

Wheat Leaf Rust Disease Management: A Review

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Abstract: Leaf rust is one of the three wheat rusts and is economically important disease of wheat worldwide. It is a devastating disease that can cause significant yield losses especially in wheat growing regions of the world where environmental conditions are conducive. In Ethiopia, wheat leaf rust is one of the most important diseases in most wheat growing areas of the country where the yield loss due to its impact is reaching up to 75% in susceptible wheat varieties at hot spot areas. This review discusses recent information on economic importance, epidemiology, geographic distribution, life cycle, host range of wheat leaf rust disease as well as its management methods such as cultural, chemical, biological and use of host resistance cultivars. Under the use of host resistance method, information on the types of resistance and sources of resistance has been presented.

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

1. INTRODUCTION

Wheat is one of the world's most important staple grains (Curtis *et al.*, 2002; FAO, 2014) and is the most widely grown cereal crop (Snape and Pánková, 2007) in more than 122 countries (FAOSTAT, 2015). It is the leading source of calories for millions of people worldwide (Curtis *et al.*, 2002; Hawkesford *et al.*, 2013). 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). However, wheat production is highly constrained by rusts that can cause significant yield losses (Marasas, 2004). The rust pathogens are among the most important pathogens causing a continuous threat to wheat production (Singh *et al.*, 2008) and have been reported to cause a vast amount of losses in different areas, years and environments favouring disease epidemics (Sharma *et al.*, 2011; Pardey *et al.*, 2013; Beddow *et al.*, 2015; Singh *et al.*, 2016). Wheat leaf rust, caused by *Puccinia triticina*, is one of the three wheat rusts and is economically important disease of wheat worldwide (Bolton *et al.*, 2008; Wang *et al.*, 2016). It is the most common and widely distributed of the three wheat rusts (Samborski, 1985; Roelfs *et al.*, 1992; Oelke and Kolmer, 2004; Mebrate *et al.*, 2008; Kolmer *et al.*, 2009; Huerta-Espino *et al.*, 2011) and therefore, has adapted to a wide range of environments, allowing it to coexist with wheat in all growing environments (Winzeler *et al.*, 2000; Kolmer *et al.*, 2009; Huerta-Espino *et al.*, 2011; McCallum *et al.*, 2016). Leaf rust is a devastating disease that can highly threaten wheat production and can cause significant yield losses especially in wheat growing regions of the world where environmental conditions are conducive (Roelfs *et al.*, 1992; Sayre *et al.*, 1998; Cook, Hilms & Vaughan, 1999; Marasas *et al.*, 2004; Carpenter, 2018). Historically, an epidemic that caused yield losses up to 40 to 50% has been recorded in different parts of the world (Roelfs *et al.*, 1992; Marasas *et al.*, 2004). Moreover, reports indicate that, early infection of leaf rust on wheat generally causes higher yield losses (Roelfs *et al.*, 1992; Huerta-Espino *et al.*, 2011). In Ethiopia, wheat leaf rust, is one of the most important diseases in most wheat growing areas of the country (Badebo *et al.*, 2008; Tekly, 2015; Gadisa, 2019; Habtamu *et al.*, 2020) where the yield loss due to its impact is reaching up to 75% in susceptible wheat varieties at hot spot areas (Badebo *et al.*, 2008; Shimelis and Pretorius, 2005; Tekly, 2015; Gadisa, 2019; Habtamu *et al.*, 2020).

