Biology, Epidomiology, Ecology and Managment of stripe rust (*Puccinia striiformis f.sp. tritici*) disease of Wheat

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Abstract

Stripe rust disease caused by Puccinia striiformis f.sp. tritici (Pst) is one of the biotic factors that constrained wheat production. The aim of this paper is to review life cycle, dispersal mechanism and management of stripe rust of wheat. The symptoms appear as very small chlorotic islands on infected leaves. A single uredospore is not visible with the naked eye but in masses appear yellow. Uredia are produced in stripes on the wheat leaf surface. Spores are produced in these pustules until the leaf senescence, and the teliospores form around the edges of the pustules. Urediospores of P. striiformis are dispersed by air over long distances among regions or even between continents. Rain and irrigated conditions create high moisture in the soil and in the air, thus more dew formation occurs at night, which ultimately favors disease development. Stripe rust disease development is mainly dependent on three environmental factors: moisture, temperature and wind. Moisture is responsible for germination, infection and survival of rust spores. Urediospores require moisture (dew) on the host surface for germination and infection. High relative humidity facilitates contact between the urediospores and the plant surface and also increases disease severity by triggering more urediospores to germinate. Temperature governs spore germination, the infection process, the latent period, sporulation, spore survival and host resistance. Cool weather favours stripe rust infection and disease development. Management of stripe rust through resistance is economical and environmentally safe but combination of cultural control practices with disease resistance and fungicide applications are the most effective means for wheat rust control.

Introduction

Stripe rust (P. striiformis f.sp. tritici. Westend) is a common wheat disease in temperate regions as well as in the cool highlands of the tropics and subtropics (Stubbs, 1988). Historically, stripe rust has caused and still causing significant and severe losses on susceptible wheat cultivars worldwide (Wellings, 2011). Infection can occur anytime from the one-leaf stage to plant maturity provided plants are still green (Chen, 2005). The cereal rust fungus P. striiformis belongs to the family Pucciniaceae, order Pucciniales (formerly Uredinales), class Basidiomycetes and the Phylum Basidiomycota (Hibbett et al., 2007).

Early studies by Hassebrauk and Schroeder (1965) suggested the Transcaucasia to be the center of origin of P. striiformis, where wild grasses harbor stripe rust and the pathogen spreads from there in all directions (Hovmøller et al., 2011). Ali et al. (2010) considers Asia, and China in particularly, to be a center of origin of P. sriiformis, based on prolific teliospore production by recombinant populations compared to clonal populations. Stripe rust has been reported from about 60 countries across continents except in Antarctica (Chen, 2005). The areas in the world prone to serious damage by this pathogen are the Arabian Peninsula (Yemen), Australia, East Africa (Ethiopia, Kenya), East Asia (China North-West and South-West), New Zealand, South Asia (India, Pakistan and Nepal) Western Europe (East England) and the USA (Pacific Northwest in particular) (Wellings, 2011).

Stripe rust can cause up to 70% yield loss, although loss varies depending on host resistance, time of initial infection, rate of disease development and disease duration. Stripe rust affects yield and quality of the produce. Seed obtained from stripe rust infected field has reduced vigor and poor germination. Total yield loss is common in areas where the disease starts early in the season and continues to develop for several months.

Pandemics of wheat stripe rust occurred in the 1970s in North Africa, the Indian subcontinent, the Middle East, the East African highlands and China due to the breakdown of the Yr2 resistance gene which was present in most of the cultivars (McIntosh, 2009). Cultivation of susceptible cultivars and favorable weather resulted in the first stripe rust epidemics in South Africa in 1996 (Pretorius, 1996). Epidemics were observed in South Africa in 1997 and 1998 and losses of nearly US$ 2.25 million were estimated in the eastern Free State of South Africa (Pretorius, 2004). In 2002, stripe rust epidemics occurred in China on 66 million ha of wheat that resulted in a yield loss of 13 M tonnes (Wan et al., 2004). Stripe rust caused approximately 20-40% losses in 1999 and 2000 in central Asia (Morgounov et al., 2004). In Australia, US$ 40 million was spent on crop protection to manage the 2003 epidemic of stripe rust (Wellings and Kandel, 2004). The most damaging stripe rust epidemic noted in the USA in 2000, when the disease appeared in at least 20 states of the USA (Markell and Milus, 2008). This epidemic of stripe rust resulted in the loss of more than 0.32 million tonnes of wheat from eight states of the USA (Chen et al., 2010). Stripe rust was reported to cause losses in wheat yield and a reduction in grain quality in soft white spring wheat in Alberta during the 1980s (Conner et al., 1988). Stripe rust epidemics in the past few years in Canada indicated that the disease could cause significant losses in the Canadian prairies (Kumar et al., 2012).

