EVALUATION OF WHEAT (*Triticum aestivum* L.) GENOTYPES FOR RESISTANCE TO LEAF RUST (*Puccinia triticina* Eriks) IN KENYA

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DECLARATION AND RECOMMENDATION

Declaration

I hereby declare that this is my original work and has not been presented for examination in this or any other university for award of degree.

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Recommendation

This thesis has been submitted with our approval as university supervisors.

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DEDICATION

This thesis is dedicated to my family members for their patience and support.

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ABSTRACT

Leaf rust (*Puccinia triticina*) of wheat (*Triticum aestivum* L.) is one of the major foliar diseases contributing to yield losses in wheat worldwide. Objectives of this study were: (i) to determine genotypic variation among Kenyan wheat genotypes against leaf rust at adult plant stage (ii) to determine genotypic variation among Kenyan wheat genotypes against leaf rust at seedling stage (iii) to determine leaf rust virulence in Kenya using leaf rust differential sets. Three experiments were conducted at Kenya Agricultural and Livestock Research Organization (KALRO) in Njoro. In the first experiment, 144 wheat genotypes were evaluated for response to infection at adult stage in the field. The experiment was conducted in the field in 12 ×12 partially balanced lattice design to evaluate wheat genotypes for leaf rust infection and agronomic traits for two seasons. In the second experiment, the same genotypes were evaluated for resistance to leaf rust at seedling stage in the greenhouse. Genotypes sown in the greenhouse were inoculated with urediniospores after seedlings had attained growth stage 12. In the third experiment, 91 leaf rust differential lines were used for leaf rust virulence analysis in the greenhouse. Fifty-six percent of the screened genotypes in the greenhouse exhibited resistance (IT's of ";", "1", "2" or combinations) and the rest 44 % genotypes showed susceptible reaction. Genotypes *K. Tai, K. Korongo, Fletcher, Verder, R1244* exhibited both seedling and adult plant resistance during season one and two. Considering the adult plant disease response and yield potential, genotypes *R1301* and *R1305* showed lowest leaf rust infection and highest grain yield. Mean grain yield ranged from 0.06 to 6.81 tonnes ha^{-1}. Significant ($p \leq 0.001$) variations were noted among the seasons, genotypes tested over seasons and the interaction between genotype × season for plant height, a thousand kernel weight, and harvest index. There were significant ($p \leq 0.01$) effects due to seasons and genotypes for spike length, days to maturity, AUDPC and grain filling period, biomass, yield, respectively. Effects due to seasons were significant ($p \leq 0.05$) for hectoliter weight and AUDPC of stem rust infection. Resistant genotypes identified can therefore be utilized in Kenyan wheat breeding programmes for improvement of yield and leaf rust resistance with emphasis on adult plant resistance. Results of virulence analysis revealed varied disease infection types ranging from '0'to '3^{+}'. Leaf rust genes namely; *Lr1, Lr2a, Lr2b, Lr2c, Lr3, Lr3a, Lr3bg, Lr3ka, Lr9, Lr10, Lr11, Lr12, Lr13, Lr14, Lr15, Lr16, Lr17, Lr18, Lr19, Lr20, Lr21, Lr22a, Lr23, Lr24, Lr25, Lr26, Lr28, Lr29, Lr30, Lr27+Lr31, Lr32, Lr34, Lr35, Lr36* and *LrB* were resistant to Kenyan leaf rust races. These leaf rust genes could be valuable sources of resistance to leaf rust.

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LIST OF ABBREVIATIONS AND ACRONYMS

APR	Adult Plant Resistance
KALRO	Kenya Agricultural and Livestock Research Organization
AUDPC	Area Under the Disease Progress Curve
CIMMYT	International Maize and Wheat Improvement Centre
NPBRC	National Plant Breeding Research Centre
FAO	Food and Agriculture Organization
Lr	Leaf Rust Gene in Wheat
USA	United States of America
FDS	Final Disease Severity
ITs	Infection Types
Masl	Meters above sea level

CHAPTER ONE
INTRODUCTION

1.1 Background information

Bread wheat (*Triticum aestivum* L.) is one of the most widely cultivated and important food crop in the world. Besides being a high nutritive cereal, wheat sub-sector is identified as leading employer in Kenya especially in the primary growing areas of the country through its value chain (KALRO, 2013). The wheat sub-sector contributes 1.4% and 30% to overall and cereal Gross Domestic Product, respectively, employing over 500,000 people through linkages with several sectors such as transport, storage and distribution. Kenya is among the sub-Saharan countries rated as food insecure and partly contributed by both biotic and abiotic factors (McIntosh *et al*., 1995; FAO, 2016). The major biotic constraints affecting wheat are diseases, weeds and insect pests. However, there are abiotic constraints including drought and low soil fertility. Important diseases of wheat include; rusts, bunts, leaf blight, powdery mildew and head scab (Priyamvada *et al.*, 2011). Leaf rust caused by *Puccinia triticina* Eriks, is one of the most destructive wheat (*Triticum aestivum* L.) foliar diseases worldwide and it mostly infects wheat in low to medium altitude wheat growing areas of Kenya (Roelfs *et al*., 1992; Marasas *et al*., 2004). In Kenya, there is availability of rusts inocula resulting from growing of wheat throughout the year in different agro-ecological zones.

Yield losses due to leaf rust can be substantial. The final amount of loss depends on the crop development stage when the initial infections occur, and the relative resistance or susceptibility of the wheat genotype (Kolmer *et al*., 2007). High yield losses result when the initial infections occur early in the growing stage, especially before the jointing and tillering stages. Leaf rust and stripe rust (*Puccinia striiformis* f. sp. *tritici*) cause 60% loss of yield while stem rust (*Puccinia graminis* f. sp. *tritici*) can cause up to 100% loss in case of an epidemic, or when a susceptible cultivar is grown (Park, 2007). Yield losses caused by severe leaf rust incidence in a durum wheat field have been reported to range from 5%-16% on average, and up to 70% in epidemic years (Hurrerra-Fossel *et al*., 2006; Huerta- Espino *et al*., 2014). Although the yield reduction caused by leaf rust is lower than the yellow and stem rust, the level of its annual damage on the wheat plant is greatest because of its high frequency and wide spread occurrence (Naser *et al*., 2013).

Leaf rust may kill wheat seedlings by elevating respiration rate, reducing photosynthetic area on the leaf surfaces and lessening translocation of carbohydrates (Arslan *et al*., 2002). It acquires pathogenicity on resistant wheat genotypes because of its ability to

mutate and evolve into new pathotypes (Kolmer *et al*., 2005). The urediniospores are airborne and new races are introduced into a new area through migration and develop rapidly under optimal weather condition (Singh *et al*., 2005). Genes that condition effective resistance to the current leaf rust population need to be added to wheat breeding programmes in order to maintain high levels of resistance (Singh *et al*., 1998; Hovmoller, 2001). Wheat leaf rust infects leaf blades, although in some susceptible genotypes infection occurs on leaf sheath.