2. ECONOMIC IMPORTANCE OF WHEAT LEAF RUST DISEASE

Leaf rust is a wheat disease potentially causing a huge yield loss in the world's wheat crop production (Eversmeyer & Kramer, 1996; Sayre *et al.*, 1998; Cook, Hilms & Vaughan, 1999; Singh, 1999) and is now recognized as an important pathogen, causing significant yield losses over large geographical areas (Marasas *et al.*, 2004; Roelfs *et al.*, 1992). The effect of leaf rust on plant development and wheat quality depends on the onset of the disease, yield potential and the level of cultivar resistance (Knott, 1989; Schultz & Line, 1992; Sayre *et al.*, 1998; Eversmeyer & Kramer 2000; Everts *et al.*, 2001; Singh *et al.*, 2002). Yield reductions are higher if the disease occurs early in the crop growth stage and occur on the flag leaf (Roelfs *et al.*, 1992; Kolmer *et al.*, 2009; Huerta-Espino *et al.*, 2011). Leaf rust causes greater annual losses due to its more frequent and widespread occurrence almost every year worldwide (Singh *et al.*, 2002). For example, in Australia, estimated crop losses of up to 30% were reported in wheat cultivars susceptible to leaf rust (Rees and Platz 1975) and up to 55% in cultivars susceptible to both stem rust and leaf rust (Keed and White 1971). In 1992, a widespread leaf rust epidemic in Western Australia caused yield losses of up to 37% in susceptible cultivars and an average loss of 15% across fields (McIntosh *et al.*, 1995). Murray and Brennan (2009) estimated the financial losses due to leaf rust in Australia were AUD 12 million per annum; however, has the potential to cause national losses up to AUD 197 million. Reports showed that there was an estimated loss of over 3 million tons, worth over \$350 million due to leaf rust from 2000 to 2004 in the USA. Similarly, estimated losses in Kansas winter wheat alone were estimated at 13.9% in 2007, 4.7% in 2008 and 1.37% in 2009 (Appel *et al.*, 2009). In the same way, in Mexico, wheat yield losses due to leaf rust were estimated at \$32 million between 2000 and 2003 growing seasons (Singh *et al.*, 2004a). Yield losses in the southern part of the state of Sonora in north-western Mexico during 2008–2009 growing season were estimated at \$40 million, including the cost of two fungicide applications to avoid serious yield losses. Likewise, economic assessments in the United Kingdom (Priestley and Bayles, 1988) provided estimates of losses in susceptible winter wheat due to stripe rust and leaf rust of £83 million with the value of disease resistance estimated at £79.8 million (Priestley and Bayles, 1988). Volkova and his co-workers (2009) in Russia showed that Annual yield losses have varied from 18 to 25% at different wheat growing regions of the country (Volkova *et al.*, 2009). In South America (Argentina, Brazil, Chile, Paraguay, and Uruguay) between 1996 and 2003, the yield loss amounted to \$172 million due to the most significant changes in the leaf rust pathogen population affected 10 cultivars and caused yield losses in the region (German *et al.*, 2004). The costs of annual fungicide applications from 1999 to 2003 were estimated at more than \$50 million. Leaf rust epidemics between 1999 and 2003 in Argentina alone, resulted in estimated costs of US\$74 million when three popular varieties became susceptible to leaf rust (German *et al.*, 2004). The total annual cost of fungicide applications to control leaf rust in the South Cone region of South America is about US\$50 million (German *et al.*, 2007). In Pakistan, in 1978, yield loss that cost a national financial loss of US\$ 86 million was reported (Hussein *et al.*, 1980). Similarly, in China, annual yield losses were estimated to be 3 million tonnes. Losses up to 30% has been reported in West Asia region and in Central Asia, more than 90% of the area is planted in leaf rust prone areas of 13.3 million hectares (Singh *et al.*, 2004b). In Egypt & Tunisia estimated yield losses up to 50% & 30%, respectively were reported (Deghais *et al.*, 1999). Likewise, rusts cost South African wheat farmers millions of rands annually (Boshoff, 2001). In Ethiopia, yield loss due to leaf rust reached up to 75% on susceptible wheat varieties at hot spot wheat growing areas of the country (Badebo *et al.*, 2008; Tekly, 2015; Hei *et al.*, 2016; Gadisa, 2019; Habtamu *et al.*, 2020).

