Agriculture

Barley Germplasm Improvement and Screening for Scald Disease Resistance Review of Breeding Experiences in Ethiopia

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Abstract

Scald, caused by the fungus *Rhynchosporium secalis*, is one of the most destructive diseases affecting barley (*Hordeum vulgare L.*) across the world. This pathogen, an asexual haploid ascomycete, thrives particularly in the Ethiopian highlands above 2000 meters, where low temperatures and high rainfall during the growing season create favorable conditions for infection. Yield losses attributed to scald range from 21% to 67%, often accompanied by a decline in grain quality depending on environmental conditions and varietal susceptibility. Efforts to screen and identify resistant barley genotypes are currently constrained by limited information on the genetic and pathogenic variability of *R. secalis* globally and within Ethiopia. Enhancing host resistance remains the most effective and sustainable strategy for managing scald disease and improving barley productivity.

Keywords: *Rhynchosporium secalis,* barley, disease resistance, breeding, Ethiopia

Introduction

Barley is the fourth most important cereal crop grown in the world produced after wheat, maize and rice with area under production of 51.41 million hectares and production of 159.88 million tons (FAO, 2021) et al [13]. In Ethiopia barley is the fifth most important cereal crop in area coverage and production and fifth in yield ton ha-1, with around 0.93 million ha, 2.30 million ton and 1.97 ton ha-1 respectively (CSA, 2021) [10].

The factors constraining the production of barley in the different barley production systems have been includes both biotic and abiotic stress. The most important biotic stresses include diseases and insect pests like, scald, net blotch, spot blotch, rusts, shoot fly and aphid (Bayeh M. and Berhane L., 2011) [1]. There are around 23 fungi, two bacteria, two viruses, and nine nematodes infect barley (Yitbarek *et al.*, 1996) [57]. Scald (*Rhynchosporium secalis*), blotches (*Helminthosporium* spp.), rusts (*Puccinia* spp.) and powdery mildew (*Erysiphae graminis*) are among the most widely distributed foliar diseases in barley producing areas of Ethiopia (Eshetu, 1985) [12].

The pathogen *Rhynchosporium secalis* is the causal agent of scald, which is a leaf disease of barley (*Hordeum vulgare* L.) was first reported in Ethiopia by Stewart and Dagnachew (1967) [43]. The disease is the most severe in the highlands (above 2000 m) where precipitation is high and temperature is low during the cropping season. Reported losses in yield due to scald vary between 21-67% and reduced grain quality depending on season and cultivar. Screening and selection of barley genotypes for resistance to the disease is currently hampered by the dearth of knowledge on the variability of the pathogen in Ethiopia (Kiros *et al*, 2004) [27]. The largest problem in barley selection in high pathogen variability causing resistant cultivars to rapidly become susceptible. Therefore, the selection process is considered completely successful if the production of barley cultivar is maintained for 5-7 years (Milomirka *et al*, 2012) [34]. Scald-resistant barley lines were also high-yielding across locations in Ethiopia, whereas other scald-resistant lines were low- yielding. This suggests that interactions between foliar diseases of barley may have a considerable influence on the field performance of scald-resistant cultivars (Yitbarek, 1990) [56].

Disease resistance has been the prime interest of barley breeding programs world-wide for Ethiopian germplasm. In addition to phenotypic diversity, Ethiopian barley is important source of resistance genes for scald (*Rhynchosporium secalis* (Oud.) (Demissie, 2006). Frequent selection of Ethiopian accessions in international evaluation work might lead to the erroneous conclusion that Ethiopian barleys are in general disease resistant (Harlan,1976) [21]. But diseases are a major yield limiting factor in the Ethiopian barley production and improving disease resistance in Ethiopian genotype is one of the primary objectives of the national breeding program (Gebre *et al*. 1996) [16]. Saying to this objective this review paper is to identify causative agent, diversity and yield losses of scald on barley and to describe barley improvement strategies for scald (*Rhynchosporium secalis)* diseases.

