Novelty Journals

ISSN 2394-966X

International Journal of Novel Research in Life Sciences
Vol. 7, Issue 2, pp: (35-69), Month: March - April 2020, Available at: www.noveltyjournals.com

Wheat Yellow Rust Disease Management: A Review

WorkuAbebe

Ethiopian Institute of Agricultural Research, Holleta Agricultural Research Center (HARC), P.O. Box 2003, Holeta, Ethiopia

E-mail: workuabebe2005@gmail.comTel: +251913317619

Abstract: **Wheat is a widely planted crop throughout the world, with an estimated 734.8 million metric tons annual global production. Wheat plays an important role in everyday life of the world's population and provides over 21% of the food calories and 20% of the protein to more than 4.5 billion people, thereby playing a fundamental role in food. Wheat was one of the miracle crops of the 20th Century playing a significant role in the Green Revolution led by Norman Borlaug, which dramatically reduced poverty, hunger and saved millions of lives worldwide. However, it is threatened by several diseases, of which yellow rust is one of the most important wheat disease that can cause up to 100% yield loss. In Ethiopia, reports indicate that yield losses caused by stripe rust have ranged from 40 to 100% depending on the degree of susceptibility of cultivars, the time of the initial infection and environmental conditions during epidemic development. Therefore, it should be managed using integrated disease management methods. Thus, wheat producers should thoroughly know the elements of integrated wheat yellow rust disease management. This review discusses recent information on economic importance of wheat yellow rust disease, epidemiology of yellow rust disease, life cycle of yellow rust disease pathogen, host range of yellow rust disease pathogen and different elements of wheat yellow rust disease management. Under wheat yellow rust disease management, elements of wheat yellow rust disease management methods such as cultural, chemical, and use of host resistance methods have been discussed. Under the use of host resistance method, information on the types of resistance and sources of resistance have also been discussed.**

Keywords: **Wheat, yellow rust, disease, management, cultural method, chemical method, host resistance, types of resistance, sources of resistance.**

1. INTRODUCTION

Wheat is one of the most important and significant cereal staple food crops in the world, both in terms of food production and for providing the total amount of food calories and protein in the human diet (Gupta *et al.*, 2008). Moreover, it is the main source of caloric intake in most developing countries, and thereby an important source in order to maintain food security for the growing populations in those countries. Wheat is a widely planted crop throughout the world, with an estimated 734.8 million metric tons annual global production (Daniel, 2016). Wheat plays an important role in everyday life of the world's population and provides over 21% of the food calories and 20% of the protein to more than 4.5 billion people, thereby playing a fundamental role in food security (Braun *et al.*, 2010). Wheat was one of the miracle crops of the 20th Century playing a significant role in the Green Revolution led by Norman Borlaug, which dramatically reduced poverty, hunger and saved millions of lives worldwide (CIMMYT and ICARDA, 2011).

In Ethiopia, wheat is among the most important staple food crops, cultivated in a wide range of agro-ecologies and produced at 1.69 million ha of land with an annual yield approximated to 4.64 million metric ton (Alemu *et al.*, 2019). Bread wheat and durum wheat are the two species of wheat cultivated in Ethiopia and mainly smallholder farmers cultivate it. However, various wheat diseases caused by different pathogens (fungal, bacterial, and viral pathogens)

seriously challenge wheat production. Of these, the rust pathogens are among the most important pathogens causing a continuous threat to wheat production (Singh *et al.*, 2008; Dean *et al.*, 2012) and the three rusts of wheat have been reported to cause a vast amount of losses in different areas, years and environments favoring disease epidemics (Dean *et al.*, 2012; Pardey *et al.*, 2013; Beddow *et al.*, 2015; Singh *et al.*, 2016). Yellow rust, of the three rust, has been reported as an increasing problem (Singh *et al.*, 2000; Hovmøller *et al.*, 2010) with repeated cases of worldwide invasions (Wellings, 2007; Ali *et al.*, 2014a; Hovmøller *et al.*, 2016; Walter *et al.*, 2016) possibly due to a combination of long distance migration capacity (Zadoks, 1961; Brown and Hovmøller, 2002), high rates of mutation from avirulence to virulence (Hovmøller and Justesen, 2007b), adaptation to different climatic conditions (Markell and Milus, 2008; Milus *et al.*, 2009), existence of recombinant and highly diverse populations (Ali *et al.*, 2014a; Thach *et al.*, 2016a) and the potential creation of new variants through a sexual cycle (Jin *et al.*, 2010; Rodriguez-Algaba *et al.*, 2014). Yellow rust is among the oldest known diseases and is important worldwide (Singh *et al.*, 2005). The first reports of stripe rust and its distribution around the world were given by Hassebrauk (1965), Stubbs (1985), Line (2002), and Li and Zeng (2003). Yellow rust of wheat has been reported in more than 60 countries and on all continents except Antarctica. Moreover, it is globally the most damaging diseases in many cool and temperate regions of the world (Stubbs 1985, 1988; Wellings and McIntosh 1990; Wellings *et al.*, 2003; Roelfs *et al.*, 1992; McIntosh and Brown 1997; Boshoff *et al.*, 2002; Line 2002; Yahyaoui *et al.*, 2002) and is considered to be the most economically important disease of wheat production worldwide (Hovmøller *et al.*, 2011). Historically, yellow rust has caused and is presently causing significant and severe losses on susceptible wheat cultivars worldwide (Wellings, 2011). It is widespread globally and causes significant yield losses every year (Chen 2005). Destructive yellow rust epidemics in wheat have often proven difficult to control even with fungicide application, and in recent epidemics in Central Asia, West Asia and Africa up to 80% yield losses have been reported. One reason is that the pathogen has overcome the major resistance genes in widely cultivated wheat cultivars (Wellings 2011; Solh *et al.*, 2012; Beddow *et al.*, 2015; Jighly *et al.*, 2015; Boyd 2005; Kolmer 2005; Hodson 2011). With the emergence of new yellow rust races, several important resistance genes, such as Yr2, Yr6, Yr7, Yr8, Yr9, Yr17 and Yr27, are no longer effective (Zegeye *et al.*, 2014; Jighly *et al.*, 2015; Maccaferri *et al.*, 2015). Therefore, there is a need to utilize different genetic stocks in order to obtain an effective disease management strategy and to broaden the genetic base of yellow rust resistance in wheat. Since yellow rust is a very important disease of wheat, particularly in Central and West Asia and North Africa, it is thought to have caused recurrent, severe damage in crops since the dawn of agriculture in these areas (Wellings 2011; ICARDA, 2011). The historical view is that yellow rust is principally a disease of wheat grown in cooler climates (2°C - 15°C), and generally associated with higher elevations, northern latitudes or cooler years. But recent outbreaks have defied this assumption with current strains of the disease more adapted to high temperatures, and hence countries closer to the equator (Yahyaoui *et al.*, 2004; Milus *et al.* 2009; ICARDA 2011). Moreover, because the disease attacks from early in the growing season, plants are often stunted and weakened and the crop losses can be severe (50 - 100%), due to damaged plants and shriveled grain. Epidemics of yellow rust continue to cause severe losses and have been reported in Afghanistan, Azerbaijan, Ethiopia, Georgia, Kenya, Kyrgyzstan, Morocco, Iran, Iraq, India, Pakistan, Syria, Tajikistan Turkey, Turkmenistan, and Uzbekistan in recent years (ICARDA, 2011). Yellow rust epidemics have also occurred in the Australia, Europe, China and the United States. In 2009-10, the outbreak of Yr27 an aggressive new strain of yellow rust caused significant yield losses in Azerbaijan, Ethiopia, Iran, Iraq, Kenya, Morocco, Syria, Turkey, Uzbekistan, and threatening the food security and livelihood of resource-poor farmers and their communities.

In the highlands of Ethiopia, at altitudes ranging from 2150 to 2850 meters above sea level, yellow rust is an important disease of wheat production (Bekele *et al.*, 2002; Dereje and Chemeda F. 2006; Wubishet *et al.*, 2015; Tilahun, 2018; Alemu *et al.*, 2019). Frequent and widespread yellow rust epidemics have been observed at higher elevations in the southeastern part of wheat growing areas of the country (Badebo *et al.*, 1990; Huluka *et al.*, 1991; Alemu *et al.*, 2019). This frequent and widespread epidemic was attributed to varietal susceptibility, production of mega cultivars, and expansion of wheat mono-cropping, introduction of new virulent races and favorable environmental conditions for disease development (Dereje, 2003; Wubshet *et al.*, 2015; Alemu *et al.*, 2019). Reports indicate that yellow rust, during epidemics, can cause a huge damage to wheat crops (Dean *et al.*, 2012; Alemu *et al.*, 2019). It can cause 100% yield loss if infection occurs very early and the disease continues to develop during the growing season on susceptible cultivars (Chen, 2005; Afzal *et al.*, 2007; Wubshet *et al.*, 2015).

In Ethiopia, reports indicate that yield losses caused by stripe rust have ranged from 40 to 100% depending on the degree of susceptibility of cultivars, the time of the initial infection and environmental conditions during epidemic development

(Badebo *et al*., 2001) and at high altitude, when susceptible cultivars are grown and the environmental condition is conducive, yellow rust can cause yield loss up to 100% (Badebo *et al.*, 2008; Alemu *et al.*, 2019). In 2010, Ethiopia experienced one of the most serious yellow rust epidemics in recent times, with more than 600,000 ha of wheat affected and an estimated $US3.2 million spent on fungicides (Abeyo *et al.*, 2014). In Ethiopia, repeated epidemics of yellow rust were recorded in the last three decades. The first yellow rust epidemics occurred in 1977 on wheat variety 'Laketch' in state farms of Arsi and Bale (Hulluka *et al.*, 1991; Tilahun, 2018; Alemu *et al.*, 2019). In 1988, another yellow rust epidemic occurred on the wheat variety; 'Dashen' which carried Yr9 gene in Arsi and Bale zones (Zewde *et al.*, 1990; Badebo *et al.*, 1990; Tilahun, 2018; Alemu *et al.*, 2019). In 2010, a devastating yellow rust epidemic affected widely grown 'Kubsa' and 'Galema' bread wheat varieties and the Yr27-virulent strain of *P. striiformis f.sp. tritici* was attributed to be a major cause of this epidemic (Worku, 2014; Tilahun, 2018). Recently, another new race was detected in Ethiopia in 2016, after being first detected in Afghanistan in 2012 and 2013 on resistance gene 'PstS11'. This race caused epidemics in Ethiopia in 2016 (Hovmoller *et al*., 2017; Tilahun, 2018).

