Barley Net Blotch Disease Management: A Review

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Abstract— *Barley (Hordeum vulgare L.) is one of the ancient grain crops cultivated and used worldwide. In Ethiopia, barley is among important staple crops next to tef, maize, wheat and sorghum mainly grown on about 1 million ha of land with average yield of 2.1t ha. It is the predominant cereal in the high altitudes and it accounts nearly 25% of the total production in Africa.*

The fungi Pyrenophora teres f. teres (Ptt) and P. teres f. maculata (Ptm) cause net form net blotch (NFNB) and spot form net blotch (SFNB) of barley, respectively. Net blotch is one of the most important barley diseases which reduce both quality and quantity of barley grain. Yield loss due to this disease reaches up to 100% in susceptible cultivars under severe epidemics. In Ethiopia, barley net blotch is among widely distributed and destructive diseases in cool highland areas and yield losses reaching about 67% have been recorded. Currently, the disease can be controlled using different approaches such as cultural, chemical and biological controls as well as using resistant cultivars of which development and deployment of resistant cultivars is the best management method. However, it is argued that using integrated disease management is one of the most important strategies that should be followed to reduce the effect of barley net blotch diseases. This review discusses recent information on economic importance, epidemiology, life cycle, host range, geographical distribution and disease management of barley net blotch disease. It also presents the barley net blotch disease management methods such as cultural, chemical, biological and use of host resistance methods. Under host resistance method, information on types of resistance, sources of resistance have been presented.

Keywords— Barley net blotch, Disease management, Methods, Cultural, Chemical, Host resistance.

I. INTRODUCTION

Barley (*Hordeum vulgare* L.) is one of the most important crops grown worldwide at an altitude ranging from 1400 to 4000 meters above sea level (Zemede, 2002). At global level, barley ranks fourth among cereal crops in both yield and hectare coverage after wheat, rice and maize (Munck, 1981). In Ethiopia, barley grown from 1500 to 3500 meters above sea levels predominantly as food crops (Berhane, 1996) and ranks fifth after teff, maize, sorghum and wheat (Abdi, 2011).Barley is staple food for many people globally, especially for poor households, in addition to its uses in malting and as an animal feed (Newton et al., 2011). Barley grain is used for the preparation of different foodstuffs in Ethiopia, such as malt production, injera, porridge, roasted grains; and different local drinks and the straw and stem stub are good source of feed for animals and roof thatching, respectively, in Ethiopia (Fenta, 2018).

Barley productivity is low (1.97 t/ha) in Ethiopia as compared to world average of 3.1 t/ha and the reduction in productivity of the crop is mainly directed to multidimensional abiotic and biotic stresses (EIAR, 2019). In Ethiopia, net blotch is one of the most important barley diseases causing significant yield and quality loss (Yitbarek et al., 1996). Diseases such as scald, net blotch, spot blotch and rusts, can reduce yields by up to 67% (Chilot, 1998). Net blotch is one of the most widespread and a common foliar disease of barley, occurring in all barley-growing regions of the world (Weibull et al. 2003). This disease is of economic importance worldwide and can cause yield losses ranging from a trace to 100%, but typically cause losses from 10% to 40% (Mathre, 1997). The disease occurs in two forms, *Pyrenophora teres* f. sp. *teres* causes the net-form of net blotch (NTNB) and *P. teres* f. sp. *maculata* causes the spot-form of net blotch (STNB).

II. ECONOMIC IMPORTANCE OF BARLEY NET BLOTCH DISEASE.

The ascomycete *P. teres* is the causal agent of net blotch on barley. During the last decades, *P. teres* has spread throughout the world and ravaged crops in many countries: Australia (Mclean et al., 2010), Canada (Akhavan et al., 2016), Europe (Plessl et al., 2005), South Africa (Campbell et al., 1999), the United States (Lartey et al., 2012), Ethiopia etc.

The most serious effect of net blotch disease is a reduction in the quality of barley grain ((Jayasena et al., 2007). Net blotch reduces grain carbohydrate content thereby reducing brewing quality (Kamul and Naguib, 1957) and yield (Smedegaard-Petersen, 1974). For instance, in Australia, the economic losses are estimated to be $ 117 × 106 per year (Murray and Brennan, 2010). In addition, yield losses might reach 40% in years with extensive rainfall in Germany (Plessl et al., 2005). Shipton (1966) reported a yield loss of 17% and a reduction in bushel weight, thousand-grain weight and grain size in net blotch-infected plots when compared to crops that were controlled by regular fungicide sprays. Jordan (1981) found that the greatest yield loss occurred when infection occurred at GS 30 (Zadoks *et al.*, 1974), before the end of tillering, causing a reduction in grain number, thousand-grain weight and an overall yield decrease of 19%. When infection occurred later (GS 45), yield was only reduced by 2.8%. These results agree with those of Rintelen (1969) who reported that with spring barley, yield losses of 20% resulted when plants were infected before tillering and losses of 10% resulted from when infection occurred between tillering and flowering. A reduction in leaf area, plant height and total weight result from seed infection (Wallwork *et al.*, 1995) and sowing infected seed enhances the losses associated with subsequent P. *teres* infections (Smedegaard-Petersen, 1974).