Biology of causative agent of scald

Scald (*Rhynchosporium secalis*) disease is the most destructive pathogens of barley in worldwide. It is caused by the haploid imperfect fungi (ascomycete) *Rhynchosporium secalis* (Oudem.) J. J. Davis, *i.e*. without known sexual stages since no teleomorph has been described for the fungus. It is most prevalent in temperate area where the relative temperature is low combined with humid weather condition as well as in tropical areas where there is high rainfall and temperatures are low because of the altitude difference (Gilchrist-Saavedra and McNab, 2006).

Barley leaf scald is a polycyclic disease, normally involving several pathogen generations during the growing season, and secondary disease spread by splash-dispersed conidia (Zhan *et*

al., 2008) [59]. The pathogen causes lesions that initially appear as spots and short yellow streaks on leaves, and the lesions can expand into longer longitudinal and transverse necrotic streaks on susceptible genotypes (Mathre, 1997) [31]. The development of *Rhynchosporium secalis* on the host plant is taking place predominantly in the subcuticular area of the infected leaf. After penetration of the cuticle, the hyphae grow extracellularly above the epidermal cells throughout most of the fungus life cycle. However, epidermal cells and later the mesophyllcells collapse leading to the typical symptoms of gray and water-soaked lesions at about 8-12 days after infection. Only in the late stages of the pathogenesis the mesophyll tissue is penetrated by the fungus (Xi *et al*., 2000) [52].

Barley, rye and other grass species are the main hosts of the pathogen and so the pathogen can cause significant yield losses during cool and wet condition (Mathre, 1997) [31]. The fungus persists on dead leaves and other plant residues to initiate primary infection. Seed borne spores may contribute to initial infections (Bockelman, *et al*., 1981). However, left over residues from previous year crops are considered the most important source of primary inoculums. Spore production is abundant during moist period and secondary spread of the inoculums takes place via wind or splashing rain. The disease may develop rapidly during cool weather and in severe cases may virtually cause defoliation by coalescing of the lesions (Yitbarek, *et al*., 1998) [58]. Sporulating potential of fungal material on crop residues left in the field could survive for up to a year. Overwintering mycelia will produce spores when environmental conditions are favorable, serving as primary inoculums to initiate an epidemic (Shipton, *et al*., 1974) [41].

Genetic diversity of scald (*Rhynchosporium secalis)*

Research showed that there is a high variability between *Rhynchosporium secalis* isolates of a population regarding pathogenicity, sporulation rate, colony morphology and color, conidial dimensions, response to nutritional conditions, and fungicide sensitivity. The high variability of the *R.secalis* pathotypes causes breakdown of single resistance gene in the field making breeding to scald resistance as difficult task (Zhan *et al*., 2008) [59].

Screening and selection of barley genotypes for resistance to disease is currently hamperd by dearth of knowledge on variability of pathogen in the world as well as in Ethiopia (Yitbarek, 1990) [56]. The pathogen apparently possesses limited mechanism for generation of variability but morphological and phatogenical characterization as well as population genetic analysis using molocular marker have reveal high genetic diversity within phatogen (Habgood, 1973) [19]. Genetic diversity has been found to be high within a small spatial scale (Mc Donald *et al,*1999) and up to 74 % of genetic variability was distributed within collection area of approximately $1m^2$ (Salamati *et al*, 2000) [39]. The source of high-level genetic diversity was not well known. Although asexual recombination(Newton, 1989) spontanouse mutation and sexual reproduction (Salamati *et al*, 2000) [39] has been proposed as possible mechanism responsible for high diversity of pathogen (Kiros *et al*, 2004) [27]. The variulance structure of *Rhynchosporium secalis* population may change over relatively short period of time (Jackson *et al,*1978) [25] and major risistance gene diploid in barley to control scald have frequently exhibit a fine life span due to break down of risistance associated with selection for increase virulence in the phatogen. pathogenic variation of *Rhynchosporium secalis* present risk to the use of single gene resistance in barley cultivar. It is there for important to identify and develop line carrying as many different gene for resistance as possible in order to provide stable resistance against abroad spectrum of fungal pathogen (Kiros *et al*, 2004) [27].