Two types of resistance have been identified in wheat-rust pathosystem; race-specific and race-nonspecific resistance. Race specific resistance is controlled by the major genes and it protects the plant against virulent pathogen during their entire growing period (Parlevliet, 2001). In contrast, race-nonspecific genes do not confer high level of resistance but due to the slow rusting effect they prevent epiphytoty of disease and provide longtime resistance (Herrerra-Fossel *et al*., 2007). To date, there are four known loci which contain *Lr*-genes designated as *Lr34*, *Lr46*, *Lr67* and *Lr68* that provide race non-specific resistance (Da-Silva *et al*., 2012). Introgression of major and minor genes in spring and winter wheat have been utilized to confer adult plant resistance (APR) to stem rust, leaf rust and stripe rust (Singh *et al*., 2008).

Fungicides can be used effectively in reducing leaf and stem rust severity and increasing yield of susceptible wheat genotypes (Wanyera *et al*., 2009). However, it can be an expensive method of disease control. Identification and subsequent introgression of resistant genes to susceptible but adapted wheat genotypes minimize utilization of fungicides and, consequently, it remains to be economical and environmental friendly way to reduce devastation of leaf rust disease of wheat (Martinez *et al*., 2001). Despite the fact that it takes long time to breed varieties, breeding for host plant resistance is one of the most viable and sustainable control measures (Singh *et al.*, 2004). This contributes to development of high yielding wheat genotypes which is the major objective of breeding programmes (Heidari *et al*., 2005). Knowledge on availability of new leaf rust races and type of genetic resistance is important in efforts to fight the leaf rust.

1.2 Statement of the problem

Leaf rust is among the most devastating foliar diseases that limits wheat production worldwide. High mutation rate of leaf rust occurs on wheat grown in areas with environmental conditions favouring infection on susceptible genotypes. The buildup of inocula of leaf rust variants contribute to the increase of virulence on resistant wheat genotypes. Leaf rust disease severely reduces the yield of wheat on the susceptible genotypes

depending on the stage of infection. Breeding of wheat genotypes with durable resistance to leaf rust continues to be a priority, but also a challenge due to resistance breakdown. The complexity of interactions among resistance genes with newly evolved pathotypes results in high turnover of released new varieties. For the past three and a half decades, Kenyan wheat improvement programme has not been laying emphasis on determination of virulence and pathogenicity of new leaf rust races. Currently, there is little knowledge of new leaf rust pathotypes in Kenya which has reduced the efficiency in breeding for the resistant genotypes. Identification of leaf rust resistant genotypes therefore, should be emphasized in order to counter the effect of new leaf rust races. Genetic resistance is the primary tool to protect wheat crops from leaf rust disease. Consequently, there is need for identification of genotypes with new sources of resistance genes which can be introgressed into susceptible but adapted genotypes to combat this disease and more importantly, improve wheat yield. Breeding for resistance, therefore, offers double benefits of both controlling the disease and reducing cost of production on wheat.

1.3 Objectives

1.3.1 General objective

To contribute to food security through identification and development of resistant wheat genotypes against leaf rust races in Kenya.

1.3.2 Specific objectives

i) To determine the genotypic variation among selected Kenyan wheat genotypes for resistance against leaf rust at seedling stage.

ii) To determine the genotypic variation among selected Kenyan wheat genotypes against leaf rust at adult stage.

iii) To determine leaf rust virulence in Kenya using leaf rust differential lines.

1.4 Hypotheses

i. There is no genotypic variation for leaf rust among selected on wheat genotypes at seedling stage.
ii. There is no genotypic variation for leaf rust among selected wheat genotypes at adult stage.
iii. There is no leaf rust virulence identified in Kenya using leaf rust differential lines.

1.5 Justification

Leaf rust is a devastating disease that reduce wheat yield in many wheat growing areas; it negatively affects quality and quantity of wheat resulting in low outputs which cannot meet the demand from ever increasing population in Kenya (Bolton *et al*., 2008). The rising demand in wheat and its products due to progressive increase in human population necessitates the growing of improved varieties with higher grain yield and durable resistance to major diseases such as leaf rust. Presently, the demand for wheat in Kenya, is at 900,000 tonnes and yet it produces about 450,000 tonnes (FAO, 2016). Consequently, wheat is imported from other countries such as Argentina, United States, Canada, Russia, Germany, Latvia and Lithuania, yet Kenya has the capacity to be self-sustainable in wheat production and in meeting demand for wheat products as it was between 1960 and 1972. Breeding for the resistant genotypes has been used as the main protection method against leaf rust but the challenge to host resistance is the emergence of new pathotypes. Rust resistance in wheat has traditionally been based on the use of specific resistance genes but the short-lived nature of the race specific resistance has created the necessity to search for the more durable type of resistance. Although chemical control of the wheat leaf rust is a short term mitigation measure, it increases the cost of production, whereas breeding for resistance to leaf rust is the most cost effective method with an estimate of 1:27 cost to benefit ratio (Marasas *et al*., 2004). As far as wheat leaf rust is concerned, there is no reason why yields from resistant genotypes in wheat growing zones should not be similar or even more than those achieved from wheat that was sprayed with fungicides. Identification and promotion of new leaf rust resistant genotypes with high yielding potential and desirable agronomic traits compared to the current genotypes would be the best strategy. Host resistance, therefore, should be used to minimize yield losses due to pathogens and consequently, feed the continuously increasing world population.

CHAPTER TWO
LITERATURE REVIEW

2.1 Origin of wheat (*Triticum aestivum* L.)

Bread wheat (*Triticum aestivum* L.) came in to existence around 7000 B.C.in the region extending from Transcaucasia to the southwest coastal areas of the Caspian Sea (Zohary and Hopf, 1993). It was domesticated 15,000 years ago in the Fertile Crescent, marking the start of modern civilization (Harlan, 1992). It is a hexaploid wheat which resulted from hybridization of tetraploid *Triticum turgidum* and diploid *Aegilops tauschii (*McFadden and Sears, 1946*).* It has been widely accepted that *A. tauschii* ssp. *strangulata* is the source of the wheat D genome and it is distributed from Transcaucasia (Armenia and Azerbaijan) to eastern Caspian Iran (Dvorak *et al*., 2012). Today wheat is grown from within the Arctic Circle to higher elevations near the equator (Miller, 1995). The domestication of the early wheat progressed by subconscious selection by the earliest growers, deliberate selection among variable material in the field of the primitive farmer for increased yield and planned breeding for uniformity (Feldman *et al.*, 2014).