2. ECONOMIC IMPORTANCE OF WHEAT YELLOW RUST DISEASE

Yellow rust of wheat is the most important rust disease of wheat worldwide and because the disease attacks from early in the growing season, plants are usually stunted and weakened, causing severe yield losses and on susceptible varieties, disease development at the seedling stage can cause total yield loss (Chen, 2005). The disease reduces yield, quality and size of the harvested grains (Alemu *et al.*, 2019). It is a widespread disease across major wheat growing regions with diverse cropping systems, growing seasons and germplasm characteristics (Stubbs, 1985; Manners, 1988; Singh *et al.*, 2004; Wellings, 2011). Resulting losses have been estimated to be at least 5.5 million tons per year at worldwide level (Beddow *et al.*, 2015), *i.e.*, it results in significant yield losses to wheat production worldwide (Kolmer 2005; Hovmøller *et al*., 2011; Szabo *et al*. 2014). Moreover, Yellow rust can cause up to 100% yield losses in susceptible wheat cultivars (Chen 2005; Huerta-Espino *et al*., 2011; Singh *et al.*, 2015). However, the magnitude of yield loss is influenced by resistance level, time of initial infection, rate of disease development and disease duration (Luig, 1985; Badebo *et al*., 2001; Alemu *et al.*, 2019). Yellow rust can cause up to 100% yield losses in susceptible wheat cultivars when favorable environmental conditions are present (Chen 2005; Huerta-Espino *et al*. 2011; Singh *et al*. 2015). Several epidemics and outbreaks of yellow rust significantly threatened the food security and livelihoods of poor farmers in many wheat-growing regions of the world (Wellings 2011; Solh *et al*. 2012). Moreover, the emergence and spread of novel yellow rust races have led to the breakdown of most of the widely deployed resistance genes used in wheat production (Huerta-Espino *et al.*, 2011; Wellings 2011; Singh *et al.*, 2015). Reports indicate that there was a yield loss of up to 75% in wheat in USA (Doling and Doodson 1968; Roelfs 1978). In Addition to USA, a large epidemics of wheat yellow rust occurred in other regions in the world including North Africa and the Middle East in the 1970s (Saari *et al.*, 1985). These epidemics occurred because of the presence of susceptible gene (Yr2 gene) in most of the cultivars at that time (McIntosh *et al.*, 2009). Losses of nearly 2.25 million US $ were estimated in the 1998 in South Africa (Pretorius, 2004). In China, a widespread yellow rust epidemic affected about 6.6 million hectares of wheat in 11 provinces during the growing season of 2001-2002, causing a yield loss of 13 M tones (Wan *et al.*, 2004). Substantial losses were reported between 1999 and 2000 in central Asia with a yield losses ranged from 20 to 40% (Morgounov *et al.*, 2004). Similarly, during the last decades, several yellow rust epidemics in most of the wheat-growing areas of Iran have caused over 30% crop loss and estimated grain losses were 1.5 million tons and 1.0 million ton in 1993 and 1995, respectively (Torabi *et al.*, 1995). In Australia, fungicide costs was estimated to 40 million AU$ in 2003 (Wellings *et al*., 2004). The most severe yield losses recorded in the USA were estimated in more than 9 M bushels of wheat in 2000, when the disease appeared in at least 20 states (Markell *et al.*, 2008).

Historically, the epidemics of yellow rust have occurred in moderate regions with cool and wet spring and summer (Stubbs, 1985; Zadoks and Vandenbosch, 1995) and have been significant in some locations, causing huge yield losses which require serious financial investment in order to manage the crops from loss (Wellings, 2007). The major epidemics were caused due to development or mutations of new races of *Puccinia striiformis f.sp. tritici* and then the breakdown of resistant genes within in a short period of time (Chen, 2005; Wellings, 2011; Hovmøller *et al*., 2011).

The most and recent destructive epidemics have taken place in China, Northern and Eastern Africa, Western Asia, Central Asia and Middle East, and the epidemics may become even more aggressive with races that can tolerate and develop in higher temperatures (Hovmøller *et al.*, 2010; 2011). According to Milus *et al.* (2009), the new races of yellow rust have

significantly increased adaptation to warmer temperatures and therefore continue to cause disease epidemics. Over the last decade, a series of regional outbreaks of yellow rust epidemics have been reported worldwide, including Central and West Asia and East and North Africa (Sanders, 2018). The 2009-2010 epidemic severely affected many countries including Morocco, Turkey, Algeria, Syria, Lebanon, Iraq and Uzbekistan (Ezzahiri *et al.*, 2010; Rahmatov *et al.*, 2012) of which Syria and Lebanon were the worst hit of this epidemic; Syria lost nearly half of its wheat harvest (El Amil, 2015). Since 2010, yellow rust was widely spread in East Africa causing economic losses in low-input farming system (Singh *et al*., 2016). Widespread epidemics were observed in Tajikistan in 2010 and later on in Uzbekistan and other countries of Central Asia (Rahmatov *et al.*, 2012). These regular epidemics caused not only economic losses and additional need for fungicide sprays, but also threatened seed availability for the next cropping season (Shean, 2010). In Europe, the established *P. striiformis* population has largely been replaced since 2011 by distinct new lineages, generally known as Warrior, causing increased epidemics on multiple wheat varieties (Rahmatov, 2016), and another lineage associated with epidemics on triticale in 2009–2010, particularly in Scandinavia (Hovmøller *et al.*, 2011). In 2014, the Central Research Institute for Field Crops in Ankara and the Regional Cereal Rust Research Center in Izmir confirmed the detection of a new *Puccinia striformis f.sp. tritici* race in Turkey. The newly detected strain was "Warrior" race previously identified in the United Kingdom in 2011. Some of Turkish commercial cultivars known to be resistant to the previously characterized races of *Puccinia striformis f.sp. tritici* were recorded as fully susceptible to this new race. The warrior race was much more widespread in the following year after its first detection. It was already present in high frequencies in most European countries and North Africa (Mert *et al.*, 2016) and it was confirmed in Morocco in 2013 and in Algeria in 2014 (RustTracker, 2016). This race was very dissimilar to pre-2011 European races. It showed relatively higher genetic diversity than other previous races (Hovmøller *et al*., 2016). Yield losses reported from yellow rust infection are $360 million in USA, in 2004, $100 million in Pakistan in 2005, $AUD127 million in Australia in 2009, $30 million in Morocco in 2009 and above 1 million tonnes in Syria in 2010 (Long, 2005; Duveiler *et al.*, 2007; Murray and Brennan, 2009; Hodson, 2010; FAO, 2010). Five major epidemics of yellow rust have occurred in Central Asia in 1998, 2000, 2005, 2009 and 2010 (Ziyaev *et al.*, 2011). In Tajikistan, the yellow rust is a serious disease with significant yield losses in susceptible cultivars. The severity of the yellow rust in 2010 resulted in damages up to 80-100% and yield losses of 30-50%, in widely grown farmers bread wheat varieties and 70% of the breeding lines showed susceptibility to yellow rust in the field trials (Rahmatov *et al.*, 2011a; 2011b). In addition, new aggressive races of yellow rust were found in Sweden and Denmark damaging triticale and affecting wheat, rye and barley (Jørgensen *et al.*, 2010). However, deployment of host-plant genetic resistance is still seen as the most economically and environmentally safe approach to reduce losses due to rust diseases in wheat (Burdon *et al.*, 2014; Singh *et al.*, 2016). Therefore, cultivation of resistant cultivars is the most effective, economical and environmentally safe control measure of great value for the growers (Line and Chen 1995; Chen, 2005).

In Ethiopia, frequent yellow rust epidemics have occurred in the past owing to the emergence of virulent races that caused susceptibility on popular bread wheat verities like Lakech (Huluka *et al.*, 1991; Tilahun B., 2018; Alemu *et al.*, 2019) and Dashen (Badebo *et al.*, 1990; Tilahun B., 2018; Alemu *et al.*, 2019). Yield losses caused by stripe rust in Ethiopia have ranged from 40 to 100% depending on the degree of susceptibility of cultivars, the time of the initial infection and environmental conditions during epidemic development (Badebo *et al*., 2001; Solh *et al.*, 2012; Tilahun B., 2018; Alemu *et al.*, 2019). In 2010, Ethiopia experienced one of the most serious yellow rust epidemics in recent times, with more than 600,000 ha of wheat affected and an estimated $US3.2 million spent on fungicides (Abeyo *et al.* 2014). During this time, emergency fungicide applications were done and 30% of the wheat area affected was sprayed.

3. EPIDEMIOLOGY OF WHEAT YELLOW RUST DISEASE

3.1 Sources of inoculums

Puccinia striiformis f. sp. tritici belongs to the Pucciniaceae family, order Uredinales, phylum Basidiomycota and class Basidiomycetes (Chen *et al.*, 2014). Until recently, the alternate host of stripe rust was unknown and urediniospores were considered the only source of inoculum. However, in recent studies Berberis spp. have been shown to serve as the alternate host for stripe rust populations (Jin *et al.*, 2010). The teliospores germinate into aerial basidiospores and then infect the alternate Berberis host (Hovmøller *et al.*, 2011; RodriguezAlgaba *et al.*, 2014). It is thought that the centre of origin for the stripe rust pathogen is South-East Asia, the Middle East, East Africa, Transcaucasia, Himalayan, Mediterranean and Central Asia (Ali *et al.*, 2014). However, stripe rust is widespread globally and causes significant yield

losses every year (Chen 2005; Wellings 2011). Destructive stripe rust epidemics in wheat have often proven difficult to control even with fungicide application, and in recent epidemics in Central Asia, West Asia and Africa up to 80% yield losses have been reported. One reason is that the pathogen has overcome the major resistance gene/s in widely cultivated wheat cultivars (Wellings 2011; Solh *et al.*, 2012; Beddow *et al.*, 2015; Jighly *et al.*, 2015). With the emergence of new stripe rust races, several important resistance genes, such as Yr2, Yr6, Yr7, Yr8, Yr9, Yr17 and Yr27, are no longer effective (Zegeye *et al.*, 2014; Jighly *et al.*, 2015; Maccaferri *et al.*, 2015). Therefore, there is a need to utilise different genetic stocks in order to obtain an effective disease management strategy and to broaden the genetic base of stripe rust resistance in wheat.

3.2 Disease development

Wheat stripe rust can develop on triticale, barley, barley grass, brome grass and some other grasses, but wheat is the main host. Wind spreads spores of stripe rust from pustules that develop on infected leaves. If the spores land on another living wheat leaf, they can germinate and infect the leaf. The rust grows inside the leaf and then produces pustules containing new spores. Wind is the main means of spread or dispersal for stripe rust. The spores are produced in huge numbers in pustules on the upper surface of leaves. Once the spores become airborne, their spread is a matter of chance. Most will land on soil or other plants, while some stay airborne until sunlight kills them in a few days. However, they are produced in such high numbers that some land on other living wheat plants. During high humidity in winter, most spores remain in small clumps: these are relatively heavy and fall out of the air quickly, so their spread is mostly over in a very short distances, leading to the 'hot-spots' of infection seen in crops in late winter and early spring. In lower humidity, spores disperse singly in the air and can travel for much longer distances. This may result in a uniform pattern of disease development in crops. Long distance dispersal means that rust developing in any part of the wheat belt can spread rapidly to other areas. Some travel very far: spores produced in Western Australia can reach eastern Australia while those from eastern Australia have reached New Zealand. Spores of stripe and other rusts can also adhere to clothing, so that travellers can inadvertently carry them. It is likely that stripe rust entered Victoria on an air traveller's clothes from Europe in 1979 and Western Australia from North America in 2002. For this reason, air travellers who have visited agricultural enterprises are strongly advised to wash or dry clean clothing immediately on return to Australia to reduce the chance of introduction of rusts and other plant diseases.