Yield loss assessments of scald on barley

Research reports revealed that in Ethiopia scald is considered among the most important biotic stresses in barley causing high yield loss in Ethiopia (Bekele *et al*., 2011) [2]. In the high lands where precipitation is high and temperature is low during the cropping period. Scald causes a yield loss of 67% on susceptible cultivar in Ethiopia (Yitbarek *et al*., 1998) [58]. The disease affects the foliage of barley and severely reduces its photosynthetic capacity, resulting in yield losses both in food and malt barley and especially on malt barley it reduce starch accumulation in the kernel, which result poor malt quality (Horsley and Hochhalter, 2004) [22].

The research conducted between in late 1980s and early 1990s showed that the incidence and severity of scald varied considerably between seasons in central region while not varied considerably between locations. The relationship between environmental factors and the incidence and severity of scald disease was influenced by topography and growth stage of the plant (Yitbarek *et al*., 1996) [57]. Planting dates has showed influence on scald incidence. For example at Holeta, yield loss of 31% to 43% were recorded on cultivars when planted until mid June while minimum losses occurred on cultivars planted at end of June due to scald (Getaneh, *et al*, 1996) [57]. In South eastern Ethiopia areas an incidence of 100% and severity of about 80% was recorded for scald both during *Bona* and *Ganna* at *Sinana* and *Dinsho* areas as well as at *Goba* and *Adaba* during *bona* growing seasons. In central and northeast of Ethiopia similarly 100% incidence and about 53% scald severity was recorded on barley in both *Belg* and *Meher* seasons. It was observed that severity was increased progressively starting from the tillering stage in the presence of high moisture level. On the other hand, the investigation of the scald occurrence and severity in western Ethiopia areas showed an incidence and severity of 19% and 5%, respectively, during the *meher* season which is lower than the other areas (Bekele *et al*., 2011) [2]. Whereas worldwide experiences about scald showed that under severe epidemics 100% losses in susceptible cultivars have been reported (Yahyaoui, 2004) [54].

Response of barley to *Rhynchosporium secalis*

Barley is attacked by a large number of fungal pathogens to most of which it responds as a resistant non-host and host resistant. In most cases the leaf epidermis is the first tissue to be penetrated by mostly asexual spores and this commonality puts forward barley responses in the epidermis as outstandingly important for the success or failure of the individual fungal attacks (Patrick, 2014) [36]. Fungal pathogens of barley can be placed along a gradient of different life styles ranging from obligate biotrophic (B. graminis and Puccinia sp.) over hemibiotrophic (B. sorokiniana, P. Teres, M. Oryzae) to necrotrophic (R. Commune and Fusarium sp.). Obligate biotrophic pathogens can only exist on living host tissue and

are therefore entirely dependent on constant support by the host plant. By contrast, necrotrophic pathogens secrete toxins and shrive on dying or dead plant material. Lastly, hemibiotrophic pathogens start softly by leaving host cells alive and switch usually 1–3 days after initial infection to the more brute-force approach by killing invaded host tissue via toxins or removal of cell death suppressors (effectors) thereby provoding host cell suicide as a co-opted defence reaction (Horbach *et al*. 2011) [23]. Barley responds to these fungal pathogens with altered gene expression often leading to the accumulation of pathogenesis-related (PR) proteins, with cell-wall appositions and sometimes with local cell death responses known as hypersensitive response (HR) (Liu *et al*. 2011) [30]. Ultimately, the presence or absence of strong resistance genes and the different in efficiencies of host factors are important to limit fungal infection and different efficiencies of co-opting host susceptibility factors are also used to determine the severity of an infection (Collinge *et al*. 2010) [9].

Barley possesses a number of major R-genes against R. *secalis*. The NIP1 toxic peptide has been found to be recognized as AvrRrs1 by the Rrs1 resistance protein in barley, which resulted in a more pronounced accumulation of some transcripts encoding PR proteins (Rohe *et al*. 1995) [38]. Transcripts of some PR protein genes analysed on northern blots accumulated either in leaf epidermis or mesophyll, suggesting that some infection- or defence-related signals also reach the inner leaf before epidermal collapse (Steiner-Lange *et al*. 2003) [42].