3. EPIDEMIOLOGY OF WHEAT LEAF RUST DISEASE

Consecutive developmental stages have to occur before a healthy plant becomes diseased. These stages consist of the arrival of a pathogen to the host, attachment to the host, recognition between host and pathogen, spore germination, appressorium formation, penetration, infection, colonization, and dissemination, often by air and or water. In epidemiology, different factors that affect the disease cycle and especially the rates of the events are studied (de Vallavieille-Pope *et al.*, 2000). Leaf rust is heteroecious and macrocyclic fungus. It is heteroecious, because it requires two taxonomically unrelated hosts to complete its full life cycle. The primary host is wheat and the alternate hosts are *Thalictrum speciosissimum*, *Isopyrum fumarioides*, *Anchusa spp.* and *Clematis spp.* On the two different hosts, leaf rust fungus produces five spore stages: urediniospores, teliospores, basidiospores, pycniospores and aeciospores stages. The urediniospore, teliospore and basidiospore stages are produced on the primary host, wheat. The pycniospore and aeciospore stages are produced on the alternate hosts *Thalictrum speciosissimum*, *Isopyrum fumarioides*, *Anchusa spp.* and *Clematis spp.* (Samborski 1985). *Thalictrum speciosissimum* is considered to be the main alternate host with the other

hosts only occurring in specific regions (Samborski 1985). The fungus is a macrocyclic because it produces spores more than one time in the growing season of the crops. Each new spore is capable of reinfecting wheat and so new infections can occur every 7 to 10 days depending on the environment, host age and genotype (Bolton *et al.* 2008; Tomerlin *et al.*, 1983). Leaf rust produces round to ovoid pustules up to 1.5 mm in diameter on the adaxial and abaxial leaf surfaces (Bolton *et al.*, 2008). Approximately 20,000 spores can be produced per pustule when leaf rust infections occur at the heading growth stage through senescence (Tomerlin *et al.*, 1983). Once urediniospores land on susceptible host, the spore germinates to develop a germ tube. Optimal spore germination occurs with temperatures between 15 to 20°C with continuous dew for 4 to 8 hours (Roelfset *et al.*, 1992; Zhang *et al.*, 2003). After germination, the germ tube will move across the leaf surface to a stoma (Bolton *et al.*, 2008). When a stoma is encountered, the germ tube protoplasm will concentrate at the hyphal tip and form an appressorium (Bolton *et al.*, 2008). A septum will form between the germ tube and the newly formed appressorium (Bolton *et al.*, 2008). The stoma will close in response to the appressorium and a penetration peg will push through the closed stoma to enter the host substomatal space (Bolton *et al.*, 2008). The penetration peg hypha forms a substomatal vesicle. Infection hyphae and haustorial mother cell are developed from the substomatal vesicle when in contact with mesophyll cell (Bolton *et al.*, 2008). From the haustorium mother cell a penetration peg is developed to penetrate the host cell wall forming a haustorium (Bolton *et al.*, 2008). The haustorium invaginates the plant cell membrane leaving the membrane intact. A tight association between the host and fungal membranes allows the transport of nutrients from the host to the fungus (Baka *et al.*, 1995). The haustorium might also modify the metabolism of the host cell to the pathogen's own requirements (Szabo and Bushnell, 2001; Panstruga, 2003). Following infection, after 3–4 days, colour variation (i.e. faint flecks) is visible. After 8–10 days urediniospores are visible while after 12–14 days maximum sporulation is observed under favorable temperatures (Stubbs *et al.*, 1986; Kolmer *et al.*, 2009). The sexual cycle of wheat leaf rust consists of four different developmental stages, two of which infect the alternate host *Thalictrum spp.* If present, alternate host provides little initial inoculum at the beginning of the growing season in most wheat growing areas (Zhao *et al.*, 2016). In general, volunteer wheat, winter wheat and/or wind-dispersed spores transported from region to region are the prime source of inoculum each year (Roelfset *et al.*, 1992; Nagarajan & Singh, 1990). However, the alternate host is thought to play a role in the evolution of new races (Samborski, 1985) as is somatic hybridization (Park *et al.*, 1999) although the main source of variation in the pathogen is most likely mutations (Groth, 1984). The spores produced by the leaf rust fungus would be detached from the leaf for dispersal (Eversmeyer and Kramer, 2000; Barnes *et al.*, 2009; Li *et al.*, 2009). Teliospores are produced at the end of the growing season and help the pathogen to overwinter (Roelfs, 1992).