Once released from a leaf, spores can live for only a few days. A few land on wheat where they can infect if conditions are suitable. Infection requires high humidity for 4 to 6 hours at 10 to 15°C, with increasing time required at lower and higher temperatures. Infection seldom occurs below about 2°C, and ceases above 23°C. After infection, the pathogen grows within the leaf, deriving its nutrients from living wheat cells. Growth is most rapid at 12 to 15°C, reducing to almost nil at 3°C and above 25°C. If temperatures are outside the range for growth for any part of the day, the rust stops growing for that time but resumes growth when the temperatures become favourable again at other times of the day. Sporulation at 12 to 20°C, the fungal pathogen grows for about 14 days (shorter in some highly susceptible varieties) before the pustules erupt through the leaf, with longer times of up to 80 days at 3°C, and cessation of growth much above 25°C. The time between infection and appearance of symptoms is termed the latent period. High humidity and similar temperatures to the other stages of growth favour spore production. Thus fresh spores are usually seen in the morning because cooler temperature and still air are more conducive for sporulation. Moreover, temperature affects all parts of the disease cycle in a similar way. At ideal temperatures, the cycle from spore infection to new spore production takes about 12-14 days. Providing there are susceptible wheat plants and sufficient humidity, the average daily temperature (average of the maximum and minimum temperatures each day) has the biggest effect on development of stripe rust. Yellow rust can develop on the inside of the glumes, lemma and palea (cup-shaped bracts that enclose the seed). Most of this infection occurs when the florets open at anthesis. Spores enter the open floret, infect and develop within the lemma and palea, and so accumulate, often piling up on the top of the seed as it grows. The seed in the infected spikelet can be smaller, but there seems to be little effect on seeds growing in uninfected spikelets in the rest of the head. Infection of the seed does not occur, and hence there is no seed transmission of stripe rust. Head infection can be common in some moderately susceptible and susceptible wheat. If fungicide spraying is done before head emergence, the heads are not protected: if there are large numbers of spores blowing into the crop, head infection can occur. Spraying after head emergence and before anthesis will contribute to control head infection, but spraying after head infection does not.

As with other diseases, the three factors in the disease triangle are all essential for disease development. However, the development of stripe rust, compared with many other diseases, depends even more on the very specific weather conditions when pathogen inoculum (urediniospores) and susceptible host plants are present. The three most important weather factors affecting epidemics of stripe rust are moisture, temperature, and wind. Moisture affects spore germination, infection, and survival. The uredial infection process begins when an urediospore lands on a susceptible wheat or closely related grass leaf with free moisture and air temperature ranging from 0 to 25 °C for at least 3 hr (Rapilly 1979; Chen *et al.*, 2014; Chen 2005b), although newer races of *Puccinia striiformis f. sp. tritici* have been found to be more aggressive in warmer conditions (Milus, Kristensen, and Hovmøller 2009). The urediniospore produces a germ tube, which develops into a haustorium, which is able to penetrate the stomata of the wheat leaves. Urediniospores of *Puccinia striiformis f. sp. tritici* and *P. graminis* have been shown to be very sensitive to UV radiation and air pollution (Aylor 2003; Chen *et al.*, 2014). Moist regions with frequent dew formation during the growing season provide conditions conducive for stripe rust. Because high moisture promotes infection, irrigated cereal crops are vulnerable to stripe rust. In rain-fed areas, light rains create the ideal conditions for infection. After rains, high moisture in the air and soil often creates dew for several nights, providing favorable conditions for infection. Rain can also disperse spores to spread the disease because raindrops release urediniospores either by direct impact or by splashing (Rapilly 1979). Stripe rust can be predicted by monitoring precipitation in a region and dew formation in the fields. High moisture promotes disease by favoring spore germination, but also adversely affects spore survival. Because urediniospores do not exhibit fungistasis, they can germinate immediately after they are produced if dew is present and the temperature is in the correct range. Since urediniospores kept under high-moisture conditions lose viability more quickly than those kept dry, spores should be kept dry to maintain viability during sample shipment and storage. With regard to epidemics of stripe rust, dry spores survive longer than moist spores, and therefore, are more likely to survive to infect next crops and be disseminated over longer distances. The dry weather in late summer and the various stages of wheat crops in the PNW allow urediniospores produced on late-harvested spring wheat to survive the summer and be able to infect seedlings of winter wheat planted in the fall. This is one of the reasons why stripe rust occurs every year in this region. Moisture also affects spore dispersal. Depending on the relative humidity, urediniospores can spread individually or in clusters (Rapilly 1979). Clusters increase in size as humidity increases. High humidity leads to stronger adhesion of urediniospores to the leaves (Rapilly 1979). Nevertheless, high humidity generally increases disease by increasing infection frequency. Temperature affects spore germination and infection, latent period, sporulation, spore survival, and host resistance. The disease can start very early in the crop season and, therefore, can cause more severe damage in some areas than leaf rust and stem rust which have higher temperature optima for development. Variation exists among isolates of *P. striiformis* for response to temperature. In a recent study, Milus and Seyran (2004) determined the spore germination rate and latent period of 6 isolates collected before 2000 to represent "old races" and of 14 isolates collected after 2000 to represent "new races". They found significant temperature × isolate interactions for latent period and spore germination rate. Eight of the 14 new isolates, and only 1 of the 6 old isolates, germinated significantly faster at 18 °C than at 12 °C. Two of the six old isolates germinated significantly faster at 12 °C than at 18 °C. All new isolates had significantly shorter latent periods at 18 °C than at 12 °C. Of the six old isolates, four had similar latent periods at both temperatures, one had a shorter latent period at 18 °C, and one had a shorter latent period at 12 °C. These results indicate that stripe rust caused by the new isolates tends to develop faster than the old isolates at relatively high temperatures. With regard to the effects of temperature on development of stripe rust, night temperatures play a more critical role than daytime temperatures (Stubbs 1985). Both dew formation and low temperatures occur together most frequently at night, and therefore, infections are more likely to occur at night. Hot weather, especially hot nights, limits disease development and survival of the pathogen. The use of average and maximum daily temperatures to determine if stripe rust occurs in a region or in a season is misleading. For example, if an area has a daily maximum temperature of 28 °C and minimum temperature of 22 °C, while another area has a daily maximum temperature of 34 °C and minimum temperature of 16 °C, then both areas have an average daily temperature of 25 °C. If we use both the daily average and maximum temperatures, we would conclude that the first area is more likely to have stripe rust. In reality, the second area is more likely to have stripe rust because it has a period with temperatures that allow infection, while the lowest temperature in the first area would be marginal for infection to occur. The cool weather at night allows stripe rust to develop and the pathogen to survive. Temperature is the major factor affecting the winter survival of the pathogen of stripe rust. Rapilly (1979) considered that temperatures below –10 °C might halt the pathogen development. Cold weather conditions reduce the pathogen winter survival by winter killing the pathogen in the infected leaves. Therefore, temperature is one of the major weather factors used to predict occurrence of

stripe rust. Wind can be a limiting factor for spreading rust spores over long distances. The timing, type, and direction of winds determine the earliness, scale, and development rate of epidemics of stripe rust. Furthermore, when pathogen inoculum and susceptible host are present, the development of wheat stripe rust disease depends even more on weather conditions such as moisture, temperature and wind (Chen, 2005). Spore germination, infection and survival of *Puccinia striiformis f. sp. tritici*, are directly affected by moisture. A continuous period of three hours of moisture is required for urediniospores germination and infection. A relative humidity near to saturation before inoculation increases rates of spore germination considerably (Line, 2002). Precipitation, especially light rains provide conducive conditions for infection. However, high moisture can also negatively affect spore viability. Spores kept in high moisture conditions lose their viability more quickly than those kept under dry conditions, because they lack the ability to induce fungistasis. Spores dispersal is also affected by moisture. Individual or cluster dispersal of urediniospores depends on the level of relative humidity. Cluster dispersal is limited on high humidity (Chen, 2005). Temperature also influences the germination, infection and survival of spores. Spores are capable of germinating between 2.8-21.7 °C. However, they germinate most easily at 10-12 °C (Line, 2002). Sporulation can occur at 5-20 °C. Latent period, the time elapsing between infection process and sporulation, is estimated to be about 10-15 days at 12- 19 °C. The latent period of stripe rust can last up to 180 days at temperatures near to freezing (Sørensen, 2012). Lower temperatures adversely affect winter survival of the pathogen. Pathogen development could be stopped in temperatures below -10 °C (Chen, 2005). High temperatures above 30 °C limit pathogen development and survival. Infections are more likely to occur at night, where both dew formation and cool temperatures occur together (Sørensen, 2012). Wind inhibits spore germination by decreasing the moisture content of inoculum. Therefore, the infection rate is reduced, while the viability of the inoculum is increased. Wind also facilitates the spread of the inoculum over new territories and controls the time, rate and extent of infection. Long distance dispersion of stripe rust by the air resulted in its reintroduction and widespread (Chen, 2005).

3.3 Disease symptoms

Yellow rust appears as a mass of yellow to orange urediniospores erupting from pustules arranged in long, narrow stripes on leaves (usually between veins), leaf sheaths, glumes and awns on susceptible plants. Pustules arrangement into stripes is an important distinguishing characteristic of this disease. All growth stages of the plant are susceptible to infection (Line, 2002), *i.e.*, Infection can occur anytime from the one-leaf stage to plant maturity provided plants are still green. Symptoms appear about 1 week after infection, and sporulation starts about 2 weeks after infection, under optimum temperature conditions. Its optimum temperature for infection is between 9°C and 11°C and optimum development occurs at temperatures below 23°C (Wiese, 1987; Knott, 1989). Predisposing (environmental) factors are: cool, humid weather conditions with intermittent rains, cooler climates (10-16ºC) and Leaf wetness from rain or dew. Heavy dew or intermittent rains can accelerate the spread of the disease. The fungus forms tiny, yellow- to orange-colored rust pustules, called uredia. Each uredium contains thousands of urediniospores. A single urediniospore is too small to be seen with the naked eye, but spores on mass are yellow- to orange-colored and powdery. Initial symptoms of stripe rust appear about one week after infection as small, yellow spots or flecks on the leaf sheaths. These spots develop into long and narrow stripes on leaf sheaths, glumes and awns and as the disease progresses, tissues around the pustules turn brown and dry resulting in a scorched appearance. Depending on the level of plant resistance and the temperature, various amounts of chlorosis or necrosis appear (hypersensitive response), with or without sporulation. The necrotic stripes or elongated spots that form on leaves of adult plants are distinguishable from spots caused by necrotrophic pathogens, i.e., leaves chlorosis can be quite evident and fields with severe symptoms may be easily detectable from a distance. Mature pustule will break open and release yellow-orange masses of urediniospores. The infected tissues may become brown and dry when plants begin to senesce or become stressed. The pathogen reduces plant vigor because it removes plant nutrients and water, and result in desiccation of leaves, i.e., the pathogen of stripe rust utilizes water and nutrients from the host plants, which weakens the plants. Severe early infection can result in plant stunning (Line, 2002; Chen, 2005; Singh *et al.*, 2017). The basidiospores, produced by germinating teliospores, infect barberry (Berberis spp.) leaves and produce pycnia on the upper surface and acia on the lower surface. Symptoms appear also on Oregon grape (Mahonia aquifolium), another alternate host of *Puccinia striiformis f. sp. tritici*. Similarly, pycnia and aecia are produced on the upper and lower side of leaves respectively (Wang *et al*., 2013).