Resistance mechanisms of barley

Plants defend themselves against pathogens by a combination of weapons from two arsenals:

(1) structural characteristics that act as physical barriers and inhibit the pathogen from gaining entrance and spreading through the plant and (2) biochemical reactions that take place in the cells and tissues of the plant and produce substances that are either toxic to the pathogen or create conditions that inhibit growth of the pathogen in the plant. The combinations of structural characteristics and biochemical reactions employed in the defense of plants are different in different host–pathogen systems. In addition, even within the same host and pathogen, the combinations vary with the age of the plant, the kind of plant organ and tissue attacked, the nutritional condition of the plant, and the weather conditions (Agrios, 2005).

Barley lines up with many other plant species in terms of the current co-evolutionary model of plant innate immunity (Jones and Dangl, 2006) [26]. PAMP-triggered immunity (PTI) is the basis for strong and durable resistance against most non-adapted pathogens that have not co-evolved with a specific plant species such as barley. A few co-evolving host pathogens managed to suppress the critical components of PTI by secreted effector molecules thus establishing what is also known as “basic compatibility”. Effector mediated defence suppression is not complete and varies depending on the allelic status of host genes underlying the many resistance QTL that have been identified. QTL-mediated resistance was found to act against many pathogen races (Patrick, 2014) [36]. The kind of defense or resistance a host plant employs against a pathogen or against an abiotic agent, it is ultimately controlled, directly or indirectly, by the genetic material (genes) of the host plant and of the pathogen (Agrios, 2005).

During each *R. Secalis* generation, ‘gene-for-gene’ interactions occur, directly or indirectly, between barley major resistance genes (Bjørnstad *et al*., 2002) [4] and the corresponding avirulence effectors (the products of *R. Secalis* ‘avirulence’ genes) in incompatible interactions, to result in a resistant phenotype. In compatible interactions, the ‘virulence’ gene products or effectors, which include toxins such as NIP1(Hahn *et al*., 1993) [20], interact with specific host targets to result in a susceptible phenotype. Since there are a number of resistance genes in barley and corresponding genes in *R. Secalis*, a barley cultivar may possess several resistance genes, and *R. Secalis* has many races or pathotypes (Xi *et al*., 2003) [53] with different combinations of avirulent/virulent alleles. Thus, major-gene-mediated resistance may be referred to as race-specific resistance (Lehnackers & Knogge, 1990) [29], i.e. it involves interactions which have specificity in molecular recognition events. Such major-gene-mediated resistance can be identified in seedling tests with specific barley cultivars and *R. Secalis* isolates (Xi *et al*., 2003) [53]. Ideally, near-isogenic barley lines with/without a specific R allele and near-isogenic isolates of *R. Secalis* with avirulent/virulent alleles should be used (Lehnackers & Knogge, 1990) [29].

1. Host resistance mechanism

The impact of scald can be minimized through increasing host resistance which is by far the most important defense mechanism that can be used to control diseases in crops (Thakur, 2007) [46] and improve yield in quality and quantity. Thus development of barley cultivars with durable resistance to *Rhynchosporium secalis* is one of objectives of barley breeding. Ethiopian barley landraces are important sources of resistance genes for many barley diseases including scald (IBC, 2008) [24] but not adequately utilized in development of resistance or tolerance to scald disease in barley. The information on the type and magnitude of gene actions governing resistance genes and indirect selection of desirable parents *via* combining ability test would contribute in development of disease resistant cultivars. Some research showed that resistance genes to *R. secalis* in barley is governed by both ‘major’ and smaller ‘minor’ genes, generally additive effects (partial resistance) (Zhan *et al*., 2008) [59].