III. EPIDEMILOGY OF BARLEY NET BLOTCH DISEASE.

Pyrenophora teres can persist as mycelia in seed, rendering it seed-borne, but it may also survive in crop debris between growing seasons (Ma et al. 2004; Steffenson 1997). Seed-borne inoculum serves to introduce net blotch to new fields, whereas conidia and ascospores formed in fields with a history of net blotch epidemics are considered to be the most important sources of primary inoculum (Steffenson 1997). Disease development in barley seedlings by seed-borne mycelium occurs best at temperatures of around 10-15 °C. Inoculum in crop debris generally survives as pseudothecia on the surface of infected barley stubble from one season to another. When conditions become favorable, cool and moist for a sustained period of time, ascospores are produced from the pseudothecia. As many as 400 ascospores may be produced per square centimeter of surface area of stubble (McLean et al. 2009). Infection of barley by ascospores requires free surface moisture or high (95-100%) relative humidity. Each ascospore will germinate to form an appressorium and an infection peg, which subsequently penetrates the epidermis of the host. Differences in growth patterns of *Ptt* and *Ptm* have been reported inside the host. *Ptm* is reported to grow biotrophically, forming a vesicle intracellularly in epidermal cells before switching to intercellular necrotrophic growth, whereas, *Ptt* does not have the initial biotrophic growth phase (Hysing and Wiik 2013; Lightfoot and Able 2010).

Ascospores are considered to be the primary inoculum driving net blotch epidemics, and they may be aerially or splash dispersed to initiate infection (Mathre, 1982). Conidia, which also produced on infected tissue in stubble, may also serve as primary inoculum (Mathre 1982), although they usually serve as a source of secondary inoculum when they are produced on mature and senescent leaves in the later part of the growing season (Jordan 1981). Sporulation occurs on conidiophores formed on the surface of the primary lesions when the relative humidity is near 100% (Mathre 1982). Conidial sporulation is diurnal, with light promoting sporulation. An eighteen hour light period is generally enough to stimulate spore production from the conidiophores at temperatures between 15 °C and 25 °C (Mathre 1982). Necrosis and chlorosis can occur within a distance of 10 cells from the hyphae, which results in the characteristic necrotic net-like pattern in *Ptt* infected plants. The symptoms are believed to be caused at least partly by necrotrophic effectors secreted by the pathogen that induce programmed cell death. Sarpeleh et al. (2007) hypothesize that proteinaceous metabolites are responsible for the necrotic symptoms, while low molecular weight compounds produce the chlorosis. Neupane et al. (2015) attributed the high variability in symptoms caused by different isolates on the same host or by the same isolate on different hosts to different necrotrophic effectors and their effect of different host genotypes.At the end of the growing season, the fungus colonizes the senescent tissues and forms the sexual stage, which overwinters and initiates infection in the following year (McLean et al. 2009; Liu et al. 2011).

IV. LIFE CYCLE OF BARLEY NET BLOTCH PATHOGEN

The life cycles of *Pyrenophora teres* f. *teres* and *Pyrenophora teres* f. *maculata* are almost identical and involve both asexual and sexual stages. Both *Ptt* and *Ptm* are residual borne pathogens that can overwinter as pseudothecia (sexual fruiting bodies)

or conidia on plant stubbles (Mathre, 1997). It takes up to six months to develop a fertile pseudothecia under field condition when temperatures range between 10-15 ^{0}C (Shipton et al., 1973). However, it takes about two months under laboratory conditions to form pseudothecia. Following the growing season, pseudothecia actively release ascospores as far as 35 cm into the air, which act as a primary source of inoculum (Jordan, 1981). Alternatively, the mycelia and conidia that overwinter on plant stubbles or infected seed may also serve as a primary source of inoculum (Shipton et al., 1973). The ascospores germinate under 95-100% relative humidity, form appresoria, and produce penetration pegs that directly penetrate host epidermal cells to initiate intracellular growth and colonization (Hargreaves and Keon, 1983). After successfully infecting the host, *Pyrenophora teres* produces conidia throughout the growing season in multiple cycles (polycyclic), which serves as a source of secondary inoculum. Conidia are often disseminated via rain splash and wind to neighbouring plants or fields (Mathre, 1997). Towards the end of the growing season, either pseudothecia are developed on the plant stubble, or conidia and mycelia overwinter on stubble or infected kernels, which serves as a primary source of inoculum for the next growing season. However, only *Ptt* has been shown to transfer across generations via infected seed to the subsequent growing seasons (Mathre, 1997).

V. HOST RANGE OF BARLEY NET BLOTCH PATHOGEN

Barley (*Hordeum vulgare* (L.)) and its wild progenitor (*Hordeum spontaneum L*., are the primary hosts of *P. teres*, although the pathogen may also infect a few wild relatives of barley including (but not limited to): *Hordeum marinum* (Hudson), *Hordeum murinum* (L.), *Hordeum brachyantherum* (Nevskii), and *Hordeum distichon* (L.). *P. teres* can also attack other more distantly related species, including *Avena sativa* (L.) (oats), *Avena fatua* (L.) (common wild oat) and *Triticum aestivum* (L.) (bread wheat) (Liu et al.2011).