2.2 Wheat production and its importance

Worldwide, wheat is the second most important cultivated cereal in the world, after rice (*Oryza sativa*), and its current world production is around 600-700 million tonnes from approximately 225 million hectares with China, India and USA being the leading producers respectively (FAO, 2012). A report by FAO (2016) estimates the world wheat production to have risen to 730 million tonnes in the year 2016 from 711.4 million tonnes in 2013/2014, 607 million tonnes in 2007 and 655.7 million tonnes in 2010. Wheat cultivation spans from 44^{o}S – 60^{o}N on both sides of the equator and an altitude range from sea level to 3,000 m.a.s.l.

In Kenya bread wheat is the second most important cereal after maize (*Zea mays* L.) (KALRO, 2013). The wheat industry contributes over KES 20 billion to the economy and supports about 11.3% of national population (KALRO, 2013). However, Kenya produces only 38.8% of its national requirements for wheat despite being the second important cereal grain after maize. The demand of wheat flour in Kenya at present cannot be sustained by local production, so the country relies on imports to meet almost half of its consumption. Kenya is among the sub-Saharan countries rated as food insecure and this is partly contributed by both abiotic and biotic factors with wheat leaf rust being one of the major threats (FAO, 2016).

Wheat is the leading crop with respect to use of land area, followed by rice and then, maize. This crop is a strategic pillar that contributes to food security and livelihood support in Kenya (KALRO, 2013). Approximately150,000 ha year^{-1}, with 20% from large scale and 80% from small scale. The crop is grown largely for commercial purposes on large scale farms. Kenya is self-sufficient in the hard variety of wheat but a net importer of softer variety. In Kenya, wheat production started at the beginning of the nineteenth century where, Lord Delamere a pioneer farmer in Nakuru region, began producing wheat in 1904 (Oehmke and Makanda, 1993). It was not until 1927 that formal wheat breeding research programme was initiated at the then National Plant Breeding Research Centre (NPBRC) in Njoro-Kenya (Gamba *et al*., 2003). Wheat has since been produced on large farms on Kenyan highlands and within Rift valley. The major wheat growing areas include; part of Central, Eastern and Rift Valley of Kenya (Feldman *et al.,* 2014).

Durum wheat (*Triticum turgidum*) is grown on heavy black clay soil (Vertisol), it has very narrow adaptation and also has lower yield potential as compared to bread wheat. Its kernels are bigger and heavier and are mainly suitable for pasta, *macaroni* and *pastini*. In addition, its stalk as other cereals residue is used as the animal feed and can also be used for mulching purposes in different agronomic practices in agriculture (Lemma *et al*., 2015). There has been increase in the *capita*$^{-1}$ consumption of wheat in Kenya and the three-year average capita^{-1} increased from 25 kg to 27 kg year^{-1} in 2003 to 2005 and 2006 to 2008 periods, respectively (FAO, 2008).

Wheat cultivars are superior to most other cereals in their nutritive value (Zohary and Hopf, 1993). Wheat is an important source of food, feed, employment and income in developing countries (FAO, 2008). It is one of the best cereal foods and provides more nourishment for humans than any other food source. This is attributed by its diet component, agronomic adaptability, ease of grain storage and converting grain into flour for making edible, palatable, interesting and satisfying foods. It is the most important source of carbohydrates in a majority of countries and its starch is easily digested as is most wheat protein. Wheat provides 20% of calories and 20% of daily protein to the world's population and 2.5 billion people, respectively in less developed countries. Additionally, wheat contains starch which vary between 60 to 75% of the total dry weight of the kernel (Shimizu *et al*., 2008) and the average concentration of zinc in the whole kernel of wheat is between 20 – 35 mg Kg^{-1} (Cakmak, 2004). Furthermore, wheat kernel contains vitamins B12 and B6 as well as lipids (palmitate, linoleate, oleate and triglycerides) (Cornell, 2003). A predominantly wheat based diet is high in fibre such as β-glucan whose consumption offer protection against heart

disease and cancer, normalizes blood lipids, regulates glucose absorption and insulin secretion.

2.3 Botany and genetics of wheat

Wheat is an annual crop which belongs to the tribe Triticeae in the family Gramineae (Symko, 1999). Wheat plant can be divided into two distinct parts, *viz* root and shoot system. There are two sets of root in wheat; the seminal or seedling roots and clonal roots. Seminal roots are produced by germinating seed and arise at the depth where the seed is planted, whereas, clonal roots, arise from the compact vegetative mass known as "crown". The entire roots are adventious. Shoot system comprises of stems, leaves and inflorescence (spike and spikelet) (Noda *et al*., 1994).

Worldwide, there are three commonly grown wheat species: Bread wheat *Triticum aestivum* (2n = 42 chromosomes), which forms the classes; hard red winter, hard red spring, soft red winter, hard white and soft white. *Triticum compactum* which consists of club wheat and *Triticum turgidum* ssp. Durum (2n=28), which mainly has durum wheat. Bread wheat is a segmented hexaploid, which regularly forms 21 chromosome pairs (2n=6x=42) during meiosis (Caldwell *et al.,* 2004). These chromosomes are sub-divided into 3 homologous groups of chromosomes, A, B and D genomes and contains 7 pairs of chromosomes each. This homology in hexaploid (AABBDD) wheat and tetraploid (AABB) wheat allows a range of chromosomal abnormalities to survive which is in contrast to diploid species such as maize and barley. At present, it is understood that hexaploid, *Triticum aestivum* (AABBDD) wheat is the product of two unique hybridization events of tetraploid, *Triticum turgidum* (AABB) and *Aegilops tauschii* (genomes DD) *(*McFadden and Sears, 1946*).* In the first event, the A genome progenitor joined with the B genome progenitor in B genome cytoplasm to form a basic tetraploid wheat (2n=4x=28, AABB). The second event involved hybridization between the tetraploid (AA BB) form and the D genome progenitor in D genome cytoplasm to form the uncomplicated hexaploid configuration, AABBDD (Junhua *et al*., 2011). The D genome in hexaploid wheat most likely contributed to a wider range of climate adaptation that facilitated the spread from the primary center of diversity and area of origin (Caldwell *et al*., 2004).

2.4 Overview of wheat leaf rust

Wheat leaf rust is a fungal disease that is devastating in wheat growing regions and has drastically decreased wheat production in most parts of the world (Kolmer, 2005). Leaf

rust originated from the Fertile Crescent region of the Middle East, where the natural arrays of the primary and secondary hosts are found (D'oliveira and Samborski, 1996). The earliest epidemic of leaf rust was reported in Kenya in 1908 (Thorpe, 1959). Although leaf rust is found almost everywhere that wheat is grown, suitable alternate hosts are rarely present for the fungus to complete the sexual cycle. Among plant pathogens, wheat leaf rust has somewhat long history of population studies, with nationally race surveys for this rust in the USA in 1926, in Canada in 1931 and in Australia in 1920 (Garvin *et al*., 2008).