This type of resistance is inherited as a single Mendelian trait and thus is easy to handle in breeding practice. Its durability often is very limited thereby requiring a constant pipeline of novel R-genes in germplasm as additional burden to competitive breeding (Brown *et al*. 1993) [5]. However, durable, monogenic resistances acting in a race-specific manner against fungi do exist implying that a priori pessimism with regard to the usefulness of race specific R-genes may not be appropriate (Jorgensen, 1994). Linkage of scald resistance in several BC3-lines from H. spontaneum spp. spontaneum to the isozyme locus Acp2. The chromosomal position of the resistance gene designated Rrs12 on 4H was inferred from its linkage with isozyme (Garvin *et al*. 1997) [14].

2. Disease escape

‘Disease escape’ associated with cultivar height, maturity or canopy structure, which limits the upward spread of splash-dispersed *R. secalis*conidia (ACN, unpublished data). Early stem elongation, for example, could decrease spread of late epidemics. Terms such as field resistance or adult plant

resistance, normally used to describe resistance assessed in field plot.

experiments, may include components of both genetic (major-gene-mediated or partial) resistance and disease escape (J. Zhan *et al*, 2008) [59].

Improving durable barley resistance

Genetic resistance is a cost effective and sound approach to disease control. However, disease resistance genes are often found in unadapted germplasm. Transfer of these genes to adapted germplasm can be a laborious proposition, particularly when they show quantitative inheritance (Salvaraj *et al*., 2011) [40]. There are three principal ways to improve durable resistance of barley to major fungal pathogens: (1) stacking of carefully selected major R-genes by breeding, (2) marker-assisted introgression of multiple QTL by breeding and (3) generation of transgenic events introducing novel resistance or defence genes derived from barley, wild Hordeum relatives or other plant species; or silencing of susceptibility factors (J. Zhan *et al*,2008) [59].

Breeding

Ethiopian barley landraces are important sources of resistance genes for many barley diseases like leaf rust, net blotch, septoria, scald, spot blotch, barley stripe mosaic virus (IBC, 2008) [24] but not adequately utilized in development of resistance to scald disease in barley. The information on the type and magnitude of gene actions governing resistance genes and indirect selection of desirable parents via combining ability test would contribute in development of disease resistant cultivars (Zhan *et al*., 2008) [59].

In breeding of high yielding varieties crop with desirable qualitative and quantitative traits, breeders often face with the problems of selecting parents and crosses. Combining ability analysis is one of the valuable tool available to ascertain the combining ability effects and helps in selecting the desirable parents and crosses (Salvaraj *et al*., 2011) [40].

Race-specific major R-genes are often overcome in the field by new pathogen races within a short period of time, due to the ease of eliminating or modifying one out of a larger set of redundantly acting effector proteins. Although not a priori expected, even simultaneously introduced pairs of R-genes against the same pathogen were readily broken down (Brown *et al*. 1993) [5]. Therefore, in order to improve the durability of this type of resistance, more efforts are required. Especially, deeper knowledge about pathogen populations and effector functions would allow searching for and selecting R-genes that recognize highly conserved and (more) essential effectors. Stacking two R-genes of this category might provide a new level of resistance durability (Stergiopoulos *et al*. 2010) [45].

Marker-assisted selection

Selection of plants carrying genomic regions that are involved in the expression of traits of interest through molecular markers is possible by using marker-assisted selection (MAS). With the development and availability of an array of molecular markers and dense molecular genetic maps in crop plants, MAS has become possible for traits both governed by major genes as well a quantitative trait locus (QTLs) (Choudhary *et al*., 2008) [7]. QTL mapping has been useful to study resistance under complex genetic control to address i) how many loci are involved in complex resistance ii)are race specific resistance involved in quantitative resistance iii) what are the effects of plant development and environment on field resistance (Williams, 2003) [51]. In QTL mapping, a cross between two inbred lines is made and the co segregation of alleles of mapped marker loci and phenotypic traits allows the identification of linked markers (Kraakman *et al*., 2004) [28].