Resistant wheat cultivars are characterized by various infection types from no visual symptoms to small hypersensitive flecks to uredinia surrounded by chlorosis or necrosis with restricted urediniospore production. On seedlings, uredinia

produced by the infection of a single urediniospore are not confined by leaf veins, but progressively emerge from the infection site in all directions, potentially covering the entire leaf surface. Individual uredinial pustules are oblong, 0.4–0.7 mm in length and 0.1 mm in width. Urediniospores are broadly ellipsoidal to broadly obovoid, (16–)18–30(–32) × (15–)17–27(–28) µm, with a mean of 24.5 × 21.6 µm, yellow to orange in colour, echinulate, and with 6–18 scattered germ pores. Urediniospores can germinate rapidly when free moisture (rain or dew) occurs on leaf surfaces and when the temperatures range is between 7 and 12 °C. At higher temperatures or during the later growing stages of the host, black telia are often produced, which are pulvinate to oblong, 0.2–0.7 mm in length and 0.1 mm in width. The teliospores are predominantly two-celled, dark brown with thick walls, mostly oblong-clavate, (24–) 31–56(–65) × (11–) 14–25(–29) µm in length and width, and rounded or flattened at the apex.

4. LIFE CYCLE OF WHEAT YELLOW RUST PATHOGEN

Considering the economic importance of rust fungi, a solid understanding of their life cycles is quite necessary. Rusts are phenotypically and genetically plastic organisms that exhibit the most complex life cycles shown by all fungi, having as many as five spore types: basidiospores, pycniospores, aeciospores, urediniospores, and teliospores, with considerable variation in spore stage and in some cases a need to alternate between two unrelated hosts (Hart 1988; Cummins and Hiratsuka 2003; Maier *et al.,* 2003). Rust fungi that produce all five-spore forms are termed macrocyclic. Such species can be autoecious, or heteroecious (Petersen 1974). For example *Puccinia striformis f.sp. tritici* & *Puccinia graminis f.sp. tritici* are heteroecious, alternating between cereals as the main host and barberry as the alternate host (Petersen 1974; Singh *et al.* 2008c). Host alternation takes place after the uredinial and telial spore forms. On barberry, *Puccinia graminis f.sp. tritici* & *Puccinia striformis f.sp. tritici* occurs as pycnia and aecia and on cereals, as uredinia and telia (Voegele *et al.,* 2009).

Yellow rust, caused by *Puccinia striiformis f.sp. tritici* fungus, is a macrocyclic and has separate hosts for asexual and sexual phases (Berlin *et al.,* 2017) and therefore, it requires two hosts, primary and alternate to complete its life cycle (Chen *et al.,* 2014). Life cycle of yellow rust consists of both dikaryotic uredial and telial stages, and the teliospores can form haploid basidiospores. Moreover, its life cycle is similar to most rust fungi that have been studied for more than a century, and its alternate host was challenging to identify (Roelfs, 1992; Singh *et al.,* 2002; Jin *et al.,* 2010). In the late 19th century, only telial and uredinial stages of yellow rust were known while the alternate host was searched for among Boraginaceae species (Eriksson and Henning, 1894). However, recently the *Puccinia striiformis* pathogen was identified in barberry species (Jin, Szabo, and Carson 2010), which were therefore suggested as alternate hosts, and sexual recombination was found to play a role in the contribution to the pathogen variability (Jin *et al.,* 2010). *Puccinia striiformis f.sp. tritici* includes five types of spores in the life cycle (Schwessinger, 2017) on two taxonomically unrelated hosts; it alternates between a *graminaceous* host for asexual reproduction and *barberry* for sexual reproduction (Jin *et al.,* 2010). In its primary host, sexual cycle, haploid basidia infect the alternate host, barberry (Berberis spp.), which then develop haploid pycnia. The pycnia produce pycniospores, which disperse and cross-fertilize with receptive hyphae to form the dikaryon, with two unfused nuclei in each cell. The hyphae grow through the barberry, producing the dikaryotic aecia, from which aeciospores are dispersed. Urediniospores and teliospores of the fungus are dikaryotic, whereas teliospores produce haploid basidiospores (Chen, 2005). Pycnial and aecial spore stages of the fungus were recently confirmed (Jin *et al.,* 2010). The diakaryotic phase of the life cycle is confined to the primary host (wheat), upon which urediniospores teliospores and basidiospores are produced. As the nutrient supply from the infected tissues declines, the telia stage is initiated. Teliospores overwinter on residual senesced tissues and germinate the following growing season to produce four haploid basidiospores that infect the alternate host (Berberis spp.) (Sørensen, 2012; Baily, 2013) upon which pycniospores and aeciospores are produced on the upper and lower leaf surface, respectively (Jin *et al.,* 2010). Furthermore, teliospores from wheat leaves infected with *Puccinia striiformis f.sp. tritici* are produced late in the growing season). These abiotic stress tolerant spores are capable of overwintering on infected straw. At the beginning of a growing season, diploid teliospores germinate on the host plant or even dead tissues. The *teliospores* undergo meiosis to produce four or more haploid basidiospores, which are later dispersed into the air (Leonard and Szabo, 2005). The *basidiospores* infect the alternative host through the stomatal cell walls to produce haploid spores, *pycnia* which are of different mating types. Upon the dispersal of the *pycniospores* through rain, dew or insects to other plants, fertilization or plasmogamy occurs leading to formation of a single cell with *aecium* and *aeciospores (*Kolmer *et al.,* 2009). The dikaryotic *aeciospores* are deposited on the host plant from which successful infection and colonization leads to formation of *uredinium* containing *urediospores. Urediniospores* are primarily dikaryotic (n + n) and maintain the dominant asexual

stage of the pathogen population on the primary hosts. This phase is responsible for wide-scale stripe rust epidemics reported on cereal crops. As temperatures rise late in the epidemic phase, *Puccinia striiformis f.sp. tritici* typically produces thick-walled, predominantly two-celled teliospores. Each cell of a mature teliospore contains a diploid (2n) nucleus formed by karyogamy. *Puccinia striiformis f.sp. tritici* isolates vary in their ability to produce telia even under similar environmental conditions (Chen XM *et al.*, 2012). The uredinium can repeatedly infect same host plant epidemics leading to severe. *Puccinia striiformis f.sp. tritici* has been considered until very recently as it reproduce only asexually through dikaryotic uredospores on wheat with the teliospores produced at the end of cropping season unable to encounter an alternate/aecial host to finalize the sexual cycle. However, recently *Puccinia striiformis f.sp. tritici* has been shown to complete its sexual cycle on different species of Berberis (Jin *et al*., 2010). The sexual cycle has been suggested to play important role in offseason survival of many uredinales, while for yellow rust clonal survival has been assumed on volunteer plants and wheat seedlings (Ali *et al*., 2010; de Vallavieille-Pope *et al*., 2012). Aeciospores can only infect the primary host, wheat, which then develop into telia, the site of karyogamy, producing diploid teliospores, from which germinate basidia, the site of meiosis, to complete the cycle.

In the secondary, asexual cycle, the aeciospores germinate into dikaryotic uredinia, which produce urediniospores capable of reinfecting wheat, and developing into new uredinia, completing the cycle (Hovmøller *et al.*, 2011). The alternate host was only recently identified (Jin, Szabo, and Carson 2010). In the Pacific Northwest, only the asexual uredinial stage is of importance in contributing to the disease, and the sexual stages of the pathogen have rarely ever been observed (Wang and Chen 2015) in agricultural settings. Despite the extreme rarity of sexual reproduction of *Puccinia striiformis f. sp. tritici* and therefore the near total absence of sexual recombination, the pathogen has been shown to be capable of quick genetic changes. In three comparing isolates of *Puccinia striiformis f. sp. tritici* from California from 1980 and post-2000, there was less than 50% genetic similarity (Milus, Kristensen, and Hovmøller 2009). The rapid nature of these genetic changes appears to be largely due to the massive proliferation of transposable elements, DNA strands that can move their relative position within a genome (known as self-transposing). Transposable elements have been shown to comprise 17.8% of contig sequences; however, this number is probably considerably lower than the actual representation of transposable elements in the genome, as sequences with similar repeats are often assembled into common contigs, thereby masking many transposable elements (Cantu *et al.*, 2011). Transposable elements were found to make up nearly 45% of the *P. graminis f.sp. tritici* genome (Duplessis *et al.*, 2011), and it would be reasonable to believe the *P. graminis f.sp. tritici* genome contains a fairly similar percentage of transposable elements, as it has been shown that all *Pucinia* species infecting grasses form a closely related cluster (Grasso *et al.*, 2006). These transposable elements are sections of the genome hypothesized to be derived by horizontal gene transfer, which allow for significant rearrangements of pathogen genomes without requiring sexual recombination, and which have been shown to contain high concentrations of effector genes, which encode cysteine-rich secreted proteins used by microbes in pathogenesis (Ali *et al.*, 2014; Stergiopoulos and de Wit 2009). The high rate of asexual evolution of *P. graminis f.sp. tritici* likely plays a significant role in the pathogen's ability to overcome genetic resistance in wheat, as the population structure of *P. graminis f.sp. tritici* has been shown to change dramatically within 2-3 years (Bayles *et al.*, 2001), with these changes often including losing redundant effectors (Enjalbert *et al.*, 2005), which can be allowed a population of *P. graminis f.sp. tritici* that was once recognized by the host's plant defenses, to cause disease. Using transcriptomics, more than 400 genes encoding secreted proteins were discovered in the haustoria, over two-thirds of which exhibited upregulated expression compared with germinated spores, and 94 candidate effectors genes 4 were found, targeting several compounds present in wheat cells (Garnica *et al.*, 2013). The fields of genomics, transcriptomics, and biochemistry of fungal effectors has been burgeoning in recent years, with several reviews that have examined the various classes of pathogen effectors and their mechanisms in allowing for pathogenesis (Boch, Bonas, and Lahaye 2014; Lo Presti *et al.*, 2015; Stergiopoulos and de Wit 2009), including reviews focused on the effectors of biotrophic fungi (Koeck, Hardham, and Dodds 2011; Yin and Hulbert 2011). Similarly, long distance migration has also been suggested to play an important role in re-establishment of this rust each year (de Vallavieille-Pope *et al*., 2012).

5. HOST RANGE

Wheat and barley are the major hosts of *P. striiformis*. The host range of stripe rust fungus includes 320 grass species, which are from up to 50 genera belonging to subfamilies of Festucoideae and Eragosteae. Aegilops, Agropyron, Bromus, Elymus, Hordeum and Triticum are the main genera that are affected by stripe rust (Brar, 2015). *Puccinia striiformis* has been categorized into formae speciales based on specialization on different genera and species of host plants (Chen,

2005). Until now, six formae speciales were reported; Eriksson (1894) named five and more recently, Wellings *et al.*, (2004) added a new forma specialis on Hordeum spp. discovered in Australia (Line, 2002). The relationship between *P. striiformis f.sp. tritici* and *P. striiformis f.sp. hordei* is not clear because they overlap their host range by infecting barley and wheat, respectively (Chen, 2005). For such anomalies, Gassner and Staib (1932) preferred to remain with race description and avoid the use of formae speciales (Chen, 2005).

6. WHEAT YELLOW RUST DISEASE MANAGEMENT

Wheat rust can be managed either by chemicals or by disease resistance in the host plant. A large number of highly effective fungicides like Propiconazole (Tilt 25EC), Tridemifon (Bayle ton 25EC) Tebuconazole (Folicur, 250WP) etc. are available, which can control the rust fungi easily (Andenow, 1988). But chemical control is not practical and economically feasible for large scale applications. On the other hand, management of wheat rust through host by the application of resistance genes in wheat is the most effective, economic, environment friendly and practical approach for controlling rusts (Chen *et al.*, 2005). Similarly, management of yellow rust including cultural practice, application of fungicides and breeding for host resistance are the major control options (Tilahun, 2018).

