomal location of a gene for resistance to Septoria trictici blotch (*Mycosphaerella graminicola*) in the hexaploid wheat 'Synthetic 6x'. *Theoretical and Applied Genetics,* 103, 758-764.

Brading, P.A., Verstappen, E.C.P., Kema, G.H.J. & Brown, J.K.M. (2002). A gene-for-gene relationship between wheat and *Mycosphaerella graminicola*, the Septoria tritici blotch pathogen. *Phytopathology,* 92, 439-445.

Brown, J.K.M., Chartrain, L., Lasserre-Zuber, P. & Saintenac, C. (2015). Genetics of resistance to *Zymoseptoria tritici* and applications to wheat breeding. *Fungal Genetics and Biology,* 79, 33-41.

Browning, J.A. (1979). Genetic protective mechanisms of plant pathogen populations: Their coevolution and use in breeding for resistance. In: MK Harris (ed). Biology and Breeding for Resistance. pp. 52-57.

Burke, J.J. & Dunne, B. (2008). Investigating the effectiveness of the Thies Clima ''Septoria Timer'' to schedule fungicide applications to control *Mycosphaerella graminicola* on winter wheat in Ireland. *Crop Protection,* 27, 710-718.

Chartrain, L., Berry, S.T. & Brown, J.K.M. (2005b). Resistance of wheat line Kavkaz-K4500 L.6.A.4 to Septoria tritici blotch controlled by isolate-specific resistance genes. *Phytopathology,* 95, 664-71.

Chartrain, L., Joaquim, P., Berry, S.T., Arraiano, L.S., Azanza, F. & Brown, J.K.M. (2005a). Genetics of resistance to Septoria tritici blotch in the Portuguese wheat breeding line TE9111. *Theoretical and Applied Genetics,* 110, 1138-1144.

Chartrain, L., Sourdille, P., Bernard, M. & Brown, J.K.M. (2009). Identification and location of *Stb*9, a gene for

resistance to Septoria tritici blotch in wheat cultivars Courtot and Tonic. *Plant Pathology,* 58, 547-555.

Cooke, B.M. & Jones, D.G. (1970). A field inoculation method for *Septaria tritici* and *S. nodarum. Plant Pathology,* 19, 72-74.

Cowling, S.G. (2006). Identification and Mapping of Host Resistance Genes to Septoria Tritici Blotch of Wheat. PhD Dissertation. University of Manitoba.

CSA (Central Statistical Agency of Ethiopia), (2017). Agricultural sample survey: Report on area and production of major crops (Private peasant holdings, Meher Season). Volume I Statistical Bulletin 584, Addis Ababa, Ethiopia. 122p.

Cuthbert, R.D. (2011). Molecular mapping of septoria tritici blotch resistance in hexaploid wheat (*Triticum aestivum* L.). PhD Dissertation. University of Manitoba, Winnipeg.

Eyal, Z. & Brown, M.B. (1976). A quantitative method for estimating density of *Septaria tritici* pycnidia on wheat leaves. *Phytopathology,* 66, 11-14.

Eyal, Z. & Wahl, I. (1975). Chemical control of septoria leaf blotch disease of wheat in Israel. *Phytoparasitica,* 3, 76-77.

Eyal, Z. (1981). Integrated control of septoria diseases of wheat. *Plant Diseases,* 65(9), 763-768.

Eyal, Z., Scharen, A.L., Prescott, J.M. & Ginkel, M. (1987). The Septoria Diseases of Wheat: Concepts and methods of disease management. Mexico, DF: CIMMYT. 54p.

Eyal. Z. (1971). The kinetics of pycnidiospore liberation in *Septoria tritici. Canadian Journal of Botany,* 49, 1095-1099.

FAOSTAT (Food and Agriculture Organization of the United Nations Statistics), (2018). [Online] Available: http://www.fao.org/faostat/en/#data/QC (January 9, 2018).