Inheritance of resistance studies of barley cultivars to scald (*Rhynchosporium secalis*) started since some 80 years ago (Mackie, 1929). Since then several resistance genes (R genes) against *Rhynchosporium secalis* have been identified and mapped. There are four major resistance loci, the Rrs1 complex on chromosome 3H with at least 11 known alleles, the Rrs2 locus on 7HS, Rrs13 on chromosome 6H and the Rrs15 locus on 2H (Bjørnstad *et al*., 2002) [4]. Similarly some resistance genes have been detected in wild barley, H. vulgare subsp. spontaneum as Rrs12, Rrs13, Rrs14 and Rrs15 on 7H, and Hordeum bulbosum (Rrs16). Many QTL studies revealed scald resistance on several chromosomes whose loci often coincided with locations of known scald resistance genes (Wagner *et al*., 2008) [50]. Genetic mapping for resistance genes to scald made on doubled haploid barley populations developed by using AFLP, RFLP, SSR and STS markers (Grønnerød *et al*., 2002) [18]. Most genes for resistance to barley leaf scald were mapped either to the Rrs1 locus on the long arm of chromosome 3H, or the Rrs2 locus on the short arm of chromosome 7H (Genger *et al*., 2005) [17]. Evaluation of scald resistance gene, Rrs14, transferred from wild progenitor was done by RFLP and storage protein markers using susceptible cultivar (Clipper) and third backcross (BC3) line homozygous resistance for Rrs14 (Garvin *et al*., 2000) [15].

Barley resistance to R. secalis is governed by both ‘major’ or complete resistance and ‘minor’ genes of smaller, generally additive effects (partial resistance). In addition crop growth stage and plant or canopy architecture can modify the expression of resistance. Resistance genes are distributed unevenly across the barley genome, with most being clustered on the short arms of chromosomes 1H, 3H, 6H and 7H, or in the centromeric region or on the long arm of chromosome 3H (Zhan *et al*., 2008) [59]. Molecular markers will greatly assist in the preservation and exploitation of germplasm, allow marker-aided selection, and facilitate in generating particular combinations of resistance genes and in resistance gene deployment. Markers allow the selection of individuals carrying favorable alleles from either parent and avoids the inclusion of individuals that are homozygous for unfavorable alleles (Michelmore, 1995) [33]. In addition recent developments in molecular techniques have lead to the realization that host resistance may be the result of more dynamic interactions than those proposed in evolutionary models which assume either gene-for-gene or matching-allele mechanism, while gene-for-gene model is a specific genetic interaction between a host and its pathogen, a qualitative resistance, which is as a result of relatively simple genetic control, and it renders a cultivar immune to disease (Clay and Kover, 1996) [8].

Transgenic Approaches

It is the introgression of defines genes by gene transfer resulting in transgenic barley events. Efficient barley transformation protocols exist, especially for a small number

of model cultivars. A first promising transgenic approach to durable resistance is the introduction of major R-genes from highly resistant wild relatives of crop plants (Van der Vossen *et al*., 2005) [48].

A second interesting approach is the silencing of susceptibility-related genes of barley. If successful, transgenic events would be released from effector-mediated defence suppression similar to the situation in mlo loss-of-function mutants showing immunity to Bgh (Piffanelli *et al*. 2002) [37]. Alternatively transgenic plants might also refuse to deliver nutrients to fungal pathogens, although this strategy will most likely be restricted to (hemi)biotrophic pathogens that are dependent on regulated active nutrient export from the host plant, at least during the early (biotrophic) phase of the interaction. Promising target genes in this respect might be glutamate or aspartate transporters as well as SWEET sugar transporters localized in lipid raft like membranes around haustoria (Chen *et al*. 2010a) [6]. Other potentially interesting, susceptibility-related genes of barley encode bax inhibitor 1 or WRKY1-3 transcription factors. Indeed, transgenic barley carrying RNAi constructs against these targets showed clearly enhanced resistance to Bgh (Eichmann *et al*. 2010) [11].