6.1 Cultural practice

Cultural methods provide strategy to partially control wheat stripe rust. Using a series of cultural practices significantly enhances the existing sources of resistance. As a result, crop management in terms of a combination of crop choice, timing of seeding and removing volunteer cereals may provide effective control of stripe rust (Roelfs, 1992; Wan *et al.*, 2007).

Yellow rust requires green material to survive from one season to next, it is known as “green bridge”. That means it survives all year round by having a continuous supply of living host plants. Thus volunteer wheat plants growing over summer provide a ‘green bridge’ for yellow rust to survive, making it very difficult to find at that time of year. Removal of the “Green Bridge”; that is to remove volunteer wheat plants that support yellow rust infection, six weeks prior to sowing the seed can minimize the impact of yellow rust. Uncontrolled volunteer wheat, particularly susceptible varieties, on one farm is a threat to neighbouring farms and, with longer distance spread, to the whole area. Thus, it is vital that all farmers reduce susceptible volunteers to a minimum. A key to reduce the threat from yellow rust is to remove volunteer wheats by grazing, spraying or cultivation before the next season’s crops are sown. This has been reported that it is an effective control measure for epidemics that result from endogenous inoculum (Roelfs, 1992). This strategy will not eliminate all yellow rust, because some wheat plants may survive along roadsides, etc. However, the widespread elimination of the green bridge throughout the area substantially reduces the early season pressure from yellow rust, delaying the epidemic and helping in crop management. The other cultural practice that can be used to control wheat yellow rust disease is planting a mixture of wheat varieties with different resistance backgrounds that can significantly reduce disease pressure and may also increase or stabilize wheat yield (Wolfe, 1985). Mechanisms by which cultivar mixtures suppress disease may include dilution of spore’s density because of the greater distance between susceptible plants, a physical barrier created by the resistant plants in the canopy that interrupt spore movement and induced resistance (Castro, 2001; Huang *et al.*, 2012) are also convenient.

Early sown crops also provide a green bridge for yellow rust. Fungicide-treated seed or fertilizer treatments will protect these crops and reduce the chances of an early build-up of yellow rust. Because wind can carry spores for long distances, a build-up of yellow rust in one early sown crop threatens later crops over a very wide area. Where most farmers adopt a combination of controlling volunteers and protecting early sown crops, the yellow rust population will be minimal, delaying the start of the epidemic and making spring control simpler. Keeping yellow rust at a low level is also important for reducing the appearance of new pathotypes.

The nitrogen status of a wheat crop affects the level of yellow rust. This is independent of the effect of nitrogen creating a denser canopy with higher humidity that would additionally favour the development rust diseases in general and yellow rust in particular. Very little yellow rust develops in nitrogen deficient crops, and the leaves seem to develop an early and very effective adult plant reaction. Conversely, APR seems to be delayed in crops with high nitrogen status.

Nitrogen (N) is the most important fertilizer element determining the productivity of wheat. N is a vital component for vegetative growth and chlorophyll formation (Gooding & Davies 1997) and grain development in wheat (Wright Jr.

2003). However, excessive availability of N can heighten the risks of lodging, frost damage; foliar disease (Olesen *et al*. 2003a) and can also delay crop maturation (Gooding & Davies 1997). If excessive rates of N are applied which are not balanced by stored soil water and/or in crop rainfall then this can result in moisture stress during grain filling which results in premature ripening of the crop; referred to as 'haying-off' (Herwaarden *et al.* 1998). N nutrition is also an important factor affecting quantitative resistance to foliar diseases and high N is associated with increased severities of a number of foliar diseases. This can arise from changes in biochemical processes in the plant and/or creation of a more favourable microclimate for fungal diseases because of increased crop density and canopy size (Bryson *et al.* 1997; Jensen & Munk 1997). Nitrogen availability to a crop could therefore be one of the crucial factors for determining canopy growth and the incidence and severity of stripe rust. Understanding the interaction of these factors is of paramount importance in the assessment of productivity and modelling biomass and final grain yield from the crop. Crop rotation also helps to limit the genetic diversity of the pathogen population and to minimize the number of urediospores produced and accumulated in the season (Bariana *et al.*, 2007).

6.2 Chemical control

Fungicides can be the first line of defence against new varieties of wheat rust. In wealthier countries, fungicides are used on a large scale as a key part of the national food security strategy (ICARDA, 2011). However, resource poor farmers in developing countries cannot always afford chemical treatment of fields affected by rust. But chemical interventions do provide a practical, rapid-response solution, for example, to stop local intrusions of new rust races, giving time to assess the races and multiply new resistant varieties. In the high-income countries of Europe, Australia and the Americas, farmers can manage disease pressures in wheat production with fungicide applications. But farmers in developing countries cannot do this. Many developing countries do not have the right fungicides registered on time and at an affordable cost, necessary sprayers, protective clothing, and knowledge for their use or relevant training to combat wheat rusts (ICARDA, 2011). For fungicides to be effective, information on disease onset is required and for this surveillance, systems are needed. Except these problems, emergency fungicides are used where possible in developing countries. Examples in Ethiopia in 2010 over $3 million worth of fungicides were distributed and fungicides were spread on 30% of the wheat growing areas (Abeyo, 2014). Control of yellow rust by chemical products is available with new and more effective fungicides such as Tilt®(propiconazole), Quadris® (azoxystrobin), StrategoTM (propiconazole + trifloxystrobin), HeadlineTM (strobilurin), and QuiltTM (azoxystrobin + propiconazole) (Chen, 2005). Application of fungicide solution of propiconazole 25 EC (Tilt), tebuconazole 250 EC (folicur) or triadimefon 25 WP (Bayleton) is recommended to control the spread of yellow rust. Strobilurinsis a fungicide, which provides excellent control of yellow rust and is most effective when, applied before infection, and if stripe rust is already present, it is better to apply the triazoles (Eddy, 2009). However, growing resistant cultivars is the most efficient, economical and environmentally friendly approach to control the disease (Line and Chen, 1995). Much of the early research on use of fungicides to control stripe rust in the United States was done by Hardison (1963, 1975), Powelson and Shaner (1966), and later, by Line and his associates (Line and Rowell 1973; Line 1976; Rakotondradona and Line 1984). The first large-scale, successful use of fungicides to control stripe rust in North America occurred in 1981 (Line 2002). By that time, Line and his associates had demonstrated the effectiveness of triadimefon (Bayleton) and developed guidelines for the timely application for economical control. Through their efforts, triadimefon received emergency registration and was widely used in the PNW (Line 2002). Stripe rust was unusually severe throughout the PNW in 1981. When stripe rust was not controlled, highly susceptible cultivars, such as the winter club wheat 'Omar', were destroyed, while cultivars with moderate resistance to stripe rust had yield losses of 50%. Fungicide use prevented multimillion-dollar losses (Line 2002). New foliar fungicides highly effective for control of stripe rust subsequently have been registered. In the 1990s, Cu and Line (1994) developed an expert system called MoreCrop (Managerial option for reasonable economical control of rusts and other pathogens) that combined information on management practices and use of fungicides and resistant cultivars into an integrated disease management program. Use of MoreCrop continues today. Annual tests to determine efficacy, rates, and timing of fungicide application for control of stripe rust continue (Chen and Wood 2002, 2003, 2004), and new and more effective fungicides have been registered. Five fungicides, Tilt® (propiconazole), Quadris® (azoxystrobin), Stratego™ (propiconazole + trifloxystrobin), Headline™ (strobilurin), and Quilt™ (azoxystrobin + propiconazole) are currently registered in United States for use on wheat and barley to control stripe rust. Data on yield losses from stripe rust and on yield increases attained with fungicide application for each cultivar are used to guide growers on whether or not to use

fungicides. In recent years, use of fungicides has successfully reduced yield damage caused by stripe rust. In 2002, when stripe rust was widespread on susceptible and moderately susceptible spring-wheat cultivars, fungicide application (at a cost of about US$2.5 × 106) saved Washington State wheat growers about US$30 × 106 (Chen *et al.*, 2003a). Furthermore, foliar fungicides can effectively control stripe rust. Applied when the crop is at the boot stage of development, the fungicides should provide protection for the upper leaves that contribute most of the energy used to produce grain. Products belonging to the strobilurin class of fungicides (Headline, Quadirs) provide excellent activity against stripe rust but are most effective when applied before infection. If stripe rust is already present in a field at the time of application, it may be better to use products belonging to the triazole class of fungicides (Folicur, Prosaro, Tilt) or premixes of the two classes (Quilt, Stratego, Twinline). The triazole class of fungicide is generally considered to have stronger curative activity. Seed treatment fungicides can also be used for the control of stripe rust in seedlings. Four active ingredients: flutriafol, triadimenol, triticonazole and fluquinconazole can be used as seed treatment fungicides. Most are available under more than one trade name and formulation. The rates of application vary depending on which diseases require control, with generally the higher rate of application preferred for stripe rust. Flutriafol, Triadimenol and Triticonazole provide control of stripe rust for about 8 weeks after sowing, which delays the onset of the disease within a crop. The length of protection varies: experience has shown that stripe rust will begin to develop during the stem elongation stage between first node (GS 31) and flag leaf emergence (GS 37). These treatments appear to have most value for early-sown crops to provide protection during autumn and for late-sown crops with good APR. The seedlings take up Fluquinconazole more slowly than the other fungicides, so a higher rate can be applied, giving control for up to 12 weeks after sowing. In practice, control seems to have been good to about the booting stage (heading stage in later sown crops), but susceptible crops should be watched closely from this time, in case a supplementary fungicide spray is required. This treatment will also provide protection from take-all. Thus it appears most useful for replacing the need for an early spray, and where protection from take-all is desirable. Four triazoles (flutriafol, propiconazole, tebuconazole and triadimefon), and two composites, (azoxystrobin plus cyproconazole and propiconazole plus cyproconazole), are registered under several trade names as fungicide sprays to control stripe rust. All give excellent control providing they are applied early in the epidemic. For best results, foliar fungicides should be applied before stripe rust becomes well established in the crop. Once stripe rust has been occurred, the optimum timing of application depends in part on the reaction of the variety to stripe rust and on the growth stage of the crop. Regardless of variety, spraying should be done before stripe rust reaches 5% of leaf area on the flag leaf. Once this level is reached, stripe rust becomes very difficult to control. In addition, the leaves will continue to die for about a week after spraying because infections continue to develop. Late spraying of severe stripe rust in susceptible wheats has given an economic response in crops with high yield potential, but it is far less than that if the spray had been applied earlier. Fortunately, such early starts are rare. Seed or fertilizer treatment best controls them. Foliar fungicides can be applied at the low rate, but will need to be repeated at 3–4 week intervals to protect newly emerging leaves. It is critical to control stripe rust on the top two leaves, as these contribute most to the yield. A spray will protect leaves for 3–5 weeks depending on fungicide, variety and seasonal conditions.