This disease is the most prevalent of all the wheat rust diseases occurring in most wheat growing regions. It spreads through airborne spores or water splash. Before sporulation, wheat plants appear completely asymptomatic but after around 10 days of infection, the fungus begins to sporulate and symptoms become visible on wheat leaves. Leaf rust has many races with different virulence and the sexual life cycle requires a different host species (Kolmer, 2005). The most easily observed symptoms of leaf rust are brown pustules which develop on the leaf blades in a random scatter distribution which may group into patches in serious cases (Loegering, 1967). Onset of the disease is slow but accelerated in temperatures above 15 °C making it a disease of the mature cereal plant in summer, usually too late to cause significant damage in temperate areas. Infections can lead up to 50% yield loss exacerbated by drying leaves which fertilizes the fungus (Huerta-Espino *et al*., 2011).

The leaf rust fungus is specialized into several physiologic races that are known by their reactions on established set of differential wheat line. Races are identified by high to low infection type to near isogenic Thatcher lines of wheat with leaf rust resistance genes using four letter code nomenclatures (Long and Kolmer, 1989). For instance, annual survey of 2008/2009 wheat growing season, showed the existence of 43 physiological races of *Puccinia triticina* fungus where, the more prevalent races were *PTTS*, *TTTS*, *TTTT*, *PTTT*, *KTTT* and *PRTS* with 12.3%, 9.87%, 6.64%, 6.17%, 4.93% and 4.93%, respectively (*www.sydney.edu.au/.../cereal-rust-survey-2008-09*). Moreover, predominant leaf rust races in United States are *TBDS*, *MCDS*, *MCRK*, *TBBJ*, *TBBG*, *TBDS*, *TCDS*, *THBJ*, *TLGJ* and *TNRJ* (Kolmer *et al*., 2007).

2.5 Epidemiology of wheat leaf rust

The wheat leaf rust fungus is adapted to a range of varied climatic conditions, and the disease can be found in diverse wheat growing areas throughout the world (Roelfs *et al*., 1992). Being biotroph, wheat rust pathogens need live wheat plants or other alternate hosts in order to survive. For instance, leaf rust requires *Thalictrum speciosissimum* or *Isopyrum*

fumaroides as the alternate host. During crop season, large amount of urediniospores are produced and dispersed by the wind either to the new host or the same plant. There are three known modes of rust dispersal. First, is the single event extremely long-distance mode; it is categorized into either unassisted long-distance dispersal when it occurs through airborne or assisted long-distance dispersal when facilitated by travelers clothing or infected plant materials. Second is the step-wise range expansion mode; this occurs over a shorter distances within the region or country and the third is extinction and recolonization which occurs in temperate areas lacking suitable conditions for survival of pathogens throughout the year and host plants (Singh *et al*., 2006).

If infection without sporulation occurs, then leaf rust pathogen survives the same environment that the wheat leaf survives. The spore germination process requires moisture, in which it works best at 100% humidity and optimum temperature of 15 °C – 20 °C (Dyck and Johnson, 1983). The fungus requires dew period and temperature of about 20 °C, however, more infections occur with longer dew periods. Longer dew periods are required at cooler temperatures, for example at 10 °C, a 12 hours' dew period is necessary, while, few if any infections occur where temperatures are above 32 °C (Stubbs *et al*., 1986) or below 2 °C. When uredinia survive the winter at some threshold level on wheat crop or where spring-sown wheat is the recipient of exogenous inoculum at an early stage before heading, it leads to severe epidemics and losses can occur when the flag leaf is infected before anthesis. Occasionally, autumn-sown wheat can be severely infected in the autumn, resulting in reduced root growth, tillering and even plant death before anthesis (Roelfs *et al*., 1992). When canopy is highly infected, horizontal spread across the plant occurs. The horizontal spread of inoculums habitually results in heavily infected flag leaves, but little or no rust infection on the lower leaves of the wheat plants. When environmental conditions are favourable, disease spread can be very prompt (Roelfs, 1985).

2.6 Life cycle of wheat leaf rust

Leaf rust is a monocyclic and heteroecious rust fungus which forms five types of spores in its life cycle. Urediniospores, teleospores and basidiospores develop on wheat plants (primary host), whereas, pycniospores and aeciospores develop on either *Thalictrum speciosissimum* or *Isopyrum fumaroides* (alternate hosts) (Singh *et al*., 2008). Spores germinate optimally at 100% relative humidity while optimum temperature is between 15 °C–20 °C (Dyck and Johnson, 1983). Before sporulation, wheat plants appear completely asymptomatic. Wheat leaf rust has both sexual and asexual life cycle and in order to complete

the sexual life cycle, it requires a second host *Thalictrum speciosissimum* or *Isopyrum fumaroides* on which it overwinter. In areas where *Thalictrum speciosissimum* and *Isopyrum fumaroides* does not grow, the pathogen only undergoes its asexual life cycle and stagnates as mycelium or uredinum. After around 10-14 days of infection, the fungi begin to sporulate and the symptoms become visible on the wheat leaves (Garvin *et al*., 2008). The number of spores can vary greatly with production of approximately 3000 spores per uredinium per day. If wheat leaf remains alive, this level of spore production may continue for 3 weeks or more (Roelfs *et al*., 1992).

2.7 Effective leaf rust resistance genes

Knowledge of major genes for resistance in the predominant wheat genotypes is essential when evaluating crop response to leaf rust. Genetic resistance is the ideal method to reduce losses from leaf rust (Fida *et al*., 2001). To date, about 70 leaf rust resistance genes (*Lr*) have been catalogued (Mclntosh *et al*., 2007) but when used commercially in Mexico, the average life of race specific genes have been roughly 3 years. These genes have been sequestered, mapped to specific chromosomes and given official descriptions according to the criterions set forth in the catalogue of gene symbols for wheat (Singh and Huert-Espino, 2003). Almost all these genes cause resistance that is linked with chlorosis or necrosis (Drijepondt and Pretorius, 1989). Resistance gene expression is reliant on the genetics of host parasite interaction, temperature conditions, plant developmental stage and interaction among resistance genes with expressers or other resistance genes in the wheat genomes (Singh *et al*. 1991).

Genes expressed in seedling plants have not provided long lasting effective leaf rust resistance, whereas, adult plant resistance (APR) genes *Lr13* and singly and together have provided the most resilient resistance against leaf rust in wheat throughout the world. Furthermore, in the past *Lr13* was an important gene for resistance and continues to contribute to resistance in some regions such as Australia and Canada (Singh and Rajaram, 1992). Two adult plant resistance genes (*Lr34* and *Lr46*) confer stable resistance to varied leaf rust pathotypes and are believed to be durable (Singh *et al*., 1998).