Flaishman, M., Eyal, Z., Voisard, C. & Haas, D. (1990). Suppression of *Septoria tritici* by Phenazine- or Siderophore-deficient Mutants of *Pseudomonas*. *Current Microbiology,* 20, 121-124.

Fones, H. & Gurr, S. (2015). The impact of septoria tritici blotch disease on wheat: An EU perspective. *Fungal Genetics and Biology,* 79, 3-7.

Goodwin, S.B. & Thompson, I. (2011). Development of isogenic lines for resistance to Septoria tritici blotch in wheat. *Czech Journal of Genetics and Plant Breeding,* 47, S98-S101.

Hailu, E. & Woldeab, G. (2015). Survey of Rust and Septoria Leaf Blotch Diseases of Wheat in Central Ethiopia and Virulence Diversity of Stem Rust *Puccinia graminis* f. sp. *tritici*. *Advances in Crop Science and Technology,* 3(2), 1-5.

Hershman, D.E. (2012). Septoria Diseases of Wheat. Online [Available]: http://www.ca.uky.edu/agc/pubs/ppa/ppa39/ppa39 (June 24, 2017).

Jing, H.C., Lovell, D., Gutteridge, R., Jenk, D., Kornyukhin, D., Mitrofanova, O.P., Kema, G.H.J. & Hammond-Kosack, K.E. (2008). Phenotypic and genetic analysis of the *Triticum monococcum-Mycosphaerella graminicola* interaction. *New Phytologist,* 179, 1121-1132.

Kasa, D., Hundia, B. & Dembel, W. (2015). Distribution and occurrence of wheat rusts and septoria tritici blotch in Bale and Arsi Zones, 2014 *Belg* season. *Global Journal of Pests, Disease and Crop Protection,* 3 (4), 124-130.

Kettles, G.J. & Kanyuka, K. (2016). Dissecting the Molecular Interactions between Wheat and the Fungal Pathogen *Zymoseptoria tritici*. *Frontiers in Plant Science,* 7, 508(7p.).

Kildea, S., Ransbotyn, V., Khan, M.R., Fagan, B., Leonard, G., Mullins, E. & Doohan, F.M. (2008). *Bacillus megaterium* shows potential for the biocontrol of septoria tritici blotch of wheat. *Biological Control,* 47, 37-45.

Krupinsky, J.M. & Tanaka, D.L. (2001). Leaf spot diseases on winter wheat influenced by nitrogen, tillage, and haying after a grass–alfalfa mixture in the conservation reserve program. *Plant Disease,* 85(7), 785-789.

Levy, E., Gough, F.J., Berlin, K.D., Guiana, P.W. & Smith, J.T. (1992). Inhibition of *Septoria tritici* and other phytopathogenic fungi and bacteria by *Pseudomonas fluorescens* and its antibiotics. *Plant Pathology,* 41, 335-341.

Liu, Y.Y., Zhang, L.L., Thompson, I.A., Goodwin, S.B. & Ohm, H.W. (2013). Molecular mapping re-locates the *Stb*2 gene for resistance to Septoria tritici blotch derived from cultivar Veranopolis on wheat chromosome 1BS. *Euphytica,* 190, 145-156.

Lucas, J.A. (1998). *Plant Pathology and Plant Pathogens*. (3rd ed.). Blackwell Science Ltd, Oxford: UK, (Chapter 14).

Lynch, K.M., Zannini, E., Guo, J., Axel, C., Arendt, E.K., Kildea, S. & Coffey, A. (2016). Control of *Zymoseptoria tritici*, cause of septoria tritici blotch of wheat, using antifungal *Lactobacillus* strains. *Journal of Applied Microbiology,* 121, 485-494.

McCartney, C.A., Brûlé-Babel, A.L., Lamari, L. & Somers, D.J. (2003). Chromosomal location of a race specific resistance gene to *Mycosphaerella graminicola* in the spring wheat ST6. *Theoretical and Applied Genetics,* 107, 1181-1186.