Nevertheless, the use of fungicides adds a huge cost to wheat production, which is a burden for many growers, especially in developing countries. The use of fungicides may also create health problems for users, adversely affect the environment, and result in the selection of fungicide resistant strains of the pathogen. Similarly, the use of fungicides in Ethiopia is limited by the fact that most wheat farmers are small holders who are resource constrained and cannot afford chemicals (Bishaw *et al.*, 2010; Tilahun, 2018). In addition, the chemical fungicides are environmentally unsafe (Bux *et al.*, 2012; McCallum *et al.*, 2016; Tilahun, 2018). To avoid these problems, growing cultivars with adequate level of durable resistance is the best strategy to control stripe rust.

6.3 Host plant resistance

The fungal diseases stripe rust (caused by Puccinia striiformis Westend. f. sp. tritici Eriks) result in significant yield losses to wheat production world-wide (Kolmer 2005; Hovmøller *et al.* 2011; Szabo *et al.* 2014). Stripe rust can cause up to 100% yield losses in susceptible wheat cultivars (Chen 2005; Huerta-Espino *et al.* 2011; Singh *et al.* 2015). Several epidemics and outbreaks of stripe significantly threatened the food security and livelihoods of poor farmers in many wheat growing regions worldwide (Wellings 2011; Solh *et al.* 2012). Moreover, the emergence and spread of novel stripe rust races have led to the breakdown of most of the widely deployed resistance genes used in wheat production (Huerta-

Espino *et al.* 2011; Wellings 2011; Singh *et al.* 2015). However, deployment of host-plant genetic resistance is still seen as the most economically and environmentally safe approach to reduce losses due to rust diseases in wheat (Burdon *et al.* 2014; Singh *et al.* 2016) and it is an excellent means of controlling yelow rust disease. Resistance limits infection, fungus growth and spore formation. Resistance levels vary among varieties and are classified accordingly: R = resistant, MR = moderately resistant, MS = moderately susceptible and S = susceptible. A better understanding of pathogen virulence structure and the divergence of race(s) associated with major epidemic sites at different continents will be useful to facilitate breeding of resistant or less susceptible crop varieties and the development of appropriate disease management strategies based on host resistance (Johnson, 1992; Hawkesford *et al.*, 2013). New efforts have been made to investigate the yellow rust population genetic structure at worldwide scale, describing the worldwide population subdivisions, sources of invasions and the existence of center of diversity in the Himalayan and near Himalayan region (Ali *et al.*, 2014a,b; Thach *et al.*, 2016a; Walter *et al.*, 2016). However, the virulence structure of the pathogen has often only been described at country or regional scales, (Chen, 2005; Zeng and Luo, 2006; Hovmøller and Justesen, 2007a; Wellings, 2007; Bahri *et al.*, 2009b, 2011; Ali *et al.*, 2014c; Hovmøller *et al.*, 2016). Since the work of R.W. Stubbs from 1950s to 1980s (Stubbs, 1988; Thach *et al.*, 2015), only a single study about yellow rust virulence at the international scale has been published (Sharma-Poudyal *et al.*, 2013) and attempts to link virulence and race structure with the recent regional yellow rust epidemic outbreaks in different parts of the world are missing. Adaptation of new varieties which are proven resistant to stripe rust and cultivation of these resistance varieties is the best approach to control wheat losses to stripe rust (Line, 1972; Konzak et al., 1977; Robbelen and Sharp, 1978; Line and Chen, 1995; Wan et al., 2007). Australia alone saves about A$124 million annually by using resistant cultivars (Brennan and Murray, 1988). Effective management of diseases can be informed by understanding of its epidemiology (Gilligan 2002; Merz and Falloon 2008). Due to yellow rust being a polycyclic disease, in which urediniospores are aerially dispersed largely very close to the source, but with significant dispersal very far from their source lesion, a small amount of disease early in a growing season can lead to a severe epidemic (Estep, Sackett, and Mundt 2014; Severns et al. 2014). In other rusts, urediniospores have been found to travel upwards of 800 kilometers (Hermansen, Torp, and Prahm 1976). This long-distance dispersal makes effective quarantines nearly impossible. Due to the ability of yellow rust to accelerate quickly, to cause significant losses, and to be able to invade remote areas, early and regular scouting is recommended to allow field managers to make informed decisions to combat yellow rust (Gaunt and Cole 1992), along with use of trap nurseries (Duveiller, Singh, and Nicol 2007). Once disease is established in a stand of susceptible hosts, such as an agricultural field, given a conducive environment, yellow rust will rapidly accelerate if not managed. However, fungicides, most notably those with some degree of systemic or translaminar action (strobilurins and triazoles) (Conner and Kuzyk 1988) have shown some reduction in yellow rust epidemics and increase in grain yield over untreated controls, although these increase in yields were generally under 50% and did not significantly increase with two sprays relative to one (Sharma *et al.* 2016). Overuse of fungicides can also lead to fungicide resistance (Hahn 2014; Russell 1995), reducing their effectiveness in the long term. Therefore, fungicide use is best thought of as a tool to augment other management strategies, particularly breeding for resistance, in an integrated pest management approach (IPM) (Chen 2007; Mundt, Cowger, and Garrett 2002). Resistance to plant pathogens has been largely grouped into two principle classes: major gene, also termed vertical or R gene resistance, in which the infected cell of a host recognizes a specific effector compound secreted by the pathogen, causing a chemical pathway leading to a hypersensitive response (HR), also termed programmed cell death, killing the plant cell along with the attacking pathogen (del Pozo and Lam 1998; Greenberg 1996; Williams 1994); and minor gene, also known as quantitative or horizontal resistance, in which a plant exhibits certain traits which reduce the ability of a pathogen to cause disease but do not eliminate it (Nelson 1978; Niks, Qi, and Marcel 2015; Poland *et al.* 2009). Both of these forms of resistance are present in the wheat genome (Lewellen, Sharp, and Hehn 1967), and have been actively bred to increase the level of and durability of resistance (Hou *et al*. 2015). Due to single R genes controlling most major gene forms of resistance, it has been much easier for *Puccinia striformis f.sp. triticci* to overcome resistance, particularly in fields with high degrees of selection pressure resulting from extensive use of genotypes containing the same single R gene (Ellis *et al.* 2014), although a pyramiding multiple R genes into a cultivar has been employed to reduce the probability of pathogens overcoming resistance (Castro *et al.* 2003; Mundt 1991). Quantitative resistance, therefore, is considered generally more durable, and has been the focus of many breeding efforts involving quantitative trait loci (QTL) identification (Hou *et al*. 2015; Li *et al*. 2010; Liu *et al.* 2015), which can also be pyramided (Richardson *et al.* 2006). A particularly widely deployed form of quantitative resistance to yellow rust is HTAP (high temperature adult plant

resistance) (Bux, Ashraf, and Chen 2012; Uauy *et al.* 2005), in which the Yr36 gene drastically reduces yellow rust epidemics in mature plants in warm temperatures. HTAP is less racespecific than R gene resistance, and therefore has led to greater durability (Chen *et al*. 2002). In addition to resistance breeding and fungicide applications, cultural methods can reduce yellow rust epidemics, such as planting at a later date in the fall to reduce the probability of early establishment of yellow rust, refraining from over-fertilization with nitrogen-based fertilizers, removing nearby refuges for *Puccinia striformis f.sp. triticci*, such as susceptible grasses and volunteer wheat, avoiding overwatering, particularly when conditions are conducive to infection, and reducing connectivity of susceptible host populations including use of multiline cultivars (Mundt 2002; Neumann et al. 2004; Papaïx et al. 2014; Reddy 2013; Roelfs, Singh, and Saari 1992; Stevens 1960). Use of multiline cultivars and gene pyramiding to expand the life span of all-stage resistance, have been successfully used to control stripe rust (Chen, 2007; Allan *et al.,* 1993; Chen, 2005). Growing mixtures of wheat cultivars in yellow rust infested area yields higher than the pure strand of all the mixtures (Knott and Mundt, 1990; Finckh and Mundt, 1992). Wheat scientists have recognised the temperature effects on resistance, which was described as high-temperature-adult-plant (HTAP) resistance (Sharp, 1965; Lewellen *et al.*, 1967; Brown and Sharp, 1969; Line et al., 1974; Qayoum and Line, 1985). HTAP resistance has been incorporated through molecular markers. Use of HTAP cultivars is another approach in fighting with yellow rust. A combination of HTAP resistance and effective all-stage resistance is the best approach to develop durable and high-level resistance (Chen, 2013). Incorporation of resistance based on additive slow rusting genes by the use of single backcross approach has shown 5-15% of higher yield potential than the original cultivar and many varieties have also been developed by this methodology (Singh *et al*., 2005). So far, 82 Yr genes have been formally designated; about 25 of these confer adult plant resistance or high temperature adult plant resistance while the remainder provide all stage resistance (McIntosh *et al.* 2016, 2017; Wang and Chen 2017). An effective deployment of resistance genes for the management of yellow rust in wheat requires knowledge about the resistance status and the diversity of resistance genes in cultivars under consideration. Moreover, knowledge on the prevailing pathogen races is crucial as pathogens evolve their virulence frequently, thereby compromising the durability of resistance (Pretorius *et al.,* 2000; Jin *et al.,* 2008; Jin *et al.,* 2009; Tilahun, 2018). Periodic outbreaks of stripe rust occur in Ethiopia due to lack of knowledge regarding the genetic resistance present in commercial cultivars and breeding populations, and inadequate monitoring of the pathogen race population (Badebo 2002). In Ethiopia, breeding for resistance is solely based on field observations at naturally infected trial sites. There is only limited information on the genetic composition of current cultivars and even less on materials undergoing selection (Dawit *et al.* 2012). Identification and development of slow rusting resistant varieties can reduce the cost of production and frequency of serious epidemics. Wheat genetic materials with related parentage and largely carrying race-specific major gene resistance have been the backbone of the wheat improvement programs of Ethiopia (Badebo, 2002). However, most of the released varieties in the country do not possess durable resistance, became susceptible shortly after their introduction, and released. In most cases, the failures were due to new virulent pathotypes and deployment of the same resistance gene(s) in wide array of wheat cultivars (Admassu *et al.,* 2012; Wan and Chen, 2012; Wubishet and Chemeda, 2016). Slow rusting wheat cultivar is the simple solution for disease management, thus replacing susceptible cultivars with slow rusting ones is important in resistance diversity and to create an opportunity for further improvement of resistance level of wheat (Taye *et al.,* 2015). For such rapid evolution and spread of new virulent races of yellow rust, and frequent failure of new varieties with major gene yellow rust resistance in wheat improvement programs require to identify durable sources of resistance (Hei *et al.,* 2015). Therefore, achievement of slow rust resistance against wheat yellow rust requires constant characterization and identification for deployment of new resistant genotypes that resist the prevailing virulent races.

6.4.1Types of host plant resistance

The evolution of new virulence through migration, mutation, recombination of existing avirulence genes in the pathogen, and the selection of virulence has been common in the fungi that cause rust. Therefore, genetic resistance is the most economical and preferable method to control the diseases (Kolmer 1996; Singh *et al.,* 2008c). The genetic resistance to yellow rust can be characterized as qualitative and quantitative resistances. The qualitative resistance is classified into race-specific or vertical, seedling resistance, monogenic (major genes), hypersensitive, and the quantitative resistance is classified into the race-nonspecific or horizontal, adult plant resistance, slow rusting, polygenic (minor gene), durable etc. (Flor, 1956; McIntosh, 1988; 1995; Rajaram *et al.,* 1988; Singh *et al.,* 2000; Parlevliet, 2002; Chen, 2005; Clair, 2010; Lowe *et al*., 2011).