4. GEOGRAPHIC DISTRIBUTION OF WHEAT LEAF RUST DISEASE

Wheat leaf rust fungus can easily spread thousands of kilometres from the initial infection site through its spores, which have been documented to spread both within and between continents (Khan *et al.*, 2013). This widespread dispersal has led to the occurrence of epidemics on a continental scale (Roelfs, 1989). Therefore, it occurs most commonly and has the widest distribution (Kolmer *et al.*, 2009). Depending on the direction of wind spread of urediniospores in each cropping year, Huerta-Espino (1992) classified wheat growing areas of the world in to 9 epidemiological regions: Mexico; Canada and the USA; South Asia; West Asia, Eastern Europe and Egypt; Southern Africa; Northern Africa and Western Europe; the Far East; Southeast Asia; South America; and Australia–New Zealand. The Asian and African regions were delimited based on the expected airborne spread of urediniospores by wind during annual cycles. These regions were further divided based on diversity in the pathogen population (Kolmer 1992). Mountain barriers may account for the variation in geographic populations of *P. triticina* in Central Asia and the Caucasus (Kolmer and Ordon˜ez, 2007). Wheat leaf rust is distributed in all these wheat-growing regions of the world (Samborski, 1985; Roelfs *et al.*, 1992; Oelke and Kolmer, 2004; Mebrate *et al.*, 2008; Kolmer *et al.*, 2009; Huerta-Espino *et al.*, 2011). It occurs nearly every year in the United States, Canada and Mexico, causing serious losses in wheat production (Huerta-Espino *et al.*, 2011; Roelfs 1989; Singh *et al.*, 2004a). In South America, wheat leaf rust causes major yield losses in Argentina, Bolivia, Brazil, Chile, Paraguay, and Uruguay (German *et al.*, 2004). In East and South Asia, high risk regions for wheat leaf rust include China, India, Pakistan, Bangladesh, and Nepal, while in Central Asia most of the wheat crop is planted in areas prone to the disease (Singh *et al.*, 2004b). In Russia, leaf rust results in yield losses for both winter and spring wheat (Huerta-Espino *et al.*, 2011). In North Africa, leaf rust causes severe yield losses in Egypt and Tunisia (Huerta-Espino *et al.*, 2011). In South Africa, leaf rust epidemics frequently occur on the spring wheat in Western Cape, winter wheat in Orange Free State and irrigated wheat in other provinces (Pretorius *et al.*, 1987). In Australia, leaf rust is widely dispersed, occurring in all wheat-growing regions (Murray and Brennan, 2009).

5. HOST RANGE OF WHEAT LEAF RUST PATHOGEN

The primary hosts of *P. triticina* include bread wheat, durum wheat (*T. turgidum* L. var. *durum*), cultivated emmer wheat (*T. dicoccum*), wild emmer wheat (*T. dicoccoides*), *Ae. speltoides*, goatgrass (*Ae. cylindrical*), and triticale (*X Triticum secale*) (Roelfs 1992; Bolton *et al.*, 2008). *P. triticina* requires an alternate host to complete the sexual stages of its life cycle, which helps in the evolution of new races or pathotypes through genetic recombination.