McKendry, A.L., Henke, G.E. & Finney, P.L. (1995). Effects of Septoria Leaf Blotch on Soft Red Winter Wheat Milling and Baking Quality. *Cereal Chemistry,* 72(2), 142-146.

Mojerlou, S., Safaie, N., Alizadeh, A. & Khelghatibana, F. (2009). Measuring and Modeling Crop Loss of Wheat Caused by Septoria Leaf Blotch in Seven Cultivars and Lines in Iran. *Journal of Plant Protection Research,* 49 (3), 257-262.

Nolan, S. & Cooke, B.M. (2000). Control of *Stagonospora nodorum* and *Septoria tritici* in wheat by pre-treatment with *Drechslera teres*, a non-host pathogen. *European Journal of Plant Pathology,* 106, 203-207.

Perelló, A., Mónaco, C. & Cordo, C. (1997). Evaluation of *Trichoderma harzianum* and *Gliocladium roseum* in controlling leaf blotch of wheat (*Septoria tritici*) under *in vitro* and greenhouse conditions. *Journal of Plant Diseases and Protection,* 104(6), 588-598.

Perelló, A.E., Moreno, M.V., Mónaco, C., Simón, M.R. & Cordo, C. (2009). Biological control of *Septoria tritici* blotch on wheat by *Trichoderma* spp. under field conditions in Argentina. *BioControl,* 54, 113-122.

Ponomarenko, A., Goodwin, S.B. & Kema, G.H.J. (2011). Septoria tritici blotch (STB) of wheat. *Plant Health Instructor. Department of Botany and Plant Pathology,* Purdue University, West Lafayette, IN USDAARS, Crop Production and Pest Control Research Unit, Purdue University, West Lafayette, IN Plant Research International, Wageningen, The Netherlands 2012. *The American Phytopathological Society*.

Raman, R., Milgate, A., Imtiaz, M., Tan, M.K., Raman, H., Lisle, C., Coombes, N. & Martin, P. (2009). Molecular mapping and physical location of major gene conferring seedling resistance to Septoria tritici blotch in wheat. *Molecular Breeding,* 24, 153-164.

Said, A. & Hussein, T. (2016). Epidemics of Septoria Tritici Blotch and its Development over Time on Bread Wheat in Haddiya-Kambata Area of Southern Ethiopia. *Journal of Biology, Agriculture and Healthcare,* 6(1), 47-57.

Scharen, A.L. & Krupinsky, J.M. (1978). Detection and manipulation of resistance to *Septoria nodorum* in wheat. *Phytopathology,* 68, 245-248.

Simón, M.R., Fleitas, M.C. & Schalamuk, S. (2013). Recent Advances on Integrated Foliar Disease Management with Special Emphasis in Argentina Wheat Production. In Tech, (Chapter 1). 32p.

Tabib, G.S.M., Faris, J.D., Friesen, T.L., Visser, R.G.F., van der Lee, T.A.J., Robert, O. & Kema, G.H.J. (2012). New broad-spectrum resistance to Septoria tritici blotch derived from synthetic hexaploid wheat. *Theoretical and Applied Genetics,* 124, 125-142.

Tabib, G.S.M., Robert, O., Laurent, V., Lonnet, P., Margale, E., van der Lee, T.A.J., Visser, R.G.F. & Kema, G.J. (2011). Genetic analysis of resistance to Septoria tritici blotch in the French winter wheat cultivars Balance and Apache. *Theoretical and Applied Genetics,* 123, 741-754.

Takele, A., Lencho, A., Kassa, B., Woldeab, G. & Hailu, E. (2015). Estimated Yield Loss Assessment of Bread Wheat (*Triticum aestivum* L.) due to Septoria Tritici Blotch (*Septoria tritici* Roberge in Desmaz) on Wheat in Holeta Agricultural Research Center, West Shewa, Ethiopia. *Research in Plant Sciences,* 3(3), 61-67.