Stripe rust incidence in Ethiopia was first reported in the early 1940's and since the early 1980's. It is becoming more devastating and wide spread following the expansion of semi-dwarf improved wheat cultivars (Hailu et al., 1991). According to Mozgovoy (1987), epidemics of stripe rust occurred in 1977, 1980-83 and 1986, resulting in yield losses of 30-40%. Yield losses of up to 58% were reported due to the 1988 stripe rust outbreaks in the south eastern highlands of the country (Ayele and Wondimu, 1992). Damage from stripe rust depends on the susceptibility of the wheat variety, the onset of epidemics, the amount of stripe rust that develops and prevailing of low temperature during grain filling. Yield losses can be severe (60%) due to shriveling grain and damaged tillers and in extreme situations, it can incur 100% losses especially when the spikes re-infected at higher altitudes (Ayele et al., 2008a). In 2010, Ethiopia experienced severe stripe rust epidemics that affected more than 600,000 ha of wheat and led to an expenditure of more than US$ 3.2 million on fungicides, while significant, widespread losses were still realized (Abeyo et al., 2014).

Wheat stripe rust signs

P. striiformis attacks green portions of cereals (wheat, barley, triticale and rye) and grass plants. Plants may become infected at any growth stage, from crop emergence to maturity (the leaf senescence). Under favorable environmental conditions, the pathogen takes one week to cause visible symptoms and about two weeks for sporulation after pathogen penetration of the host. On adult plants, the fungus produces yellow of pustules consisting of golden yellow to orange colored uredinia on leaves along veins (Chen, 2005). On seedlings, the yellow are not restricted by leaf veins and uredia can cover the whole leaf area (Line, 2002). Unlike leaf and stem rusts, stripe rust spreads consistently beyond the initial infection point (Roelfs et al., 1992). Each uredium may contain thousands of uredospores. The pathogen reduces plant vigor because it removes plant nutrients, water and results in desiccation of leaves (Chen, 2005). Later in the season black teliospores are formed on wheat leaves (Knott, 1989). Germinating teliospores produce basidiospores, which are able to infect the alternate hosts of P. striiformis (Zhao et al., 2013).

Wheat stripe rust life cycles and dispersal mechanisms

The complete life cycle of P. striiformis was not known until 2010, when an alternate host was identified (Jin et al., 2010), thus contributing to a clear understanding of its phylogenetic relationship with other cereal rusts. Aeciospores of P. striiformis were observed on Berberis chinensis, B. koreana and on 'Emerald carousel' (an interspecific hybrid of B. koreana and B. thunbergii) in 2009; however, P. graminis, the rust pathogen of Berberis in North America was unable to infect these Berberis species. Genetic analysis concluded P. striiformis f.sp. poae to be the cause of these aecial infections. This was a starting point to further investigate the life cycle of P. striiformis. To prove Berberis spp. were the alternate hosts of P. striiformis, they inoculated with germinating teliospores from infected wheat leaves. The aeciospores produced on Berberis spp.were inoculated on wheat to prove Berberis spp. as alternate hosts of P. striiformis (where sexual reproduction occurs). The life cycle of P. striiformis consists of pycniospores (on Berberis) - aeciospores (on Berberis) - urediospores (on wheat)-teliospores (on wheat). After discovery of the alternate hosts of P. striiformis, several other Berberis species were identified as alternate hosts of P. striiformis in China (Zhao et al., 2013).

Teliospores germinate at approximately 12^0C in free water and produce a promycelia of four cells. Meiosis then produces a single haploid nucleus that later forms the basidiospore. Germinating basidiospores infect barberry and produces pycnia containing pycniospores on the upper leaf surface (Chen et al., 2014). Pycniospores are of two mating type hyphae (+ and -) and after hybridization produce aecia and aeciospores on the lower leaf surface. Aeciospores infect wheat to produce urediospores. Uredia are produced in stripes on the wheat leaf surface. Spores are produced in these pustules until the leaf senescence, and the teliospores form around the edges of the pustules. Basidiospores are produced from germinating dikaryotic teliospores after the crop season, which are able to infect Berberis spp. and thus produce basidiospores. Basidiospores produce pycnia on upper surface and aecia on lower surface of the barberry leaf.