VI. GEOGRAPHIC DISTRIBUTION OF BARLEY NET BLOTCH DISEASE

Steffenson (1997) reported that net blotch of barley occurred in most barley-growing regions of the world, but was most severe in temperate regions of high rainfall and humidity. According to Steffenson (1997) *P. teres* is present in countries of Europe, Asia, Africa, Americas and Oceania, as follows: **Asia:** Afghanistan, Armenia, China, India, Iran, Iraq, Israel, Japan, Republic of Korea, Kyrgyzstan, Myanmar, Nepal, Pakistan, Turkey, Turkmenistan, Uzbekistan. **Europe:** Austria, Baltic States, Bulgaria, Cyprus, Denmark, Faroe Islands, Finland, Former USSR, Former Yugoslavia, France, Germany, Greece, Ireland, Italy, Malta, Moldova, Netherlands, Norway, Poland, Romania, Russia, Spain, Sweden and United Kingdom. **Africa:** Egypt, Ethiopia, Kenya, Libya, Morocco, Saint Helena, South Africa, Tanzania, Tunisia, Zambia. **North America:** Canada, Mexico, USA. **South America:** Argentina, Brazil, Colombia, Peru, Uruguay. **Oceania:** Australia, New Zealand.

VII. BARLEY NET BLOTCH DISEASE MANAGEMENT

There are several methods to reduce yield losses due to foliar diseases such as barley net blotch disease. For example, crop rotation, fungicide application, and the deployment of resistant cultivars can be used to manage net blotch of barley (Turkington *et al.*, 2015). However, integrated pest management is one of the most important strategies that should be followed to reduce the effect of plant diseases in crops. A promising approach to achieve this aim while minimising use of pesticides is to apply and combine different agriculture practices that contribute to increasing crop yield by decreasing plant diseases directly or indirectly. For instance, combining good crop hygiene practices, the use of resistant cultivars and chemical control (both as seed dressing and foliar applications), is currently the most effective net blotch disease management strategy.

7.1 Cultural Control

There are three sources of primary inoculum for barley net blotch disease; infected seeds, crop debris, and straw residue. Therefore, the first step to control net blotch is the deletion of the primary inoculum of *P. teres* by sowing healthy seeds (Jalli, 2011).Use of certified clean seed is vital as seed-borne inoculum has the ability to contaminate straw on which it will produce abundant inoculum in the following year (Piening, 1968). It is well-established that seed-borne inoculum is a source of primary inoculum for both forms of the pathogen (i.e., NFNB & SFNB); conidia, perithecia and sclerotia of *P. teres* can be found on the surface of the seeds (Jordan, 1981). Although barley straw is a major source of inoculum, the seeds contribute to the introduction of the pathogen into plots which were previously free from disease (Youcef-Benkada *et al.*, 1994), highlighting the importance of using clean certified seed.

Cropping practices such as sowing date, application of nitrogen, the rogueing of diseased plants and the use of conventional tillage all affect net blotch disease development. Early sowing means a longer growing season with increased exposure to

carry-over inoculum. Late sowing results in an increase in yield, thousand grain weight and seed number per head when compared with early sowing (Delserone and Cole, 1987). Application of nitrogen fertilizer ensures high yield and quality; however it may favour disease development (Locke *et al.* 1981). Nitrogen application results in increased relative humidity within the crop canopy which favours the development of disease (Jordan and Hutcheon, 1999).

After harvesting barley kernels, debris and straw residues are other sources of primary inoculum. A study has demonstrated that amounts of residue infested can increase disease intensity and thus reduce the yield (Adee, 1989). Destruction of infested barley residues is often suggested as a means to eliminate potential sources of primary inoculum that initiate net blotch epidemics. In contrast, incidence of net blotch can increase due to retention of stubble Jordan and Allen, 1984). This leads to a build-up of inoculum that is present on straw debris from previous crops (Jordan, 1981; Jordan and Allen, 1984). Removal of inoculum may be earned out in a number of ways; deep ploughing, shallow cultivations, burning, etc. Piening (1968) found that 42% net blotch infection resulted from a plot where the straw and stubble were lightly diced, compared to only 8% infection where the stubble had been ploughed under. Ascospores of the pathogen were responsible for half the net blotch lesions produced on volunteer barley plants (Piening, 1968). In the past, burning was an important and effective method for the eradication of inoculum sources; however it is not environmentally acceptable as it may be detrimental to nesting birds, cause smoke pollution and removes organic matter from the soil.

In addition to these measures to limit the sources of inoculum, preventive agronomic measures play an essential role in the management of net blotch. Crop rotation is beneficial to reduce the severity of the pathogen. Crop rotation can help minimize plant disease potential by reducing populations of disease organisms surviving on crop residues. Although crop rotation reduces the risk of many cereal diseases, it does not eliminate them. Crop rotation is essential for the control of this disease because mono-cropping encourages build-up of pathogen populations (Pusey, 1996). Crop rotation also benefits the soil by maintaining a balance of nutrients and improving soil structure (Pusey, 1996). A minimum of 2 years between barley crops is required to prevent net blotch (Duczek et al., 1999).

7.2 Chemical Control

Fungicides are one of the most common and widely known methods used to minimise the effect of net blotch. However, fungicides can have adverse effects on the environment. In addition, the continual use of chemicals can lead to increases in resistant strains of pathogens (Vinale *et al*., 2008a). The aim of fungicide application is to maximise the green leaf area of the top three leaves during grain filling; in barley it is the 2^{nd} and 3^{rd} leaves and flag 2) that are most important (Weppler and Hollaway, 2004).