Zadoks, J.C., Chang, T.T. and Konzak, C.F. (1974). A decimal code for the growth stages of cereals. *Weed Research,* 14, 415-421.

Zillinsky, F.J. (1983). Common Diseases of Small Grain Cereals: A Guide to Identification. CIMMYT, Mexico. 156p.

Table 1: Major genes conferring qualitative resistance to septoria tritici blotch of bread wheat, with their chromosomal locations, nearest markers, *Z. tritici* isolates with which they were identified and resistant source line

Gene	Located on Chromosome	Associated markers (distance to gene)	*Z. tritici* isolates used	Source line	Reference
Stb1	5BL	Xbarc74 (2.8cM), Xgwm335 (7.4cM)	IN95-Lafayette-1196-WW 1-4 and Purdue local (USA)	Bulgaria 88	Adhikari *et al.*, 2004a
Stb2	1BS	Xwmc406 (6cM), Xwmc230 (5cM)	Paskeville local (Australia) and IPO92034	Veranopolis	Liu *et al.*, 2013
Stb3	7AS	Xwmc83	Paskeville local isolate (Australia)	Israel 493	Goodwin and Thompson, 2011
Stb4	7DS	Xgwm111 (0.7cM)	IN95-Lafayette-1196-WW-1-4, I-89, IPBr1	Tadinia	Adhikari *et al.*, 2004b
Stb5	7DS	Xgwm44 (7.2cM)	IPO94269	Synthetic 6x	Arraiano *et al.*, 2001
Stb6	3AS	Xgwm369 (2cM)	IPO323	Flame, Hereward	Brading *et al.*, 2002
Stb7	4AL	Xwmc313 (0.3 to 0.5cM), Xwmc219 (1cM)	MG2 (Canada) and IPO87019	ST6	McCartney *et al.*, 2003
Stb8	7BL	Xgwm146 (3.5cM), Xgwm577 (5.3cM)	IN95-Lafayette-1196-WW 1-4	Synthetic W7984	Adhikari *et al.*, 2003
Stb9	2BL	Xfbb226 (3.6cM), Xwmc317, Xbarc0129	IPO89011	Courtot, Tonic	Chartrain *et al.*, 2009
Stb10	1D	Xgwm848	IPO94269 and ISR8036	Kavkaz-K4500	Chartrain *et al.*, 2005b
Stb11	1BS	Xbarc008 (1cM)	IPO90012	TE9111	Chartrain *et al.*, 2005a
Stb12	4AL	Xwmc219	ISR398 and ISR8036	Kavkaz-K4500	Chartrain *et al.*, 2005b
Stb13	7BL	Xwmc396 (7-9cM)	MG96-36, MG2 (Canada)	Salamouni	Cowling, 2006
Stb14	3BS	Xwmc500 (2cM), wmc632 (5cM)	MG2 (Canada)	Salamouni	Cowling, 2006
Stb15	6AS	Xpsr904 (14cM)	IPO88004	Arina, Riband	Arraiano *et al.*, 2007
Stb16q	3DL	Xgwm494 (4.3cM), Xbarc128 (9.9cM)	IPO88018 and IPO94218	SH M3	Tabib *et al.*, 2012
Stb17	5AL	Xhbg247 (3.1cM), Xgwm617 (38.3cM)	IPO88018	SH M3	Tabib *et al.*, 2012
Stb18	6DS	Xgpw5176, Xgpw3087	IPO323, IPO98022, IPO89011, IPO98046	Balance	Tabib *et al.*, 2011
StbSm3	3AS	barc321 (1.9cM)	MG96-36, MG2 (Canada)	Salamouni	Cuthbert, 2011
StbWW	1BS	Xbarc119b (0.9-4.1cM)	79, 2, 1A	WW1842, WW2449, WW2451	Raman *et al.*, 2009
TmStb1	$7A^{m}S$	Xbarc174 (23.5cM)	IPO323	MDR043 (*T. monococcum*)	Jing *et al.*, 2008