Upon germination of rust urediospores, germ tubes develop that penetrate the stoma, where sub-stomatal vesicles are formed (Moldenhauer et al., 2006). Haustorial mother cells are formed from 2-3 infection hyphae that arise from the substomatal vesicle. Haustoria are formed in plant cells by invaginating the plasma membran (Ma and Shang, 2009); the haustoria nourish the fungus by drawing water and nutrients from the host plant (Voegele et al., 2009). Most of haustoria (~85%) is located in leaf mesophyll cells, but part (~15%) is present in the leaf epidermal layer (Hovmøller et al., 2011).

Urediospores of P. striiformis are dispersed by air over long distances among regions or even between continents (Hovmøller et al., 2011). Urediospore dispersal may also account for the expansion of the infected area in the same region (Hovmøller et al., 2011). The eastern pathway for dispersal of P. striiformis is from the Transcaucasia to mountain ranges of China and then to Japan (Stubbs, 1985). From Japan, P. striiformis most likely entered the US via the Aleutian's islands and Alaska. From there it spread south to Chile, from the western mountains of the US and to Argentina via Andean-Patogonian valleys. The northern spread of P. striiformis is from the Transcaucasia to Mongolia. The pathogen spread southward along the western mountain ranges to South America from the center of origin (Stubbs, 1985). Yellow rust moved into Europe from the Near East, and from Europe it was introduced to Australia in 1979 and New Zealand in 1982 (Wellings, 2007). According to Stubbs (1985), the fungus has moved from the center of origin to the south through Saudi Arabia and Yemen along the mountains and reached East Africa. Stripe rust disease development is mainly dependent on three environmental factors: moisture, temperature and wind. Moisture is responsible for germination, infection and survival of rust spores. Urediospores require moisture (dew) on the host surface for germination and infection..

Rain and irrigated conditions create high moisture in the soil and in the air, thus more dew formation occurs at night, which ultimately favors disease development. Rain is also helpful in spore dispersal. Urediospores generally germinate shortly after production, provided enough moisture is present and temperature is optimum. Dry urediospores can survive longer than wet uredio spores, so long distance dispersal of the spores is not an issue. High relative humidity facilitates contact between the urediospores and the plant surface and also increases disease severity by triggering more urediospores to germinate (Rapilly, 1979). Temperature governs spore germination, the infection process, the latent period, sporulation, spore survival and host resistance. Cool weather favours yellow rust infection and disease development. The optimum temperature for the germination and infection process of P. striiformis is $7-10^0C$ (minimum 0 and maximum 23^0C) (Chen, 2005). Spores that are formed between $5-10^0C$ have the highest germination; those formed above 30^0C do not germinate (Line, 2002). Moderate frost has no adverse effect on uredospore germination (Hassebrauk and Schroeder, 1965). The optimum temperature for uredospore germination and appresoria formation is 7 C (maximum 15^0 and minimum 2^0C). Germination of uredospores is always greater on the upper leaf surface than on the lower leaf surface (Stubbs and Plotnikova, 1972). Night-time temperature is more important for stripe rust infection than day-time temperature and light is not required for germination (Stubbs, 1985). Conditions favorable to infection more often occur at night than during the day because favorable temperature and dew occur together. Temperature also affects survival of spores in winter (Chen, 2005). Wind may hamper spore germination and infectivity because it can dry urediospores, although this also increases the duration of spore viability. The major role of wind is to disperse pathogen from infected fields to healthy fields. Wind spreads the spores over long distances (Chen, 2005). It is suggested that primary inoculum originates at a site far from the actual site of infection and wind plays an important role in long-distance dispersal of the urediospores.

Plants showing a resistant reaction at high light intensities could be susceptible at low light intensities (Stubbs, 1985). Ultraviolet (UV) light is more detrimental to uredospores of P. striiformis as compared with spores of P. graminis (Maddison and Manners, 1972). It was also demonstrated that P. striiformis is more sensitive to air pollution than other cereal rusts (Sharp, 1967).