Chemical control to manage net blotch has generally focused both on the application of foliar fungicides, and seed treatments (Hysing and Wiik 2013). Foliar application of fungicides to the upper leaves during grain filling provides effective chemical control. Whilst applications at early growth stages are generally not economically justifiable (Liu et al. 2011). Seed treatments have been found effective in reducing net blotch incidence (Hampton, 1980). However, seed treatments alone are not considered reliable for the management of net blotch (Hysing and Wiik 2013). The effectiveness of control using foliar fungicides varies depending on factors such as degree of disease pressure, mode of action of the active ingredient, application rate, timing and number of applications and the presence of reduced sensitivity or resistance in the pathogen population (Van den Berg and Rossnagel 1990).

One widely adopted strategy to manage barley net blotch disease is to apply foliar fungicide to the infected crop at predetermined plant growth stages to protect the flag leaf and the emerging ear from infection. This strategy aims to protect the photosynthetic potential of the top four leaves which can contribute 72% of the total yield Paveley *et al.* 2000).

Fungicides of the quinone outside inhibitors (QoI), the succinate dehydrogenase inhibitor (SDHI), and azole or demethylase inhibitor (DMI) classes are used as site-specific systemic fungicides (Mair et al., 2016). The foliar fungicide application effectiveness to control net blotch has been largely carried out (Sutton and Steele, 1983; Mclean et al., 2009). First studies have shown that triazole-based fungicides by pulverization allowed to control net blotch (Sutton and Steele, 1983; Van Den Berg and Rossnagel, 1990). Triazoles, known as DMI (propiconazole and prothioconazole), inhibit dimethylation between substrates that are necessary for the biosynthesis of ergosterol in fungi. In addition, SDHIs are also used to reduce the disease severity.

The strobilurins, a new class of broad-spectrum fungicides, have been adopted recently for net blotch control (Bartlett et al., 2002). Strobilurin fungicides were inspired by natural fungicidal derivatives of β-methoxyacrylic acid (Bartlett et al., 2002). Belonging to QoI (pyraclostrobin and picoxystrobin), strobilurins are natural substances isolated mainly from fungi and more

specifically, Basidiomycetes. The strobilurin name is derived from the fungi genera Strobilurus (Balba, 2007). First introduced to the market in 1996, strobilurins inhibit mitochondrial respiration by blocking electron transfer at the level of cytochromes b and c (Gisi et al., 2002; Balba, 2007).

The antifungal efficacies depend also on the period of their application and how they are applied, as well as on the plant growth stage (Van Den Berg and Rossnagel, 1990). Seed treatments were successful if applied early in the season corresponding at Zadoks growth stage 23–24, but less at later growth stages (Martin, 1985). Barley seeds are considered as a source of inoculum for the *ascomycete P. teres.* The severity of the barley net blotch is reduced when a fungicide seed treatment is applied (Martin, 1985). Seed treatment effectiveness depends on fungal sensitivity, chemical fungitoxicity, and seed coverage quality. Iprodione is the fungicide providing the best control of dematiaceous fungi (Bipolaris and Drechslera) on seeds (Reis et al., 2012). Another study demonstrates the efficiency of one application of propiconazole at spike emergence for the management of net blotch (Sutton and Steele, 1983). Seed infected with *P. teres* f. *teres* can be treated with thiram to reduce carry over by inhibiting spore and mycelia growth. Unfortunately, no seed fungicide is currently available for management of SFNB (Wallwork 2011).

A correct application of fungicides before the emergence of the flag leaf and the ear aims to protect the photosynthetic potential of the top four leaves, which contribute to 72% of the total yield (Mclean et al., 2009). A single application of propiconazole is not enough when the pathogen progresses quickly. A recent study demonstrates that two applications with the combination of pyraclostrobin and epoxiconazole improved net blotch control and increased the yield in two experimental years (Stepanovic et al., 2016). Belonging to QoI, metyltetraprole is a new fungicide, which is effective against important cereal diseases, including net blotch (Suemoto et al., 2019). Further, the metyltetraprole suppresses succinate-cytochrome c reductase activity in QoI susceptible *P. teres.* With time, resistant strains to these products have emerged (Jørgensen and Olsen, 2007). In Europe and in Australia, *P. teres* developed a resistance to DMI fungicides (Peever and Milgroom, 1993; Rehfus et al., 2016). Shortly after the first QoIs uses, resistant isolates to these antifungal products were detected in field populations (Gisi et al., 2002).

More specifically, in 2003, resistance to QoI fungicides in *P. teres* was detected in France, Sweden, and Denmark. The resistance mechanism to QoIs has been identified as mutations in the mitochondrial target gene, cytochromes b (Sierotzki et al., 2007). In *P. teres,* this mutation has been described as a substitution of phenylalanine to leucine at amino acid position 129 (Sierotzki et al., 2007). To conclude, the fungicide exerts a selection pressure, which leads to the selection of isolates, which have a mutation providing fungicide resistance, while susceptible isolates will be eliminated. There is a subsequent increase in the number of resistant individuals in the population. Successive rounds of fungicide use repeat the selection of resistant isolates, which leads to the increase of the resistance mutation in the population each time the fungicide is used. Eventually, the resistant isolates will dominate the population and the effectiveness of the fungicide will be reduced (Gisi et al., 2000). To reduce the risk of fungicide resistance development, the use of recommended fungicide rates is important as the application of low rate doses can increase the likelihood of fungicide resistance emerging in *P. teres* population. Also, the use of a mixture of fungicides (of different modes of action) to control net blotch disease is desirable, as it reduces the risk of fungicide resistance emerging in *P.teres* populations and the usage of fungicide mixtures can reduce the number of fungicide applications required throughout the growing season (Whitehead, 2004).