2.8 Seedling and adult plant resistance

There is high specialty of pathogens specific to certain host species which is largely due to their parasitic nature. The most influential tool to test adult plant resistance is to grow a genotype for a long period in an environment with favourable conditions for the disease.

However, it can also be tested by either testing with many races of a pathogen from an existing population or by growing the genotypes in many locations (Johnson, 1981).

In contrast to studies of seedling resistance to leaf rust, APR remains uncharacterized, although, it can be conferred by single (McIntosh *et al*., 1995), two or more genes (Singh *et al*., 2001). Being a component of some durable leaf rust resistances, there is growing curiosity in characterizing different sources of APR to advance their efficient use in breeding programmes (Amin *et al*., 2005).

2.9 Control of wheat leaf rust

2.9.1 Chemical control

Fungicides have been used to control cereal diseases for more than 100 years. In addition, they have been found to delay senescence and consequently increase yield production through prolonged duration of green-leaf area. The choice and appropriate use of fungicides is effective but least employed method in management of leaf rust. For instance, *tubuconazole* is applied at GS32 and GS39 at the rate of 62.32 g ha^{-1}. This fungicide bides to the fungal microtubules blocking nuclear division, consequently, stopping hyphal growth (*www.hgca.com/publications*). Varietal susceptibility, growth stage at application and level of infection determines fungicides effectiveness. Moreover, early application is the most appropriate since leaf, stem and transport system damage is reduced which ensures nutrient translocation and proper grain filling (Wanyera *et al*., 2010). Chemical control with *triazole*-based fungicides may be useful for control of infections up to ear emergence but it is challenging to justify economically in attacks after this stage. These chemicals have greater systemic activity and, as a group, tend to be absorbed and redistributed more quickly within the leaf and upward to new developing leaves. *Triazoles* are early post-infection fungicides and have the ability to inhibit or stop the development of infections that have already started. They have an anti-sporulant activity that helps to slow disease development by limiting the fungus and it provides 14-21 days of protection (Hershman, 2011).

In wheat fields, there is evolution of new leaf rust races which results in vanity of the previously resistant wheat genotypes. There was no need to apply fungicides in the past since the widespread deployment and cultivation of resistant genotypes had provided adequate protection of crops against rusts. Furthermore, integrated management of rust diseases is very crucial where fungicides can play a major role until new resistant genotypes are developed and released (Wanyera *et al*., 2009).

2.9.2 Cultural methods

Cultural practices on wheat are ususally employed to control leaf rust epidemics. No single practice is effective under all conditions, but the existing resistance is enhanced through use of a succession of cultural practices. These practices include: crop rotation to reduce the inoculum build-up, use of early maturing genotypes, timely planting, green-bridge removal to control epidemics that would result from endogenous inoculum and control of volunteer plants. In some areas, control of timing, frequency, amount of irrigation and fertilizer application can aid in disease control. For instance, late planting may increase the chance of spring infection by exogenous inoculation (Rajaram *et al*., 1996). Control of agronomic practices aids in limiting rust development in wheat growing fields. This can be achieved through discouragement of double cropping in order to decrease the rust movement from one plant to the next and elimination of alternate hosts (Wanyera *et al*., 2010). Eradication of alternate hosts (*Thalictrum speciosissimum* and *Isopyrum fumaroides),* which function as a source of sexual reproduction also plays a role in controlling leaf rust disease of wheat (Kolmer, 1996). Use of multiline cultivars and cultivar mixtures also contributes to the reduction of wheat leaf rust infection efficacy through the dilution effect (Jeger *et al*., 1981).

2.9.3 Genetic resistance

In any leaf rust breeding programme, the objectives of genotypes screening are to evaluate the scope of virulence of the new leaf rust races and to identify the source of resistance to the present races in -a large number of germplasm (Jin and Singh, 2006). Measurement of GE interactions for disease resistance and yield enables the plant breeder to identify broadly adapted genotypes that offer stable performance across many sites, as well as under high disease pressure conditions (Yan and Tinker, 2005). Quantitative traits such as yield are usually influenced by genotype, environment and genotype × environment (GE) interaction (Yan and Hunt, 2002). The plant breeders' aim of developing varieties that are best performing and most stable is usually complicated by the cross over type of GE interaction, since it results in inconsistent performance of genotypes across environments (Yan and Hunt, 2002). This results in reduction of the progressive selection in any one environment. However, it can be managed by selecting genotypes that are broadly adapted to a range of environments (Yau, 1995).

Use of resistant genotypes, has been the principal mechanism of wheat leaf rust control (Johnson, 1992). Since, virulence occurs for majority of catalogued resistance genes (Mclntosh *et al*., 1995), the paramount control strategy of leaf rust encompasses combination

of race-specific genes. Although, there is little information on screening of wheat genotypes for leaf rust in Kenya, the evaluation at both seedling and adult plant growth stages has been done in other countries. For instance, the study conducted in Egypt in 2010/11 and 2011/12 for leaf rust resistance screening reported 9 resistant varieties at both seedling and adult growth stages which included; *Sakha94*, *Giza168*, *Gemmiza9*, *Gemmiza10*, *Gemmiza11*, *Sids12*, *Sids13*, *Misr1* and *Misr2* (Draz *et al*., 2015). In Australia, germplasm screening and lines advancement involves routine tests against infection at seedling stage in the greenhouse and APR at the field for the three rust pathogens (Park, 2008). *Americanozs*, *Americano44d*, *surpreza*, *Fontana* and *Fronteira* were genotypes identified for leaf rust resistance in Australia (Perez and Roelfs, 1989). In the study carried out to identify and map leaf, stripe and stem rust resistance loci in Mexico, French cultivar *Sachem* was reported to be resistant to the three rust diseases of wheat. A major leaf rust quantitative trait locus (QTL) was identified on chromosome 7B at *Xgwm146* in *Sachem*. However, leaf rust severity in field nurseries was 1% at El Batan (2009), 5% at Obregon (2010) and 0 % at Toluca for *Sachem* in 2009 and 2011 (Singh *et al*., 2013). Most genotypes remain resistant for a period of more than five years where an active breeding programme exists, however, gene *Lr34* together with other unknown slow-rusting genes is involved in the durability of *Fontana* and other wheat genotypes (Singh, 1992). An enhancement of resistance involves the introgression of resistant genes through backcrossing of advanced lines to the recurrent parent which is normally done at F_1BC_5 or F_1BC_6 followed by seedlings and adult plants evaluation.