6. LIFE CYCLE OF WHEAT LEAF RUST PATHOGEN

Leaf rust pathogen is a macrocyclic and heteroecious fungal species that has five distinct spore stages (Bolton, *et al.*, 2008; Kolmer, 2013; Zhao *et al.*, 2016) involving genetically distant different host species. Under suitable environmental conditions, the wheat plant produces dark brown, two-celled teliospores (Bolton *et al.*, 2008). The teliospores germinate to yield haploid basidiospores on the wheat leaf tissue. Then the basidiospores are moved by wind and infect the alternate host to develop haploid pycnia in the pycnial structures (Bolton *et al.*, 2008). Consequently, the pycniospores are carried by insects to other pycnial infections whereby sexual propagation between two genetically dissimilar cells takes place, which results in the formation of plasmogamy (Kolmer, 2013). Finally, the aecial cups release aeciospores that will be broadcasted by wind to infect wheat. The sexual stage of *P. triticina* does not contribute significantly to the pathogen virulence diversity (Bolton *et al.*, 2008; Kolmer, 2013). Therefore, *P. triticina* is thought to reproduce asexually through production of dikaryotic urediniospores, cycling on wheat and its relatives (Kolmer, 1996). Temperatures of 10–25°C and presence of adequate moisture on the leaf surface are conducive for infections (Anikster, 1986). Depending on prevailing environmental conditions, the uredinial cycle is repeated every 8 to 14 days. The life cycle is complete when aeciospores germinate and penetrate stomata of the telial/uredinial host, resulting in production of asexual urediniospores.

7. MANAGEMENT OF WHEAT LEAF RUST DISEASE

Management of cereal 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 (Singh, 2005). Therefore, a combination of different methods should be used to manage cereal rusts in wheat production. Similarly, wheat leaf rust disease can be controlled using a combination of different methods such as cultural, chemical, biological and genetic resistance, though using genetic resistance is efficient, cost-effective and environmental friendly to control wheat leaf rust disease (Chen *et al.*, 2005).

7.1. CULTURAL PRACTICE

Leaf rust disease can be managed using cultural practices (Knott, 1988; Ellis *et al.*, 2014). 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 leaf rust (Roelfs, 1992; Wan *et al.*, 2007). The date of disease onset is directly related to the development of an epidemic (Roelfs, 1985a). Appropriate use of fertilizers and plant growth regulators helps to control wheat leaf rust disease. (Knott, 1989; Fixen, 1993; Olesen *et al.*, 2003a; Neumann *et al.*, 2004). Likewise, controlling the timing, frequency, and amount of irrigation can help decrease leaf rust infections (Roelfs, 1984). Reports indicate that using early maturing cultivars allows the plants to mature before leaf rust infections can become serious (Knott, 1988). Using multi-lines or varietal mixtures are also recommended (Huang *et al.*, 2012; Kumar, 2012). 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). The destruction of all volunteer plants would significantly reduce the survival of primary inoculum and reduce the losses due to severe epidemics (Roelfs *et al.*, 1992; Eversmeyer & Kramer 2000; Hollaway, 2007; Kolmer *et al.*, 2007; Hollaway, 2014). Heavy grazing, cultivating or spraying with herbicides to remove the 'green bridge' will reduce the amount of leaf rust in following crops (Roelfs, 1992; Hollaway and Brown, 2005).

7.2. CHEMICAL CONTROL

Chemical means of controlling wheat rusts becomes quite important as a second line of defence (Samborski 1985; Loughman *et al.*, 2005). Therefore, management of the disease through the application of chemical fungicides is necessary to ensure that wheat cultivation remains a profitable endeavour (German *et al.*, 2007). The most important aspect of fungicide application is to protect the flag leaf from infection until after the kernels have filled. Control of leaf

rust by chemical products is available with new and more effective fungicides (DeWolf, 2014) such as: Picoxystrobin, Fluoxastrobin, Pyraclostrobin, Propiconazole, Prothioconazole, Tebuconazole, Prothioconazole, Metconazole + Pyraclostrobin, Propiconazole + Azoxystrobin, Fluxapyroxad + Pyraclostrobin, Propiconazole + Azoxystrobin, Prothioconazole + Trifloxystrobin, Tebuconazole + Trifloxystrobin and Cyproconazole + Picoxystrobin

Many fungicides have been evaluated, verified, and registered for wheat disease in Ethiopia, among them, Tilt, Bumper, Progress, Topazol, Bayleton, Prevent, Orius, Soprano, Natura, Amistar Extra and Rexduo are commonly used for wheat rusts (Habtamu, 2018).