In addition to the resistance among several fungal species, azoles use has also been affected by a restriction with a wide range of significant toxicities, including hallucinations, hepatotoxicity, and QTc prolongation (Gintjee et al., 2020). Faced with these problems, varietal selection, preventive agronomic measures, and biocontrol agents might be considered as alternative solutions to fungicides products.

Generally, Present-day control of net blotch disease of barley relies on the use of seed dressing and foliar formulations of fungicides, with current chemical groupings available for control of net blotch including strobilurins, triazoles, benzimidazole, chlorothalonil, morpholine, chlorophenyl, anilinopyrimidine, guanidine, carboxamide and dithiocarbamates (Whitehead, 2004).

7.3 Biological Control

Biological control or 'biocontrol' is defined as a strategy for reducing disease incidence or severity by direct or indirect manipulation of microorganisms (Maloy, 1993). Use of non-pathogenic fungi and bacteria as biological control agents for management of pathogenic fungi are increasingly being investigated as alternatives to chemical foliar and seed fungicides (Whipps and McQuilken 2009). Although biocontrol offers a positive alternative to chemical pesticides, the overall

contribution of biocontrol represents about 1% of agricultural chemical sales (Lidert, 2001). There are currently 13 bacteria and 12 fungi registered with the US Environmental Protection Agency which can be sold as biocontrol agents against plant disease (Fravel, 2005).

While several researchers have investigated the potential of bacteria and fungi to control cereal diseases, presently, *Pseudomonas chlororaphis* strain MA 342 marketed as Cedomon is the only biocontrol agent commercially available for the control of net blotch disease of barley (Copping, 2004). This bacterium is formulated as a seed treatment containing 1 X 10^6 colony forming units (cfu) per gram. *Pseudomonas chlororaphis* MA 342 competes with pathogens for nutrients and space, encourages the plant's natural defence system, promotes the development of roots and shoots (Copping, 2004) and suppresses fungal growth by producing the antifungal compound 2,3-deepoxy-2,3-didehydrorhizoxin (Hokeberg, 1998). This product is commercially available in the United States, Sweden, Norway, Finland and Austria. It is anticipated that more biological agents will be registered for the control of net blotch disease, especially for use in the growing organic cereal production tillage sector. An important criterion for the selection of such agents will be to ensure that they are adapted to the climate and soil in which they are to be used (Leyns *et al.,* 1990; de Bruyne *et al.,* 1991; Hokeberg *et al.,* 1997). The mechanisms of pathogen suppression by bacteria include the production of antimicrobial substances, induced resistance, competition between the biocontrol microorganism for nutrients and plant surface area (Weller, 1988; Pedersen *et al.,* 1999) and competition for iron through the production of siderophores (Whipps, 2001; Baaker *et al.,* 1993). These mechanisms need not be exclusive, and it is desirable that any new biocontrol agents developed for the control of net blotch disease possess as many of these attributes as possible. Also any biological control agent should be formulated so that it has a long and stable shelf life, is easy to apply and is active once applied and multiplies on the plant surface.

Ali-Haimoud *et al.* (1993) observed that mycelial suspensions and culture filtrates of several fungal isolates (strains of *Trichoderma koningii, T viride, Ipseudokoningii* and of two unidentified fungi) and of the actinomycete *Micromonospora* spp. significantly inhibited the *in vitro* formation of sclerotioid organs on *P. teres* var. *teres*- and var. mocw/ato-inoculated barley straw. Sclerotioid organs are important survival and reproductive structures (Ali- Haimoud *et al.,* 1993). Both the mycelium and culture filtrate of *T.viride* and *T. pseudokoningii* also significantly inhibited sclerotioid organ germination on barley straw, whether applied pre- or post- *P. teres* inoculation. The spot form of the pathogen was generally more sensitive to the culture filtrates than was the net form. However, Amundsson and Hokeberg (1984) found that several known antagonists, including *Trichoderma* spp., *Serratia* spp. and various strains of *Pseudomonas fluorescens,* were not effective against *P. teres.* More recently, Hokeberg *et al.* (1997) found that *Pseudomonas chlororaphis* strain MA 342 seed treatment resulted in a > 98 % reduction in the incidence of *P. teres*-infected plants derived from pathogen-inoculated seed grown under field conditions. The disease control and yield increases resulting from this bacterial seed treatment were similar to those achieved by treating seed with the fungicide Panoctine Plus 400 (guazatine and imazalil). This bacterium also suppressed common bunt of wheat under field conditions, had a shelf life of up to six weeks and freezing did not influence its biocontrol efficacy.

7.4 Host Plant Resistance

Disease resistance is an important agronomical trait in all crop plants and the use of resistant cultivars is often the most economically and environmentally friendly means to control a disease. The same holds true for barley net blotch disease control (Shipton *et al.,* 1973).