6.4.1.1 Race-specific resistance

Race-specific resistance is also known as vertical, hypersensitive, major gene, or seedling resistance. There is an obvious differential reaction, pathotypes can be determined, and race-specific resistance genes are recognised by the presence of low infection types (Dyck and Kerber 1985; Singh and Rajaram 2002). Major genes are at risk due to pathogen plasticity (Singh and Rajaram 2002). Gene-for-gene resistance is called major gene resistance or race-specific resistance because it is effective only against some pathotypes of the pathogen population and it often "breaks down" easily with the occurrence of new pathotypes of a pathogen (McDonald and Linde 2002; Knott 2008). Because cultivars with major genes usually do not remain resistant longterm, research focus has been on using non-race-specific resistance that is controlled by many genes in recent years (Qayoum and Line 1985; Lin and Chen 2009). Furthermore, the gene-for-gene relationship states that for every resistance gene in the host plant there is a corresponding avirulence gene in the pathogen. However, the ability of an avirulent gene to mutate to a virulent gene, no longer recognizable by the corresponding resistance gene, implies a type of resistance termed race-specific resistance (Flor, 1971). According to Dyck and Kerber (1985), a race-specific or vertical resistance signifies that the resistance to some pathogens is relatively simply inherited. The race-specific resistance is virulent only to particular races of a pathogen. Race-specific resistance is often based on genes that are effective at the seedling stage and remain effective at all post-seedling stages of the plants. Race-nonspecific resistances are mainly effective at the post-seedling and adult plant stages and adult plant resistance (APR) is often detected as field resistance (Johnson, 1992; Hovmøller *et al.*, 2011). Most of the yellow rust resistance genes are determined at seedling stages, and thus interact with specific races of the pathogen to confer resistance in a gene-for-gene relationship (Flor, 1971). Race specific resistance is usually governed by a hypersensitive response, controlled by major genes. The race-specific resistance is also known as monogenic resistance (resistance determined by a single gene), often led by a boom and bust cycle (Dyck and Kerber, 1985; Nagarajan and Joshi, 1985; Priyamvada and Tiwari, 2011). Yellow rust resistance genes are postulated or characterized based on seedling resistance test. The seedling resistance genes can be detected and are effective at the seedling stages, and they are characterized by the gene-for-gene interaction model (Flor, 1971). Generally, the seedling resistance genes are also active during the adult plant stage, and they are classified into race-specific resistance types (Chen, 2005; Lagudah, 2010). So far, above sixty yellow rust race-specific resistance genes based on seedling resistance test have been identified (McIntosh *et al.*, 2010; Singh *et al.*, 2011). However, the seedling resistance genes are often broken down due to new and various races of the rusts pathogen (Chen and Moore, 2002).

6.4.1.2 Race non-specific resistance

The genetic nature of this type of rust resistance is usually complex and is based on the additive interaction of a few or several genes having minor to intermediate effects. This type of resistance is characterised by a non-differential interaction. Non-specificity is only recognised by the absence of specificity and, because all tests are of limited size, the presence of race-non-specificity can never be proved (Johnson 1984). It is not possible to define pathotypes based on this type of resistance, and it generally allows low level of rust sporulation (Parlevliet 1985; Singh and Rajaram 2002). Deployment of resistance genes at various stages of a plant development in different years and regions resulted in a durable non-race-specific, field resistance of adult plants (Börner *et al.*, 2000). Van der Plank (1968), described race non-specific resistance to be characterized by reduced apparent infection rate. Thus, resistances that varied in a quantitative way and resulted in slow rusting were accepted to be supported by race non-specific resistance genes (Parlevliet, 1985). Race non-specific resistance conditioned by polygenes or quantitative genes is generally complex, as is its identification. Most of the race non-specific resistance tests have been carried out in adult plants (Roelfs *et al.*, 1992). Thus, adult plant resistance genes are considered to control race non-specific resistance, thereby contributing with partial resistance and being associated with a slow rusting resistance (Priyamvada and Tiwari, 2011). The stem rust resistance gene Sr2 is considered to be one example of a gene contributing to partial or slow rusting resistance (McIntosh *et al*., 1995; Bansal *et al*., 2008). The race non-specific resistance is governed by minor genes and is therefore considered as a polygenetic resistance. This type of resistance is often considered as durable and the genes are pyramiding. Most commonly, race non-specific resistance is characterized by durability, having a partially resistant phenotype, and being effective to a broad range of stem rust and yellow rust races with optimal level of expression at the adult plant stages (Parlevliet, 1985; McIntosh *et al.*, 1995). Wheat breeders and pathologists have always been concentrating on adult plant resistance genes in order to identify and improve the level of resistances (Bansal *et al.*, 2008). The detection of adult plant resistance is

usually conducted at the post-seedling stage, and is often characterized as field resistance (Van der Plank, 1982; Lagudah, 2010). Adult plant resistance genes are effective only in adult plant resistance stages, but have been shown to be an important part of durable rusts resistance (Johnson, 1978; Priyamvada and Tiwari, 2011). The principle of adult plant resistance may derive at any time during the post-seedling stage and environmental factors (i.e. high and low temperature, climate change etc.) may interact for the adult plant resistance gene expression (Bariana and McIntosh, 1995). To characterize adult plant resistance genes severities scored based on the modified Cobb scale (Peterson *et al.*, 1948) and the reaction types scored based on descriptions by Roelfs *et al.*, (1992) should be carried out.

6.4.1.3 Slow rusting

Slow rusting is a type of resistance in which disease develops slowly, resulting in intermediate to low disease levels against all pathotypes of a pathogen (Caldwell 1968; Johnson 1984). The components that cause slow rusting in a variety are longer latent period, low receptivity or infection frequency, as well as smaller uredinial size and reduced duration and quantity of spore production. All these components can affect disease progress in the field (Singh and Rajaram 2002). Susceptible wheat cultivars have larger and more uredinia than the slow rusting wheat cultivars.

6.4.1.4 Partial resistance

The definition of partial resistance is based on leaf rust resistance (caused by *Puccinia hordei*) in barley. It is a form of incomplete resistance characterised by a reduced rate of disease spread despite a high or susceptible infection type (Parlevliet 1985). It is apparently more durable than hypersensitive resistance. In the field, epidemic parameters like latent period, infection frequency, pustule size, infectious period and spore production are correlated with partial resistance expression in barley leaf rust system (Qi *et al.*, 1998).

6.4.1.5 Durable resistance

Johnson (1988) defined durable resistance as resistance that remained effective in a cultivar during its widespread cultivation for a long sequence of generations or period of time in an environment favorable to a disease or pest. Self-pollinating cereal crops like wheat are often grown over large areas as genetically uniform cultivars. This gives the opportunity for the development and selection of new virulent pathotypes of a pathogen on particular cultivars. While this may happen before the cultivar is widely exploited, certain cultivars have been grown widely for many years with the resistance present remaining effective (Johnson and Law 1975). Durable resistance usually, but not always, represents the adult plant resistance which is conferred by combinations of several genes usually with minor effects but acting additively, and which is not generally associated with genes giving hypersensitive reactions (McIntosh 1992b). Examples of durable resistance in wheat include resistance to stripe rust in the French bread wheat variety 'Camp Re´my', which was grown and remained resistant to all known pathotypes of *Puccinia striformis tritici* for more than 20 years in France (Mallard *et al.*, 2005). Quantitative resistance, usually under oligogenic or polygenic control, is generally more effective in the field and appears to be more durable. The stem rust resistance gene Sr2, derived from an 'Emmer' (tetraploid) wheat via the variety 'Hope', provides adult plant resistance that has remained durable and effective in combination with other minor genes in Australia, the USA and many parts of world (Johnson 2000; Singh *et al.*, 2004). Knott and Padidam (1988) also reported the effectiveness of minor genes with additive effect for stem rust resistance. According to Roelfs (1988b), Frontana, a South American variety is the best sources of durable resistance to leaf rust carrying leaf rust resistance genes Lr34 and two to three additional genes (Singh *et al.*, 2004b). Johnson (1988) also stated that some European wheat cultivars have durable rust resistance that is quantitative in nature.

6.4.2 Sources of Resistance

Since, new races of yellow rust are spreading throughout the worldwide where wheats are produced, identification and transfer of novel sources of resistance genes is necessary. A number of wheat lines with transferred genetic material from related species are available such as wheat-rye, wheat-leymus and wheat-thinopyrum translocations/substitutions. The incorporation of genetic material from related species leads to wheat varieties adapted to the environment of interest, and to greater sustainability of the wheat production. New sources of resistance genes can be obtained from various sources in the primary, secondary and tertiary gene pools of wheat. One promising source of new genes for wheat is the tertiary gene pool, which includes Secale cereale, Agropyron spp., Leymus spp. Thinopyrum spp. and Hordeum vulgare (Mujeeb-Kazi, 2006; Dundas *et al.*, 2007). Rye has been among the most successfully used alien resources contributing against biotic and

abiotic stresses for wheat (McIntosh, 1991; Dvorak and McGuire, 1991; Jiang *et al.*, 1994; Stephen *et al.*, 1995). Moreover, genes Yr8, Yr9, Yr15, Yr17, Yr24/26, Yr35, Yr36, Yr53, Yr64 and Yr65 were obtained from diploid and tetraploid wild and cultivated relatives, e.g., Yr15 derived from Triticum dicoccoides (Gerechter-Amitai *et al.*, 1989; Zeray *et.al.*, 2020), Yr8 from Aegilops comosa (Riley *et al.*, 1968), Yr9 from Secale cereale (Macer 1975), Yr17 from Ae. Ventricosa (Tanguy *et al.*, 2005), Yr28 and Yr48 from Ae. ventricosa (Lowe *et al.*, 2011; Singh *et al.*, 2000; Zeray *et.al.*, 2020),), Yr37 from Ae. Kotschyi (Heyns *et al.*, 2011), Yr38 from Ae. sharonensis (Marais *et al.*, 2010), Yr40 from Ae. geniculata (Kuraparthy *et al.*, 2009), Yr42 from Ae. geniculata (Marais *et al.*, 2009). Yr50 from Thinopyrum intermedium (Liu *et al.*, 2013), Yr70 from Ae. umbellulata (Bansal *et al.*, 2016). Other genes came from hexaploid wheat landraces (McIntosh *et al.*, 2016; Zeray *et.al.*, 2020),). Among the permanently designated ASR genes, Yr5, Yr15, Yr53, Yr61, Yr65 and Yr69 are still widely effective and can be used in breeding for stripe rust resistance (Xu *et al.*, 2013; Zhou *et al.*, 2014) provided they are not associated with detrimental linkage drag. Following the discovery of the gene-for-gene interaction between plant hosts and their pathogens (Flor 1971), host resistance genes and their corresponding pathogen avirulence genes could be postulated. Dawit *et al.*, (2012) tested 22 Ethiopian bread wheat cultivars and 24 differential lines with 20 *Puccinia striformis trittci* races collected from Ethiopia, France and Germany. They postulated different combinations of Yr2, Yr3a, Yr4a, Yr6, Yr7, Yr8, Yr9, Yr27, Yr32 and YrSU in tested materials. Hovmøller (2007) reported Yr1, Yr2, Yr3, Yr4, Yr6, Yr9, Yr15, Yr17, Yr25 and Yr32 in 98 Danish wheat cultivars; Xia *et al.*, (2007; Zeray *et.al.*, 2020),) detected Yr2, Yr3a, Yr4a, Yr6, Yr7, Yr9, Yr26, Yr27, YrSel and YrSd in 72 Chinese wheat cultivars and advanced lines; and Sharma *et al.*, (1995) reported Yr2, Yr6, Yr8, Yr9, Yr10, Yr15, YrA and YrSu in tests of 52 wild emmer derivatives and advanced bread wheat lines from Nepal. The drawback of this method of gene postulation is that while it is the most effective for identifying race-specific single resistance genes or simple gene combinations at the seedling stage, it cannot be easily used to identify adult-plant, often non-specific resistance genes that tend to be more common in the case of stripe rust (Wang and Chen 2017; Zeray *et.al.*, 2020). Breeding for yellow rust resistance always requires a constant inflow of novel sources of resistance genes, due to the appearance of new virulent pathogen races (Singh *et al.*, 2011; Lowe *et al.*, 2011). Resistance breeding might utilize novel yellow rust resistance genes by means of wheat-rye, wheat-leymus and wheat-thinopyrum introgression lines. Ultimately, these identified genes will be used to develop high yielding wheat cultivars, keeping in mind food security, environmental issues and human health.