7.3. BIOLOGICAL CONTROL

The bacterial strain, *Pseudomonas putida* has the capability to produce several types of antibiotics, siderophores and slight quantity of hydrogen cyanide (HCN), which suppress the *Puccinia triticina* growth in vitro and vivo. Different biocontrol agents were evaluated against leaf rust of wheat with their combined effect and lonely. The bioagents like *V. lecanii*, *B. bassiana*, *P. fumosoroseus*, *M. anisopliae* and *C. cladosporioides* were used against *Puccinia recondita* in the field and lab conditions. The results showed that *B. bassiana* and *V. lecanii* while *P. fumosoroseus* and *V. lecanii* showed the best results against the suppression of pustule size.

7.4. PATHOGEN MONITORING

Pathogen monitoring allows the early detection of new races and confirms the prevalence of major existing races and the information obtained from it informs policies, research and development investments, as well as crop protection and breeding approaches (Park *et al.*, 2011). Coordinated international surveillance programmes are crucial to guide management strategies (Park *et al.*, 2011). The global cereal rust monitoring system created in response to the emergence of Ug99 was a good example and will serve as a model to monitor other important pathogens (Hodson *et al.*, 2009; Park *et al.*, 2011). This system has provided information and strategies in rust pathogen research and plant breeding.

7.5. HOST PLANT RESISTANCE

Deployment of genetic resistance is the most effective, environmentally friendly, cost-effective and long-term strategy to control leaf rust disease for reducing yields losses (Roelfs *et al.*, 1992; Chen, 2005). It is also the best strategy particularly for resource poor farmers in the developing world (McIntosh *et al.*, 2009). The genetic basis of resistance to wheat rust was described, for the first time, by Biffen in the early 1900s' (Biffen 1905). Since then, the discovery of genetic variation for rust resistance has been an ongoing component of wheat breeding programs.

7.5.1 types of host resistance

Studies of the interaction between cereal rusts and their hosts show a very close relationship between the genetics of the pathogen and of the host in the expression of disease (Stubbs *et al.*, 1986). Host-pathogen interaction could be specific or non-specific (Roelfs *et al.*, 1992). Based on this, plant disease resistance can be classified into two major categories: race-specific and race-non-specific resistance (Stubbs *et al.*, 1986).

7.5.1.1 race-specific resistance

Race-specific resistance is conferred by a single resistance gene and is also termed as qualitative, vertical, seedling, all-stage, monogenic (major genes), and hypersensitive, etc., resistance (Stubbs, *et al.*, 1986; Rolfs *et al.*, 1992). It is effective against only particular races of a pathogen and often "breaks down" easily with the occurrence of new pathotypes of a pathogen, often led by a boom and bust cycle (Dyck and Kerber, 1985; Priyamvada and Tiwari, 2011; McDonald and Linde 2002; Knott 2008; Chen and Moore, 2002; Daetwyler *et al.*, 2014). It is effective at the seedling stage and remains effective at adult stage (Lagudah, 2010). However, there are a few race-specific resistance genes that express their resistance genes at adult plant stage, for example Lr12, Lr13 and Lr22a (Dyck and Kerber, 1985). Most Lr genes are classified in this category (Samsapour *et al.*, 2010; Bariana and Bansal, 2017; McIntosh *et al.*, 1995, 2017).

7.5.1.2. race non-specific resistance

Race non-specific resistance is conferred by multiple genes or quantitative trait loci (QTLs) and is also termed as quantitative, or horizontal, adult plant, slow rusting, polygenic (minor gene), durable etc. resistance with each providing a partial increase in resistance (Flor, 1956; McIntosh, 1995; Singh *et al.*, 2000; Chen, 2005). It is effective at adult plant

stages and is often detected as field resistance (Hovmøller *et al.*, 2011). It is also effective against a broad range of races & is an important source for durable resistance (Sallam *et al.*, 2016). Therefore, 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). Among the Lr genes catalogued, only 14 confer race non-specific resistance (McIntosh *et al.*, 2017). These include Lr12, Lr13, Lr22 (alleles a, and b), Lr34, Lr35, Lr37, Lr46, Lr48, Lr49, Lr67, Lr68, Lr75, and Lr77 (Lin and Chen 2009; McIntosh *et al.*, 2017). Some of them provide partial resistance, for example, Lr34. However, there are exceptions, where some race non-specific resistance genes provide race-specific resistance (i.e. Lr13) or confer a hypersensitive response (i.e. Lr48) (Bansal *et al.*, 2008). Only a few wheat leaf rust race non-specific resistance genes confer partial resistance against multiple pathogens, and these include Lr34, Lr46, and Lr67 (Schnurbusch *et al.*, 2004).