A combination of cultural control practices with disease resistance and fungicide applications are the most effective means for wheat rust control (Roelfs et al., 1982). It is essential to understand the epidemiology of a disease before starting any management strategy, especially one involving cultural or chemical control measures. Without doubt, a combination of cultural practices with disease resistance and perhaps fungicide applications (under unusual circumstances) will be the most effective means of managing the cereal rust diseases. Because of the

airborne nature of the inoculum of cereal rusts, quarantine measures do not prevent entry of the new races of the pathogen. However, one should take care not to unknowingly transport or carry uredospores of the cereal rusts outside their epidemiological areas. Important differences in virulence, aggressiveness and adaptation exist in the different pathogen populations of these fungi worldwide. The principal management of stripe rust has been achieved through growing resistant cultivars (Chen, 2007).

Wheat stripe rust disease management methods

Cultural practices are mostly used to supplement the other disease control methods. Removing green bridge plants with tillage or herbicides reported to be effective control measure for epidemics resulting from endogenous inoculum (Roelfs et al., 1982). However, cultural practices, such as late planting, reduced irrigation, avoidance of excessive nitrogen use, and elimination of volunteer and grass plants, when used alone, may not be profitable (Chen, 2007).

Resistant varieties can be the simplest, practical, effective and economical method of plant disease control. The use of resistant varieties cannot only ensure protection against diseases but also save the time, energy and money spent on other measures of control. Management of stripe rust through resistance is economical and environmentally safe. Two types of resistance have been identified in several cereal-rust patho-systems: hypersensitive or qualitative (race-specific) and quantitative (race-non-specific) resistance. The average lifetime of the genes conferring race- specific resistance is estimated to be five years on global basis (Kilpatrick, 1975). The race specific resistance is also referred to as major gene resistance and is qualitative in nature. It functions against certain rust races or biotypes but not against others (Babiker et al., 2009). It has simple inheritance and exhibits discrete segregation pattern following simple Mendelian genetic ratios. The race specific genes are characterized by dominant or recessive patterns of inheritance. Most of these resistance genes result into hypersensitive responses; the rapid death of the infected cells which aims to restrict the spread of the pathogen to other parts of the plant (Lowe et al., 2011). The rust fungi are host-specific with either compatible or incompatible associations with their host plants in a gene-for-gene manner (Flor, 1955). The gene for gene concept implies that with each host plant resistance gene (R gene); a corresponding gene locus (race specific effectors) is present in the pathogen with alternate alleles conditioning avirulence (Avr) gene and virulence (Flor, 1971).

The rust fungi produce elicitor (effector) molecules detected by receptor molecules in wheat. The effectors contain many chemical compounds like oligosaccharides, lipids, peptides and proteins. Race specific effectors are produced only when specific avirulence genes are present in a particular pathotype of the pathogen (Flor, 1971). When the plant's receptors detect the pathogen's elicitors, a host defence mechanism is stimulated. This is followed by the death of the infected cells and the pathogen growth is hindered. Any breakdown in resistance leads to the absence of the defence mechanism (McDonald and Linde, 2002). This implies that changes in the elicitor leads to the non recognition by the receptors of host plant thus increasing the frequency of the pathogenic races which eventually cause rust infection and reproduction; acompatible host-pathogen response (Crute and Pink, 1996). In nature, pathogens with high evolutionary ability overcome host resistance. When a host resistance gene with a large effect is spread over a vast area (boom), the pathogen adapts by evolving into a new population which overcomes the subsequent resistance (bust) in the host plant rendering some rust resistance genes ineffective (McDonald and Linde, 2002).