Bread wheat (*Triticum aestivum* L.) is a hexaploid species constituted of the AABBDD genome. The donors of the wheat genome are: AA Triticum urartu, BB Aegilops speltoides and DD Aegilops tauschii (Dvorak, 1998). Wheat belongs to the tribe Triticeae of the family Poaceae. According to crossability with hexaploid wheat, other related species are divided into three major gene pools: The primary gene pool; the secondary gene pool; and the tertiary gene pool (Mujeeb-Kazi and Rajaram, 2002). These gene pools can play an important role for present day wheat breeding when introducing novel sources of resistance to develop resistant cultivars toward yellow rust (McIntosh *et al.*, 1995; Tyrka and Chelkowski, 2004; Singh *et al.*, 2011). The primary gene pool of bread wheat consist of species that have genomes homologues, with bread wheat, Triticum aestivum (AABBDD), e.g. hexaploid spelt (Triticum spelta AABBDD), tetraploid Triticum turgidum (AABB), diploid Triticum monococcum (AA), Triticum dicoccoides, Aegilops tauschii (DD), as well as landraces of hexaploid and tetraploid wheat (Mujeeb-Kazi and Rajaram, 2002). The desired genes within this group are possible to transfer via direct hybridization, homologous recombination, backcrossing, and selection (Friebe *et al.*, 1996; Mujeeb-Kazi and Rajaram, 2002). The secondary gene pool of hexaploid wheat contains polyploid Aegilops and Triticum species that have one genome in common with Triticum aestivum e.g. Triticum timopheevii (AAGG) and Triticum araraticum (AAGG). Some Aegilops species share the evolution of wheat and have played an important role in wheat domestication. Examples of such Aegilops species include the Sitopsis section related to the B genome of hexaploid wheat, e.g. Aegilops speltoides and Aegilops longissima (2n=2x=14). Thus, the genus Aegilops represents the largest part of the secondary gene pool of wheat, and several species have been used by direct crossing, backcrossing, selection via chromosome recombination, embryo rescue and cytogenetic manipulations to enhance the recombination in wheat improvement programs (Mujeeb-Kazi and Rajaram, 2002; Mujeeb-Kazi, 2003; Kilian *et al.*, 2011). Diploid and polyploid species, which are members of the tertiary gene pool of hexaploid wheat, have non-homologous genomes with hexaploid wheat. One promising source of novel genes for wheat is wheat grasses and wild rye both being included in the tertiary gene pool. This gene pool has been successfully hybridized with wheat and genes have been incorporated into the bread wheat genome, representatives are from Agropyron, Pseudoroegneria, Psathyrostachys, Thinopyrum, Elymus, Secale cereale, Hordeum vulgare and Leymus species (Dewey 1984; Mujeeb-Kazi and Wang 1995; Wang and Jensen 2009).

However, the tertiary gene pool species have been limitedly exploited in wheat, because the genomes of these species are non-homologous to those of wheat, and genetic transfers cannot be made by homologous recombination. In order to incorporate genome of these species, special techniques such as embryo rescue, irradiation etc., and further cytological manipulation are required (Friebe *et al.*, 1996; Mujeeb-Kazi and Rajaram, 2002; Mujeeb-Kazi, 2003). Consequently, in this gene pool usually linkage drag is the effect, which could be associated with undesirable agronomic traits (Qi *et al.*, 2007; Gill *et al.*, 2011), and due to homoeology the linkage block might be inherited (Hanson, 1959a; 1959b; Pumphrey, 2012). Despite this, some of the yellow rust resistance genes are originating from tertiary gene pool species.

7. CONCLUSION

Wheat yellow rust has been reported as an increasing problem with repeated cases of worldwide invasions. It has been reported in more than 60 countries and on all continents except Antarctica. Moreover, it is globally the most damaging diseases in many cool and temperate regions of the world and is considered the most economically important disease of wheat production worldwide. Global losses were estimated to be at 5.5 million tons per year. Though there are different yellow rust management methods are being practiced, growing resistant cultivars is the major component of integrated control of yellow rust. However, the "breakdown" of resistance following the introduction of new genes for resistance is a major problem. Successful deployment of resistant crop varieties at larger scales and in different regions would, however, require: a better understanding of pathogen diversity; regional and international collaboration to effectively address the disease through data sharing; a long term effort to control new and existing challenges to yellow rust through research and development of resistant varieties to emerging strains. Moreover, management of rust diseases is complex because of their rapid dissemination and the frequency of evolution of new physiologic races and thus demands a perfect knowledge of all the elements of epidemics that are interrelated. Therefore, a combination of different strategies should be used to manage rusts in wheat production. However, the use of resistant cultivars is the most important and the most practical method of controlling yellow rust of wheat and is the best strategy for resource poor farmers in the developing world. It is also the most environmentally friendly and profitable strategy for commercial farmers if they grow genetically resistant varieties using different resistance genes. Generally, there should be a need for a collective fight against the disease and this requires all partners, affected countries or those at risk, national plant protection services and research institutes, researchers, international centers and organizations, and investors; to be actively engaged. There is nothing gained from isolationism and everything to be gained from working together, cooperating and sharing information. Furthermore, the risks posed on wheat production by the emerging rust races capable of spreading quickly over continents, need intensified international collaboration and coordination. Strengthening national capacities is particularly critical to enable countries develop and implement in an effective manner their contingency plans for prevention of rust outbreaks. The global wheat rust programm should be re-focusing on re-enforcing national capacities and international collaboration for improved prevention and rapid response. These national and international efforts need adequate resources to be effective. In Ethiopia, wheat rusts remain major threats for wheat production. Yellow rust is one of the major threats to wheat production because of the extreme level of damage the disease causes to susceptible varieties. In the highlands of Ethiopia, at altitudes ranging from 2150 to 2850 meters above sea level, yellow rust is an important disease of wheat production. Frequent and widespread yellow rust epidemics have been observed at higher elevations in the southeastern part of wheat growing areas of the country. This frequent and widespread epidemic was attributed to varietal susceptibility, production of mega cultivars, and expansion of wheat mono cropping, introduction of new virulent races and favorable environmental conditions for disease development. Reports indicate that yellow rust, during epidemics, can cause a huge damage to wheat crops. It can cause 100% yield loss if infection occurs very early and the disease continues to develop during the growing season on susceptible cultivars. In 2010, Ethiopia experienced one of the most serious yellow rust epidemics in recent times, with more than 600,000 ha of wheat affected and an estimated $US3.2 million spent on fungicides. Moreover, in Ethiopia, repeated epidemics of yellow rust were recorded in the last three decades. The first yellow rust epidemics occurred in 1977 on wheat variety 'Laketch' in state farms of Arsi and Bale. In 1988, another yellow rust epidemic occurred on the wheat variety; 'Dashen' which carried Yr9 gene in Arsi and Bale zones. In 2010, a devastating yellow rust epidemic affected widely grown 'Kubsa' and 'Galema' bread wheat varieties and the Yr27-virulent strain of *P. striiformis f.sp. tritici* was attributed to be a major cause of this epidemic. Recently, another new race was detected in Ethiopia in 2016, after being first detected in Afghanistan in 2012 and 2013 on resistance gene 'PstS11'. This race caused epidemics in Ethiopia in 2016 (Hovmoller *et al*., 2017; Tilahun, 2018). The rapid breakdown of resistant genes in newly developed varieties, require for continuous research on biology and management of the yellow rust disease

as well as the development of new resistant varieties. The continual development of cultivars with a combination of different types of resistance can reduce inoculum and prevent the pathogen population from increasing to epidemic levels in future. The rapid breakdown of resistant genes in newly developed varieties; also needs fast track variety release, accelerated seed multiplication, popularization and promotion of rust resistant wheat varieties, rust disease early warning system, wheat value chain development and creating awareness among policy makers and farmers about the threat of rusts in general and yellow rust in particular. The short longevity of rust resistance and the periodic outbreak of rust epidemics pose serious threat of food security for over 4.5 million small-scale farmers engaged in wheat production in the highlands of Ethiopia. This condition requires integrated wheat rust management by combining varietal resistance, cultural practices and chemical control. With respect to varietal resistance, developing wheat varieties with diversified yellow rust resistant genes and strengthening the global partnership especially with CGIAR and NARS from neighboring countries are important activities for the national research system, particularly for wheat breeding program of the country. The development of resistant varieties need to be coupled with adopting, fast-track variety release and accelerated seed multiplication in partnership with various stakeholders along the seed value chain to ensure fast replacement, adequate variety demonstration, popularization, and dissemination of resistant varieties to create awareness and demand for seed from farmers, enhance the adoption of resistant varieties through improved market linkages and value chain development activities, devising an effective rust surveillance system for early warning coupled with adequate preparedness for chemical control as a priority in case of rust out breaks. The other component of the integration is introducing legumes and wheat to break the monoculture and ensure sustainable diversification and intensification of the framing systems; and devising a strategy for expanding irrigated wheat production through development of varieties adapted to the lowland. Fungicides are also one of the components of integrated management of wheat stem rust. They can be a valuable tool in increasing yields and profitability of wheat production, especially if disease susceptible varieties are grown and, where the disease pressure is only moderate. But combining fungicides with host resistance offers the best monetary return when disease pressure is high. This should be linked to accurate disease forecasting and timely application of chemicals. On the other hand, to avoid fast breakdown of stem rust resistance genes in the wheat varieties, the breeding efforts of the country should focus on selecting for minor genes, based on adult plant resistance. This kind of resistance is especially important for countries like Ethiopia which are considered to be under high risk and where survival of the pathogen for several years is expected due to favorable environmental conditions. It takes many years to develop new stem rust resistant varieties in convention breeding methods. This requires the use of biotechnology to accelerate the breeding process and make available stem rust resistant wheat varieties in short period of time. It is evident that recent experiences demonstrated that the conventional approach of development and deployment of rust-resistant varieties alone would not address the impeding risk of wheat production in the country. Therefore, it is important to develop an integrated strategy to elevate the rust threats. Developing a capacity for rust surveillance, use of chemical control and diversification of wheat-based production system are some of the measures need to be taken in addition to rust resistant varieties.

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