7.5.1.3. durable leaf rust resistance

Resistance is considered durable if it remains effective within a cultivar under cultivation for a significant number of years over a substantial area with favourable conditions for the respective pathogen (Johnson, 1984). Durability of leaf rust resistance is often linked to genes or gene loci that confer durable resistance to other rusts and diseases as in the case of Lr34/Yr18/Sr57 (Singh, 1992), Lr46/Yr29/Pm39 (Kolmer *et al.*, 2015b), Lr67/Yr46 (Herrera-Fossel *et al.*, 2014) and probably Lr68 (Herrera-Fossel *et al.*, 2011), Lr74 (Kolmer *et al.*, 2018b), Lr75 (Singla *et al.*, 2017), Lr77 (Kolmer *et al.*, 2018c), Lr78 (Kolmer *et al.*, 2018a). When race-specific and race non-specific resistance genes successfully combined, resistance genes often complement each other, giving reactions different from those given individually (Kloppers and Pretorius 1997). Kloppers and Pretorius (1997) observed active complementation of Lr34 (race-nonspecific) and Lr13 (race-specific) with both genes improving resistance on the selected lines even with the presence of races possessing virulence for the Lr13 gene. German and Kolmer (1992) and Kolmer *et al.*, (2007) also reported prolonged and positive interaction of resistance genes Lr16, Lr23, and Lr34 in North America. Similarly, Vanzetti *et al.*, 2011 found that race-specific resistance genes such as Lr16, Lr47, Lr19, Lr41, Lr21, Lr25, and Lr29 provided durable resistance when combined with race-nonspecific resistance genes such as Lr34, Sr2, and Lr46 in Argentina. In the same way, Silva *et al.*, (2015) combining Lr34, Lr68, and Sr2 to increase leaf rust resistance. Combinations of partial resistance genes in the CIMMYT breeding program have conferred adequate resistance to leaf rust in the field for several years (Singh *et al.*, 2005). Therefore, race-nonspecific resistance genes, such as Lr34 and Lr46, are very important for breeding (Schnurbusch *et al.*, 2004). Moreover, combinations of two or more adult plant resistance genes can provide commercially acceptable or near immune levels of resistance and this type of resistance is assumed to be durable (Bariana and McIntosh, 1995; Singh *et al.*, 2000; Singh *et al.*, 2004; Singh & Rajaram, 1992). Varieties with Lr34 and two or four additional genes, referred to as ‘near immune’ show a stable response in all environments tested so far, with final leaf rust ratings lower than 10% (Singh *et al.*, 2000). The second slow-rusting resistance gene Lr46 was identified in wheat cultivar ‘Pavon 76’ (William *et al.*, 2003). Recent studies indicate that at least 10-12 slow rusting genes are present in CIMMYT spring wheat germplasm.