To date, about 70 leaf rust resistance genes in wheat have been mapped to chromosome location and given gene designations (Mclntosh *et al*., 2010), leaf rust resistance genes were initially characterized in wheat associated species such as *T. tauschii* (*Lr21),* *Aegilops elongatum* (*Lr24*), *A. umbellulata* (*Lr9*) and common rye, *Secale cereal* (*Lr26*) (Browder, 1990). As in case of seedling resistance genes, races with virulence to these adult plant resistance genes have eroded their effectiveness. Several other genes express a partial type of resistance that is displayed by fewer uredinia of variable size that are surrounded by variable amounts of chlorosis (Caldwell, 1968). Adult plants optimally express this kind of resistance as seedlings can be susceptible. These genes have provided long-term durable resistance since; virulent forms of leaf rust have not yet been detected.

Of the genes deployed on wheat germplasm around the world, *Lr34* is the most effective and characterized gene. *Lr34* has received much attention in recent years, because it is present in many wheat genotypes throughout the world that have shown durable resistance to leaf rust and it enhances the effect of other resistance genes (German and Kolmer, 1992).

The stability of the resistance is attributed to interactions with APR genes *Lr12* and *Lr13* in particular (Sawhray, 1992). In wheat genotypes missing other effective *Lr* genes, *Lr34* expresses resistance in a quantitative way through an increased inexpression period, decreased infection type and uredinium size (Singh, 1993). Other leaf rust resistance genes *Lr46,* and *Lr68* also confer adult plant-partial resistance (Singh *et al*., 1998), however, these genes have not yet been cloned and sequenced. Due to the highly variable nature of leaf rust, durable leaf rust resistance in wheat genotypes have been problematic to attain. However, certain combinations of genes have provided long lasting resistance. For instance, hard red spring wheat genotypes with combinations of *Lr13, Lr16, Lr23* and *Lr34* have restrained high levels of resistance for over 30 years (Kolmer *et al*., 2007). Wheat genotypes advanced at CIMMYT with combinations of adult plant genes *Lr34, Lr46,* and *Lr68* have shown long lasting resistance, however, the deployment of leaf rust resistance genes is the most economical means to curb this disease and is highly recommended in all plant breeding programmes (Akin *et al*., 2013).

CHAPTER THREE
EVALUATION OF KENYAN WHEAT (*Triticum aestimum*. L) GENOTYPES FOR LEAF RUST (*Puccinia triticina* Erik) AT ADULT PLANT STAGE

3.1 Abstract

Leaf rust (*Puccinia triticina*) is one of the major rust diseases that affect wheat (*Triticum aestivum)* production worldwide. The objective of this study was to determine genotypic variation among Kenyan wheat genotypes against leaf rust at adult plant stage. A set of 144 genotypes were evaluated in a two-season field experiments at Kenya Agricultural and Livestock Research Organization (KALRO), Njoro. In the field, genotypes were sown in 12×12 partially balanced lattice design. Adult plant infection assessed by Area under Disease Progress Curve ranged from means of 42.00 to 145.00. Mean grain yield ranged from 0.06 to 6.81 tonnes ha^{-1}. Highly significant ($p \leq 0.001$) variations were noted among the seasons, genotypes tested over seasons and the interaction between genotype × season for plant height, a thousand kernel weight (TKW), and harvest index. There were significant ($p \leq 0.01$) effects due to seasons and genotypes for spike length, days to maturity, leaf rust infection and grain filling period, biomass, yield, respectively. Effects due to seasons were significant ($p \leq 0.05$) for hectoliter weight and stem rust infection. Genotypes *K. Tai*, *K. Korongo*, *Fletcher, Verder, R1244*, *R1301* and *R1305* exhibited adult plant resistance in both seasons. Considering the disease response and yield potential, genotypes *R1301* and *R1305* showed lowest leaf rust infection and highest grain yield. These genotypes are suitable candidates for utilization in yield and leaf rust resistance improvement programmes in Kenya.

Key words: Wheat genotypes, Leaf rust, Resistance

3.2 Introduction

Leaf rust caused by *Puccinia triticina* Eriks., is among the main foliar diseases limiting wheat (*Triticum aestivum* L.) production worldwide (Cherukuri *et al*., 2005). Yield losses of up to 40 % in epidemic years have been reported (Bolton *et al*., 2008). In addition to the direct yield losses, leaf rust causes quality down grade and additional cost is also incurred for disease control; for example, application of fungicides (German *et al*., 2007). Leaf rust, stem rust caused by *Puccinia graminis* and stripe rust caused by *Puccinia striiformis* are the most damaging fungal diseases of wheat that significantly reduce yield, quality and weight of kernels (Huerta-Espino *et al*., 2011). Continuous growing of wheat in Kenya has made the fields to remain infectious due to the accumulation of the inocula throughout the year. Leaf

rust may kill wheat seedlings by elevating respiration rate, reducing photosynthetic area on the leaf surfaces and decreasing translocation of carbohydrates (Arslan *et al*., 2002). Although the yield reduction caused by leaf rust is lower than the yellow and stem rust, the level of its damage is greatest because it is most common and widely distributed of the three rust diseases (Huerta-Espino *et al*., 2011; Naser *et al*., 2013). The cultivation of large area of susceptible wheat genotypes allows a large leaf rust population to proliferate, creating a reservoir for mutation and selection (Kolmer *et al*., 2005).

Leaf rust fungus is adapted to a wide range of different climates, and it can be found in diverse wheat growing areas throughout the world because the dispersal of airborne spores cannot be constrained (Roelfs and Singh, 1992; Brown and Hovmoller, 2011). The disease has remained virulent even onto genotypes which are perceived to be resistant due to its ability to mutate and evolve new pathotypes (McDonald and Linde, 2002). The urediniospores are airborne and new races are introduced into new areas from one susceptible host to another where they develop rapidly under optimal weather conditions (Brown and Hovmoller, 2011). Each of the spores released is capable of starting a new infection and can cause significant destruction on wheat within a few weeks (Watson and Luig, 1983; Brown *et al*., 2002). Wheat leaf rust infects leaf blades, although in some highly susceptible genotypes infection occurs on leaf sheath and glumes and it is most damaging when the infections occur on the upper leaves before flowering stage (Huerta-Espino *et al*., 2011).