The regional deployment of genes, multiline cultivars, mixtures of cultivars have been vital in ensuring effective resistance (Hogenboom, 1993). Gene deployment involving cultivars with complementary sets of resistance genes in combinations has led to durable disease resistance (Young, 1996). This has also ensured yield stability while compensating for disease resistance among the different cultivars. Thus, to restrict pathogen spread, separate maintenance of resistance would help to generate negative gametic disequilibria in pathogen population (Wolfe, 1993). The race-specific resistance genes could be pyramided in new wheat cultivars to develop stable sources of resistance. It is also referred to as incomplete, horizontal, field, adult plant, polygenic or minor gene resistance (Watson, 1970). The race non specific resistance is conditioned by several genes each having small effects on the phenotype. In addition, the confounding effects of environment and /or segregation of several loci lead to the continuous variation exhibited in race non specific resistance (Prashant, 2007). The resistance also works against all biotypes greatly reducing the probability of mutation through asexual reproduction since the specific resistance delays the start of an epidemic while the non specific resistance genes retard epidemic progress (Roelfs et al., 1988). This is because the pathogen would require multiple mutations to acquire virulence against all resistance genes involved in conditioning the resistance thus, the utilization of this types of resistance contributes to durable resistance. It is characterized by slow rusting or partial resistance and is associated with adult plant resistance as opposed to seedling resistance or hypersensitive reaction (Singh et al., 2000). The partial resistance is a form of incomplete resistance where the individuals show lesions indicating susceptible infection types (Parlevliet, 1988). It is conditioned by minor genes with small effects and is difficult to detect individually due to the presence of a functionally diversified and heterogeneous class of genes (Lowe et al., 2011). With partial resistance, the host shows susceptibility but the infection frequency and rate of spore production are greatly reduced and latency period increases. The germplasm with the slow rusting resistance form a thick mycelium and haustoria in parenchyma tissue, which later develop thick walled sclerenchyma tissue. These tissues eventually hinder fungal spread and pustule eruption preventing epiphytotics (Singh et al., 2007). This affects the rate of leaf penetration, causing fewer lesions per unit area, smaller lesions, fewer spores per lesion, restriction on rate of growth and sporulation over shorter periods (Naz et al., 2008). Thus, the existing pathogen populations are greatly stabilized prolonging the resistance. In determining the race non specific resistance, the epidemic expressed would be a measure of amount of disease and the rate of disease increase (Van der Plank, 1963). This goes along with effective disease management where the germplasm being tested for durability are grown in many locations under high disease pressure involving many pathogen races (Johnson, 1984). Through such a criterion, the disease progression would be attributed to the cumulative effect of various minor genes in a genotype with additive effects.

Gene pyramiding incorporates many desirable genes into elite genotypes (Ayliffe et al., 2008). The pyramids used have involved major genes, minor genes, effective genes, race or race non specific genes which confer resistance (Pedersen and Leath, 1988). The accumulation of minor genes of additive nature is usually followed by intercrosses and recurrent selection (Singh, 1992). There has been increased preference of multiple adult plant resistance genes as opposed to the R genes in gene pyramiding. This is because it's difficult for new races of the Puccinia sp to overcome the multiple resistance genes since this will require multiple mutations in the pathogen genes (Ayliffe et al., 2008).

Use of fungicides in cereal disease management is not generally emphasized and consequently the research on this aspect of disease management was much limited. Since diseases such as stripe and stem rusts of wheat could not be controlled through host resistance alone. Fungicide could be applied in order to provide options; but it should safely and economically be used for the control of major diseases. The use of fungicides should, however, be carefully thought of since their effectiveness and economical uses usually depend on the level of resistance of the variety used, the growth stage of the crop when the disease begins, disease severity and inoculums build up, and prevailing weather conditions, since frequencies and rate of applications usually depend on these factors. Environmental considerations should always be taken whenever one decides on the use

of chemicals for the control of diseases. Timing of fungicide application is important for effective and economical control of stripe rust as the start and duration of disease development can vary a lot due to the great variations in the weather conditions.

To combat wheat rust diseases effectively, integrated management approaches are essential due to the complex nature of the pathogen. For long term prevention, single control methods are not adequate but rather integrated strategies and practices are needed. Using resistant varieties is the primary tool for disease prevention, but contingency plans should also be developed to control possible outbreaks with proper use of fungicides. Other agronomic measures such as removing green bridges between crop cycles, variety deployment, appropriate irrigation and fertilization should also be considered.

Summary and Conclusions

Stripe rust is the most destructive especially when the spikes are infected in the extreme highlands. New races emerge through mutation, migration and recombination of existing virulence genes. Stripe rust affects yield and quality of the produce. Seed obtained from stripe rust infected field has reduced vigor and poor germination. Stripe rust disease development is mainly dependent on three environmental factors: moisture, temperature and wind. Night-time temperature is more important for yellow rust infection than day-time temperature and light is not required for germination. A combination of cultural control practices with disease resistance and fungicide applications are the most effective means for wheat rust control but use of resistant cultivars is the most effective, economical and environmentally safe control measure, especially for the resource poor farmers.

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