7.5.2. sources of host resistance

Because of the evolution of new races, identification and transfer of new sources of resistance genes is necessary. New sources of resistance genes can be obtained from various sources. Leaf rust resistance genes were initially characterized in wheat *T. aestivum* (Lr1, Lr2a, Lr3, Lr10, Lr11), and later in wheat related species such as *T. tauschii* (Lr21), *Aegilops elongatum* (Lr24), *A. umbellulata* (Lr9), and common rye, *Secale cereale* (Lr26) (Browder, 1980). To date, approximately 80 leaf rust resistance genes have been identified and characterized in bread wheat, durum wheat and diploid wheat species (McIntosh *et al.*, 1995; 2007; 2017). Leaf rust resistance genes, including Lr1 (Roelfs *et al.*, 2000), Lr2, Lr2a to Lr2c, Lr3 to Lr6, Lr8, (McIntosh *et al.*, 2003) Lr7 (Wisniewska *et al.*, 2003), Lr10 to Lr13, Lr14a, Lr15 to Lr18 (McIntosh *et al.*, 2003), Lr20 (McIntosh *et al.*, 2003; Neu *et al.*, 2002), Lr22b, Lr23, Lr27, Lr30 (Nelson *et al.*, 1997), Lr31, Lr33, Lr40, (McIntosh *et al.*, 2003) Lr46, (Wisniewska *et al.*, 2003) Lr48 (McIntosh *et al.*, 2003), Lr49, Lr52, Lr67, Lr68, (Shahin and El-Orabey, 2015) trp1, trp29 (Da-Silva *et al.*, 2012), Lrac104, and Lrac124 (Hussein *et al.*, 2005), have been derived directly from common wheat (*Triticum aestivum*) cultivars. Likewise, genes Lr14a (on 7BL) and Lr23 (on 2BS) were transferred to common wheat from the durum wheat cultivar ‘Hope’ and the related line ‘Gaza’, respectively. Similarly, wild cultivars of wheat or wild grasses can be major sources of resistance genes to leaf rust and the first introduction of leaf rust resistance genes into common wheat cultivars was from these wild accessions (Gill *et al.*, 1986). One gene (Lr9) was derived from the wild species *Aegilops umbellulata*, three (Lr19, Lr24, and Lr29) from *Aegilops*

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elongatum, five (Lr28, Lr35, Lr36, Lr47, and Lr51) from *Aegilops speltoides*, one (Lr37) from *Aegilops ventricosa*, one (Lr37) from *Agropyronintermedium*, seven (Lr21, Lr22a, Lr32, Lr39, Lr41, Lr42, and Lr43) from *Triticum tauschii* (Cox *et al.*, 1994; Hussein *et al.*, 1997), one (Lr44) from *Triticum spelta*, three (Lr25, Lr26, and Lr45) from *Secale cereale* (McIntosh *et al.*, 2003), one (Lr50) from *Triticummonococcum* (Mago *et al.*, 2002), one (Lr33) from *Triticum dicoccoides* (B»aszcyket *et al.*, 2004) and one from *Aegilops kotschyi*. To date, more than half of the available Lr genes have been identified from wild relatives (McIntosh *et al.*, 2017). These include Lr9 (*Ae. umbellulata*); Lr19, Lr24, and Lr29 (*Thinopyrum ponticum*); Lr37 (*Ae. ventricosa*); Lr38 (*Th. intermedium*); Lr28, Lr35, Lr36, Lr51, and Lr66 (*Ae. speltoides*); Lr21, Lr22a, Lr32, and Lr39 (*Ae. tauschii*); Lr57 (*Ae. geniculata*); Lr58 (*Ae. triuncialis*); Lr59 (*Ae. peregrina*); Lr62 (*Ae. neglecta*); Lr63 (*T. monococcum*), and Lr53, Lr64 (*T. dicoccoides*); Lr14a and Lr61 (*T. turgidum*) (McIntosh *et al.*, 2013). In the same way, because few landraces have been used in modern plant breeding, wheat landraces are important potential source of new resistance genes (Reif *et al.*, 2005). This might be due to the co-existence of rust pathogens and wheat may have resulted in the accumulation of diverse resistance in wheat (Newton *et al.*, 2010; Bux *et al.*, 2012).

8. CONCLUSION

Leaf rust is a devastating disease globally and growing resistant cultivars is the major component to control it. However, the fast “breakdown” of resistance is a major problem. Deployment of resistant crop varieties require: knowledge 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 leaf rust. Therefore, a combination of different strategies should be used to manage rusts in wheat production. 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 centres and organizations, and investors; to be actively engaged. In Ethiopia, wheat rusts are major threats for wheat production causing frequent and widespread epidemic largely due to fast breakdown of resistant cultivars. This problem can be elevated by continual development of cultivars with a combination of different types of resistance as well as integrating cultural practices and chemical control with varietal resistance. 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 in Ethiopia.

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