Resistance to leaf rust in wheat often is determined by adult plant resistance genes in combination with seedling resistance genes. The significance of disease in particular, depends upon the prevalence of aggressive and virulent races of the pathogen as well as their compatibility with the genetic constitutions of the host in a given environment (Kolmer, 1996; Kolmer, 2005). A total of 67 genes conferring resistance to leaf rust have been catalogued to date (McItosh *et al*., 2008). These genes alone or in combination provide a satisfactory level of resistance. For example, the congregating genes *Lr34* and *Yr18* have remained effective for more than 50 years (William *et al*., 2003). Two genes for leaf rust resistance in wheat, *Lr10* (Feuillet *et al*., 2003) and *Lr21* (Huang *et al*., 2003) have been isolated, cloned and sequenced. Both genes have sequences that encode nucleotide-binding site leucine-rich repeat regions which are characteristic of disease resistance genes in plants. Special mention of *Lr26* despite its susceptibility is essential since this feature significantly in Pakistani wheat cultivars. The virulence to *Lr26* appears every year and wheat varieties carrying *Lr26* continue to be cultivated globally due to the T1BL.1RS translocation that it is associated with exceptional grain yield advantages (Fayyaz *et al*., 2008).

High yielding wheat genotypes that are nearly immune to leaf rust could be developed by accumulating slow rusting resistance genes such as *Lr34* and *Lr46* through intercrossing parents that show intermediate disease levels (Hussain *et al*., 1999; Singh *et al*., 2000). Genotypes with *Lr34* and two to three additional genes have shown stable environmental response and final disease ratings lower than five percent under heavy disease pressure (Singh *et al*., 2001). Slow rusting or partial resistance has been reported to be more durable resistance than single seedling resistance genes (Li *et al*., 2010).

Despite the fact that it takes long time, breeding for durable resistant wheat genotypes to leaf rust remains a cost effective option of minimizing loss due to this disease (Yuen *et al*., 2007). Field surveys are equally important for monitoring the distribution of current pathotypes and virulence factors caused by *Puccinia triticina*. Furthermore, observations and monitoring at the field level helps greatly in knowledge of new virulence pathogen combinations. In Kenya leaf rust disease has received less attention with the presence of stem and yellow rusts which are the most aggressive hence, efforts to tackle the leaf rust problem has not been majored on. By approaching the limits of biological productivity of wheat in the recent years there has been greatly increased need for new, resistant and high yielding genotypes (Hailegiorgis and Genet, 2011). The objective of this study was to determine genotypic variation among Kenyan wheat genotypes against leaf rust at adult plant stage.

3.3 Materials and methods

3.3.1 Experimental site

The study on virulence of leaf rust disease to different wheat genotypes was conducted in the field at Kenya Agricultural and Livestock Research Organization (KALRO)-Njoro (0° 20´S, 35° 56´E), 2185 meters above sea level. This site is located in the highlands and categorized as zone III (LH_3) of the Agro ecological zones, in the Rift Valley Kenya (Jaetzold *et al*., 2012). The research station experiences an average minimum and maximum temperature of 8 ± 2 °C and 25 ± 2 °C, respectively and an average annual precipitation of 996.4 ± 4.2 mm (KALRO Meteorological Station No. 903502 (1), 2013). The soil in this area is predominantly *Molli Andosols* that is well drained with an underlying volcanic stratum.

3.3.2 Field experiment

a) Genotypes

One hundred and thirty three Kenyan spring wheat genotypes released in 20th and 21st century plus eleven introductions were evaluated for adult plant resistance in two seasons. Most of the genotypes were semi-dwarf in stature, with exception of the tall late maturing varieties. Phenologically, the test genotypes matured differently but most of them fell within the class of early and medium with a few late maturing types. A susceptible cultivar *K. Chiriku* was used as a check.

b) Experimental procedure

The genotypes were planted in a field that was previously under canola (*Brassica napus*) crop. The land was cultivated and harrowed to a fine tilth suitable for wheat growth using a disc plough and harrow, respectively. Each entry was sown in an experimental unit measuring 0.75× 0.2 m at an equivalent seed rate of 102.9 $Kgha^{-1}$, adjusted from 95% to 100% germination. The seed was sown in the rows spaced 20 cm apart while within the row seed was placed at a distance of approximately 5 cm apart. At sowing time, Di-ammonium Phosphate (DAP) (18:46:0) fertilizer was applied at the rate of 125 $Kgha^{-1}$ sufficient to supply 22.5 Kg Nha^{-1} and 25.1 Kg Pha^{-1}. The genotypes were evaluated in 12 × 12 partially balanced lattice design with three replications. The blocks and replications were separated from each other by an alleyway measuring 0.5 m. A mixture of susceptible genotypes was planted perpendicular to all the plots and in the borders separating the replicates which acted as a source of inoculum. At tillering stage (GS 20-29) (Zadoks *et al*., 1974), each experimental plot received Calcium Ammonium Nitrate (CAN) at an equivalent rate of a 100 $Kgha^{-1}$ which supplied an additional 33 Kg $N.ha^{-1}$. Growth of weeds were restricted by applying a post emergence herbicide, Hussar Evolution (*Fenoxaprop-p-ethyl* 64 gha^{-1} *+Idosulfuron methyl sodium* 8 gha^{-1} *+Mefenpyr-diethyl* 24 $g.ha^{-1}$).

The level of soil moisture was measured by soil moisture meter (Model PMS714, Film Badge Service Company) in an interval of seven days. Whenever there was inadequate rains, during the first season the field was irrigated to field capacity immediately after planting in order to initiate germination and sustain growth of seedlings, thereafter, the frequency of irrigation was determined by the level and retention of the moisture in the soil. The second season experiment was conducted during the main rainy season, where the experiment depended exclusively on soil moisture derived from the rainfall. The sucking and chewing pests on the wheat plants in the experiment were controlled by application of a

systemic insecticide, Thunder OD 145 (*imidachloprid* 30 gha^{-1} + *beta-cyfluthrin* 13.5 gha^{-1}), twice at tillering (GS 20-29) and ear emergence (GS 50-69).

c) Data collection

Leaf rust infection on wheat was evaluated as percent coverage of leaves with rust pustules following modified Cobb's Scale (Peterson *et al*., 1948) where 0% = immune and 100% = completely susceptible. Evaluation of infection was done five times, at an interval of 7 days between heading (GS 50-69) and plant maturity (GS 70-89) (Zadoks *et al*., 1974). Infection types on wheat grown in the field was classified according to Johnston and Browder, (1966) where; Immune (0) = no uredinia or other macroscopic sign of infection; Resistant (R) = small uredinia surrounded by necrosis; Moderately Resistant (MR) = small to medium uredinia surrounded by chlorosis or necrosis; Moderately Susceptible (MS) = medium-sized uredinia that may be associated with chlorosis and Susceptible (S) = large uredinia without chlorosis or necrosis.

With regard to agronomic traits, days to heading and anthesis were determined when 50% of plants in a plot had heads with anthers extruded from florets. Plants were considered mature when peduncle had attained golden colour. Height of wheat plant was estimated from a random sample of 5 plants from the base of the plant to the tip of the spikes excluding awns. At physiological maturity, yield was estimated from each plot and standardized to 12% moisture content. Thousand kernel weight (TKW) was estimated as weight of thousand kernels. In addition, hectoliter weight was estimated using hectoliter cup. Grain filling period was computed by determining the time photosynthates took to fill the kernels from anthesis to maturity.