A third approach worth is host-induced gene silencing (HIGS) of essential housekeeping, cell wall-related or pathogenicity-related target genes of fungal pathogens. It was shown recently that fungal pathogens attacking corresponding transiently silenced or transgenic barley, wheat or tobacco plants are compromised in their development and exhibit silencing of the GUS reporter as well as endogenous target genes (Tinoco *et al*. 2010) [47]. More work will have to be invested to test if this promising concept, which can only be realized in transgenic plants, might be suitable to provide strong resistance in the field. Durability of the engineered HIGS resistance traits will most likely be high because fungi are not expected to delete essential components of their gene-silencing machinery to escape HIGS. Moreover, single point mutations of HIGS target genes will have no effect because the introduced h.a.i.rpin constructs usually cover several hundred bp of fungal DNA, which will leave ample efficient siRNA molecules left and right from any eventual mutation (Yin *et al*. 2011) [55].

Past experiences of screening of ethiopian barley germplasms for scald diseases resistance

To develop improved and resistance varieties the breeding program utilized local landraces and exotic germplasm since 1968. Reports indicated that between 1970 and 1990s approximately 14,168 local landraces were evaluated in nurseries. Most of the genotypes were found susceptible to scald, net blotch, spot blotch, leaf rust, and lodging. From this effort six outstanding hulled-barley varieties have been identified and released for large-scale production. On the other hand, every year exotic germplasms had been evaluated for desirable agronomic characters and resistance to diseases (scald and net blotch) and insect pests (shoot fly and aphids). Thus between 1966 and 2001 over 28,400 genotypes of introduced germplasms were evaluated at Holeta research center. From these efforts one hulled-barley variety, AHOR 880/61, was released and some other elite lines are being also used as sources of genes for desirable agronomic traits such as grain quality and stiff straw and for disease and insect pest resistance in the national crossing program (Birhanu *et al*., 2005) [3].

Screening of several landraces for their resistance to scald from different regions of Ethiopia showed variable responses to scald disease. For instance, populations from Arsi and Bale areas tend to be more susceptible to scald than populations from other regions. Whereas populations collected from higher altitudes were more resistant to scald than were populations from lower altitudes (Yitbarek *et al*., 1998) [58] indicating may be due to co evolution of host-pathogen interaction. In host resistance tests conducted in earlier studies at several barley growing sites of Ethiopia showed some promising resistant and/or tolerant entries were identified. For instance among the 500 lines evaluated for scald HB-114, HB-115, HB-116 EH/538/F-12-6-2, Beka, EH 207 B/F-4B-11-5B-5 and HB- resistant (Getaneh, *et al*, 1996) [57].

The utilization of the available barley landraces as source of gene to develop resistant varieties with the desirable traits is very crucial for breeding program as well as for farmer. The importance of depending selection program on local barley germplasm as compared to exotics material are not only important for their adaption to the growing conditions and stress factors in the target environment but also they could meet special demands of the consumers and producers (Van Leur *et al*.,1996) [49].

Conclusion

Barley (*Hordeum vulgare L.)* is the most important cereal crop grown in the world and in Ethiopia. The factors constraining the production of barley in the different barley production systems have been includes both biotic and abiotic stress. Scald (*Rhynchosporium secalis*) disease is of the most destructive pathogens of barley worldwide. It is caused by the haploid imperfect fungi (ascomycete) *Rhynchosporium secalis*. The disease is most severe in the highlands (above 2000 m) where precipitation is high and temperature is low during the cropping season. Yield losses due to scald vary between 21-67% and reduced grain quality depending on season and cultivar. The fungus persists on dead leaves and other plant residues to initiate primary infection. Screening and selection of barley genotypes for resistance to disease is currently hamperd by dearth of knowledge on variability of pathogen in the world as well as in Ethiopia. Barley responds to these fungal pathogens with altered gene expression often leading to the accumulation of pathogenesis-related (PR) proteins, with cell-wall appositions and sometimes with local cell death responses known as hypersensitive response (HR). The impact of scald can be minimized through increasing host resistance which is by far the most important defense mechanism that can be used to control diseases in crops. There are three principal ways to improve durable resistance of barley to major fungal pathogens: (1) stacking of carefully selected major R-genes by breeding, (2) marker-assisted introgression of multiple QTL by breeding and (3) generation of transgenic events introducing novel resistance or defence genes derived from barley, wild Hordeum relatives or other plant species; or silencing of susceptibility factors.

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