Research on net blotch resistance dates back to the 1920s when Geschele (1928) discovered that it followed Mendelian inheritance. By the end of the 1950, the presence of at least three genes conferring incomplete dominant resistance was known (Mode and Schaller 1958). The first resistance loci that could be localized in the genome were found by Bockelman et al. (1977) on chromosomes 1H, 2H and 3H in the cultivars Tifang, CI7584 and CI9819. Based on these early studies, net blotch resistance was mainly understood as a gene-for-gene relationship involving major-effect genes. In the late 1980s and early 1990s, a number of studies were conducted on adult plants, which found that resistance was quantitatively inherited under field conditions (Steffenson et al. 1996). With recent advances in molecular marker techniques, the location of resistance loci can be determined in a much more exact way, and we have learned that the mechanisms underlying this pathosystem are much more complex than initially thought. Today, resistance genes/QTL is known on all seven chromosomes, and many of them are specific to either *Ptt* or *Ptm* (Liu et al., 2011 and McLean et al., 2009). Many of these QTL have been projected onto consensus maps, which facilitate the comparison of loci across different studies and populations (Richards et al. 2017). The majority of the resistance QTL found in these mapping studies confer dominant resistance, but a number of recessive resistance genes have also been identified. Ho et al. (1996) showed that resistance to

two *Ptt* isolates in the Leger x CI9831 mapping population is conferred by one and three recessive resistance genes, respectively. Abu Qamar et al. (2008) detected two dominant susceptibility loci on chromosome 6H in the Rika x Kombar mapping population that are linked in repulsion and confer susceptibility to the *Ptt* isolates 15A and 6A, respectively. In a mapping population of the parental isolates 6A and 15A, Shjerve et al. (2014) identified four putative virulence genes, two of which confer virulence on Rika and two on Kombar, and hypothesized that the previously identified 6H region contains four closely linked susceptibility genes. The locus was subsequently fine-mapped to a 0.24 cM interval in the centromeric region of 6H (Richards et al. 2016).

Chromosome 6H is considered a hotspot for both major resistance genes and small-effect QTL, although the exact number of loci still remains to be determined (Abu Qamar et al. 2008; Friesen et al. 2006a; Steffenson et al. 1996). Some of the genes found on 6H are pathotype-specific (Abu Qamar et al. 2008; Friesen et al. 2006b). Chromosome 6H also harbors the first putative susceptibility gene to a *Ptt* NE (Liu et al. 2015). This QTL named SPN1, which was identified in the Hector x NDB112 mapping population after inoculation with the *Ptt* isolate 0-1, explained 31% of the phenotypic variation. The same QTL was also found after infection with five other globally collected *Ptt* isolates, indicating that isolates producing the corresponding NE may be found around the world. It remains to be elucidated whether other known dominant susceptibility genes also encode susceptibility to NEs. No NEs have been identified in *Ptm* yet, but it seems likely that this form also secretes them, most likely during later stages of infection. Both chromosomes 3H (Liu et al. 2015), and 7H are also considered hotspots for large-effect resistance QTL (König et al. 2014).

In the last years, it has become feasible to genotype large populations with thousands of SNP markers and GWAS has gained popularity in plant pathology. Currently, there are three GWA studies on *Ptm* resistance and one on *Ptt* resistance, reflecting the increasing importance of *Ptm* in many regions worldwide. The continuous distribution of disease severity in populations and the presence of between eight and 29 QTL per population underline the quantitative nature of resistance mechanisms in the patho-system (Burlakoti et al. 2016).

Most of these studies are performed on seedlings under controlled growth conditions, and more knowledge is required about how the resistance found in these studies holds up under field conditions (Williams et al. 2003), where genotype x environment effects may play a major role. Many studies found QTL that confer resistance consistently in both seedling and adult plants under field conditions (Cakir et al. 2003), but some of the resistance was specific to a developmental stage. In a GWA study on four Australian breeding populations, 75% of the QTL conferred resistance both in seedlings and adult plants, while 17% were only effective in adult plants and 7% in seedlings only.

Sato and Takeda (1997) identified *P. teres* resistance in many *Hordeum* species, especially in *H. spontaneum*, which thus constitutes an interesting source for improved resistance, provided that closely linked markers are available. Progress is currently made in characterizing the genomes of wild relatives of barley (Wendler et al. 2014), and a NAM population generated from a cross between *H. spontaneum* and *H. agriocrithon* and the cultivar Barke is currently being used in a GWA study to map resistance to *P. teres* (Vatter et al. 2016).

Apart from the aforementioned putative effect or Ptt NE1 and the putative virulence genes in the *Ptt* isolate 6A and 15A, little is known about genes conferring virulence or avirulence in the pathogen. Lai et al. (2007) identified the locus AvrHar conferring avirulence to the cultivars Tifang and Canadian Lake Shore in the isolate 15A and the loci AvrPra1 and AvrPra2 conferring virulence to the cultivar Prato in the isolate 0-1. AvrHar and AvrPra2 co-segregate, but it is currently not known if these loci are alleles of the same gene or two different genes.

7.4.1 Types of host plant resistance

The use of resistant cultivars is a very important means to control fungal pathogens and can have a direct impact on yield (Turkington et al. 2006; Østergård et al. 2008). Plant resistance is usually divided into two different forms.

7.4.1.1 Race-specific resistance

Race-specific resistance also termed monogenic, qualitative or vertical resistance, is effective against one or a few races of the same pathogen species (Van der Plank 1968). Our classical understanding of disease resistance follows the gene-for-gene model, according to which pathogens produce virulence gene products that interact with corresponding receptors in the plant (Flor 1956; Flor 1971). If the receptor is able to recognize the pathogen molecule, a defense response often involving a hypersensitive reaction will be elicited to ward off the pathogen (incompatible reaction). If no recognition occurs because one of the gene products is missing, the pathogen will be able to evade recognition by the immune system and infect the plant

(compatible reaction) (Jones and Dangl 2006). Whereas this type of defense is largely effective against biotrophic pathogens, some necrotrophic pathogens have evolved NEs to deliberately induce a hypersentitive response, so that the pathogen can thrive on the dead plant tissue (Tan et al. 2010). NEs have been extensively studied in pathogens related to *P. teres* such as *Parastagonospora nodorum* and *Pyrenophora tritici-repentis*, the causal agents of *Septoria nodorum* blotch and tan spot in wheat, respectively (McDonald et al. 2013)

7.4.1.2 Race non-specific resistance

Race non-specific resistance also termed polygenic, quantitative or horizontal resistance,is usually effective against all races of a pathogen species and is usually governed by several genes, most of them with small effects (Clair 2010). These genes often encode pathogenesis-related (PR) proteins, phytoalexins, etc. (Golshani et al. 2015) or developmental and morphological features (Melotto et al. 2006). Genomic regions harboring loci that affect quantitative traits are termed quantitative trait loci (QTL).

Since quantitative resistance is conferred by a number of genes, it is usually more stable since many mutations in the pathogen population are required to overcome this resistance (McDonald and Linde 2002). Quantitative resistance is often dependent on environmental factors (genotype x environment effects), and often only effective in certain growth stages or plant tissues (Steffenson et al. 1996).

7.4.2 Sources of host plant resistance

The use of genetic resistance is the most cost effective and environmentally desirable method of controlling yield and quality losses caused by barley net blotch disease; however, new and relevant sources of resistance are essential to make this strategy feasible. New sources of disease resistance are needed in developing barley (Hordeum vulgare L.) cultivars that are resistant to current pathotypes of the barley net blotch disease. The introduction of new sources of resistance will also increase genetic diversity and enhance the durability of resistance.

Studies of landraces for resistance to net blotch have been carried out by many scientists (Schaller and Wiebe, 1952; Buchannon and McDonald, 1965; Gaike, 1970; Smirnova and Trofimovskaya, 1985; Lukyanova, 1990; Faiad et al., 1996). Sato and Takeda (1994) studied the variation of host resistance of 2233 accessions of the barley world collection and found sources of resistance in accessions from Ethiopia, North Africa and Korea. New sources with resistance to up to eight races of *P. teres* were found among Peruvian landrace accessions (Afanasenko et al., 2000).

Thirteen sources of host resistance to *P. teres* f. *maculata* have been investigated in worldwide barley germplasm. Several feed barley varieties are resistant to SFNB because they have the *Ha4* allele for cereal cyst nematode resistance (Vanstone *et al.* 2008), which is associated with SFNB resistance (Arabi *et al.* 1992).

Breeding for resistance to SFNB will be challenging as resistance sources can have either major or minor effect and are usually conferred by multiple genes found on different chromosomes. Minor or 'partial' resistances are typically only effective for part of the crop's developmental stages and provide a moderate resistance, while major or 'complete' resistances are effective throughout the crop's life. Liu *et al.* (2010) have recently summarised the resistances that have been characterised for *P. teres* f. *maculata* and *P. teres* f. *teres*, noting that they are spread across several chromosomes.

In some cases resistance to both forms of *P. teres* were identified in the same regions of the barley genome (Manninen *et al.* 2006). However, the majority of resistances appear to be independent and appear in different barley lines which will mean considerable effort will be required to combine resistance to multiple pathogens in a single parental breeding line.

Several genes that confer partial resistance and are effective at seedling stages have been mapped in Australian barley germplasm. The first to be mapped was designated *Rpt4*, which is on the long arm of chromosome 7H in the variety Galleon (Williams *et al.* 1999). Since then, *Rpt4* has also been identified in breeding lines and varieties such as CI9214, Keel and Tilga (Williams *et al.* 1999; Williams *et al.* 2003). Breeding programs in Australia initially used *Rpt4* as a source of resistance to SFNB, however, this resistance has since been utilised less due to a lack of expression at adult stages of plant development (Williams *et al.* 1999). Other seedling resistance genes were tentatively identified in breeding lines by Williams *et al.* (2003) at various locations on chromosomes 7H, 4H and 2H. A resistance gene in variety Chebec was located at or near *Rpt4*. Weak association of seedling resistance was observed on chromosomes 1H and 3H in the breeding line VB9104 (Williams *et al.* 2003). Effective seedling resistance has been mapped to chromosomes 7H, 6H, 4H and 3H in two Canadian-derived barley varieties, TR250 and TR251 (Gupta *et al.* 2006). Friesen *et al.* (2006) also identified a major resistance gene on chromosome 4H in barley variety Q21861, which confers moderate resistance.

Genes that confer complete or adult resistance to SFNB have more recently been targeted by barley breeders and researchers in favour of seedling resistance (Williams 2003). One particularly useful complete resistance locus has been identified in association with the *Ha4* allele, originally derived from the Egyptian land race CI3576 (Arabi *et al.* 1992). This resistance locus is associated with resistance to cereal cyst nematode (*Heterodera avenae*) and has been mapped to the 5H chromosome in a Galleon/Haruna Nijo cross (Williams *et al.* 2003).

Sources of adult resistance have been identified on chromosome 7H in the breeding line VB9104 (Williams *et al.* 2003), while a Galleon/Haruna Nijo cross revealed interactions on chromosomes 7H, 5H and to a minor extent 4H (Williams *et al.* 2003). The variety Keel appears to possess alleles associated with adult plant resistance on chromosomes 5H, 7H, 1H, 2H and 4H (Arabi *et al.* 1992). Good levels of resistance have been identified in three Canadian six-rowed barley varieties, Leduc, Argyle and Bedford; this resistance is highly expressed at the seedling stage and at intermediate levels at adult stages (Tekauz 1990). These loci are not mapped and may correspond to those in other varieties. Multiple gene loci contribute to a seedling resistance and moderate level of adult resistance in the Canadian breeding lines TR250 and TR251 and the genes have been mapped to chromosomes 7H, 6H and 4H (Gupta *et al.* 2006). The resistance gene designated as *Rpt6* in the barley breeding line CI9819 on chromosome 5H was found to only be effective against limited pathotypes of *P. teres* f. *maculata* (Manninen *et al.* 2006).Ho *et al.* (1996) identified a potential dominant gene resistance in a doubled haploid population of crosses between varieties Leger and CI9831. However, further analysis showed that the resistance was actually due to two genes (Molnar *et al.* 2000). Other potential sources of resistance have been identified in European spring barley varieties Agneta, Clermont, Nordel, Arve, Tellus, Pamina, Albert and Birka (Jorgensen *et al.* 2000). However, the genes involved have not been identified.

Wild relatives can provide novel sources of resistance that are not present in adapted germplasm and may provide good resistance to multiple pathogens from a single line. Effective resistance toward the anamorph stage, *Dreschlera teres,* has been reported in *Hordeum vulgare* subsp. s*pontaneum* accessions Gay and Leon 2004). For many of the wild relatives of barley known to possess resistance to *P. teres* f. *maculata*, the chromosomal locations of resistance loci are yet to be mapped and their responses to the worldwide population of *P. teres* f. *maculata* are yet to be investigated. These sources may yield some effective resistances, but efforts will need to be made to incorporate them into breeding lines with agronomically adapted backgrounds.

Using different molecular techniques, several studies have identified net blotch resistance genes or quantitative trait loci (QTL) on all seven barley chromosomes (Mode and Schaller, 1958; Steffenson et al., 1996; Clare et al., 2020). Major QTL have been identified on barley chromosomes 1H (Manninen et al., 2006), 2H (Tamang et al., 2019), 3H (Graner et al., 1996), 4H (Islamovic et al., 2017), 5H (Manninen et al., 2006) 6H (Adawy et al., 2013), and 7H (Mclean et al., 2009; Tamang et al., 2019). Localized on chromosome 6H, the Rpt5 locus has been reported by several studies and is considered to be essential in the P. teres f. teres – barley interaction (Clare et al., 2020). According to several studies, the majority of the markers significantly associated with NFNB resistance localize to the centromeric region of chromosome 6H (Richards et al., 2016). In the same way, the high-resolution mapping of a dominant susceptibility locus located in the centromeric region of barley chromosome 6H has been described using markers (Richards et al., 2016). In addition, the Rpt7 locus confers resistance to P. teres f. teres in barley on the chromosome 4H. Recently, 449 barley accessions were phenotyped for P. teres f. teres resistance in greenhouse trials. Using genome-wide association, the results identified 254 marker-trait associations corresponding to 15 QTLs. Four of these regions were new QTL not described in previous studies and are located on chromosome 3H at 233–350 Mpb, 5H at 579 Mbp, 6H at 406–410 Mpb and 7H at 5 Mbp, respectively (Novakazi et al., 2019).

Initially, the genetics conferring resistance to *P. teres f. maculata* contained three major designated loci and therefore has been considered less complex to compare the *P. teres f. teres* – barley interaction (Clare et al., 2020). Designated as Rpt4, Rpt6, and Rpt8, these three major loci confer in barley a resistance to *P. teres f. maculata*. The Rpt4, Rpt6, and Rpt8 loci are localized on chromosome 7H, 5H, and 4H, respectively. Burlakoti et al. (2017) revealed the effect of two- and six-row barley, and concluded that the two-row barley (13%) resistant to *P. teres f. maculata* was less than the six-row barley (43%) tested.

VIII. CONCLUSION

Net blotch of barley caused by the fungal pathogen *Pyrenophora teres* is a major foliar disease in major barley-growing regions throughout the world. It causes significant grain yield loss and reduces grain quality. Net blotch develops quickly when the environmental conditions are optimal including long periods of wet and cultural practice used.. It is the most

important constraint that limits productivity of barley and results in constantly low yield of barley in Ethiopia. The net blotch control provides a significant challenge now and in the future.

There are several methods to reduce yield losses due to barley net blotch disease. For example, crop rotation, fungicide application, and the deployment of resistant cultivars can be used to manage net blotch of barley. However, integrated disease management is one of the most important strategies that should be followed to reduce the effect of plant diseases in crops. A promising approach to achieve this aim while minimising use of pesticides is to apply and combine different agriculture practices that contribute to increasing crop yield by decreasing plant diseases directly or indirectly. For instance, combining good crop hygiene practices, the use of resistant cultivars and chemical control (both as seed dressing and foliar applications), is currently the most effective net blotch disease management strategy.

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