Harvest index was calculated using the following formula:

$$\text{Harvest index (HI)} = \frac{\text{Grain yield (g)}}{\text{Total biomass(g)}} \quad \text{(Equation 1).}$$

d) Data analyses

An equation adopted from Campbell and Madden (1990) was used to calculate AUDPC using computer software developed by CIMMYT Mexico (CIMMYT, 2008) as follows:

$$\text{AUDPC} = \sum_{i-1}^{n-1} \left(\frac{y_i + y_{i+1}}{2}\right)(t_{i+1} - t_i) \quad \text{(Equation 2).}$$

Where; n is the number of readings, t is time of each reading in days, y_i is proportion in percent of affected foliage at each reading, t_{i+1} is second assessment date of two consecutive assessment and y_{i+1} is disease severity on assessment date (i+1). The cultivars resistance was compared using Area under Disease Progress Curve and Final Disease Severity (FDS) data.

The analysis of variance was done to determine the significant differences among the selected wheat genotypes for the different agronomic traits using PROC. GLM in Statistical Analysis System (SAS) version 8 (SAS Institute Inc., Cary, 2001). The data for all agronomic traits and kernel quality was analyzed using the following statistical model:

$Y_{ijklm} = \mu + S_i + R_{j(i)} + B_{k(ij)} + G_l + SG_{il} + \varepsilon_{ijklm}$ ..... (Equation 3).

Where; Y_{ijkl} =Observation of experimental units; μ =Overall mean; S_i = Effect due to i^{th} season; $R_{j(i)}$ = Effect due to j^{th} replicate in the i^{th} season; $B_{k(ij)}$ = Effect due to k^{th} block in the j^{th} replicate in the i^{th} season; G_l = Effect due to l^{th} genotype in the k^{th} block in the j^{th} replicate; SG_{il} = Effect due to interaction between i^{th} season and l^{th} genotype in the i^{th} season in the j^{th} replicate; ε_{ijklm}= Random error component.

The following SAS procedure was used to perform a combined analysis for field data:

```
Title 'Field screening';
Data wheat;
Input Rep Block Genotype Season Height Spkl Grainfp Biomass
Maturity yield TKW Hecto Audpclr Audpcsr Audpcyr HI;
Cards;
;
Proc Print;
Proc Glm;
Class Rep Block Genotype Season;
Model Height Spkl Grainfp Biomass Maturity yield TKW Hecto Audpclr
Audpcsr Audpcyr HI=Season Rep (Season) Block (Rep*Season) Genotype
Season*Genotype/SS4;
TEST H=GENOTYPE E=SEASON*GENOTYPE;
TEST H=SEASON E=REP(SEASON);
TEST H=Rep(Season) E=Block(Rep*Season);
RANDOM Block(Rep*Season) Season Season*Genotype;
Means Genotype Season Genotype*Season/ LSD;
Run;
```

Wheat genotypes and replicates were considered as fixed effects while blocks, seasons and interaction between season × genotype were considered as random effects. From the expected mean squares, random error was used to test the effects of season × genotype

and blocks, season × genotype interaction was used as an error term for genotype while blocks were used to test the effects of replicates. Replicates were used as an error term for seasons. Means of wheat genotypes were separated using Least Significant Difference (LSD) test (Steel and Torrie, 1980). Where genotypic effects were significant at $p \leq 0.05$ following the formula:

$$LSD = \frac{t(s\sqrt{2})}{\sqrt{n}} \quad \text{(Equation 4).}$$

Where *t* is tabulated t value, *s* is standard deviation of all the plots and *n* is number of observations in each variety. A Pearson correlation coefficient analysis was done to establish the relationship between the different agronomic traits measured using the following formula:

$$r = \frac{n(\Sigma xy) - (\Sigma x)(\Sigma y)}{\sqrt{[n\Sigma x^2 - (\Sigma x)^2][n\Sigma y^2 - (\Sigma y)^2]}} \quad \text{(Equation 5).}$$

(*http://mathworld.walfran.com/correlationcoefficient.html*).

Where *r* is Pearson's correlation coefficient, n is the number of samples, x is the dependable variable and y is the independent variable.

The following SAS procedure was used to correlate yield components and wheat rust diseases:

```
'Title correlation'
Data Corr;
Input yield Tkw Hecto Audpclr Audpcsr Audpcyr;
Cards;
;
Proc Corr;
Run;
```

Stepwise multiple regression was performed using SAS PROC. REG forward elimination method to determine the effects of leaf rust, stem rust and yellow rust on grain yield, TKW and hectoliter weight (SAS, 2001) using the equation:

$$Y_i = \beta_0 + \beta_1 X_{1(i)} + \beta_2 X_{2(i)} + \beta_3 X_{3(i)} + \varepsilon_i$$

Where Y_i is expected value of dependent variable for a given set of independent variables X_1, X_2, and X_3; β_0 is expected value of dependent variable at X_1, X_2 or $X_3 = 0$; β_1, β_2, and β_3 is partial regression coefficients for every unit increase or decrease in independent variable $X_{1,}$

X_2, and X_3, respectively and ε_i is residual component. Yield, TKW and hectolitre weight were considered as dependent variables while stem rust (X_1), leaf rust (X_2) and yellow rust (X_3) were considered as independent variables.

The following SAS procedure was used in stepwise multiple regression analysis:

```
'Title stepwise regression'
Data Regression;
Input yield Tkw Hectolitre Audpclr Audpcsr Audpcr;
Cards;
;
Proc Reg;
      Model yield Tkw Hectolitre = Audpclr Audpcsr Audpcyr/Selection
= Forward;
Run;
```

3.4 Results

3.4.1 Environmental conditions during crop growth seasons

The rainfall and temperature experienced during the growth period of the crop varied. The average rainfall and temperature experienced in the first season was 3.57 ± 1.87mm and 24.25 ± 1.28 °C, respectively and second season had 2.91 ± 1.14mm and 22.87 ± 1.18 °C rainfall and temperature, respectively. The average soil moisture experienced in the first and second season was 16.16 ± 0.27 mm and 15.21 mm ± 0.55, respectively while the average temperature was 23.85 ± 0.41 °C in season 1 and 22.15 ± 0.29 °C in season 2 (Table 3.1).

Table 3.1. Means of temperature and rainfall experienced over the two growing season in KALRO, Njoro in 2016.

Season	Air temperature (°C)	Air rainfall (mm)	Soil moisture (mm)	Soil temperature (°C)
Season 1	24.25 ± 1.28	3.57 ± 1.87	16.16 ± 0.27	23.85 ± 0.41
Season 2	22.87 ± 1.18	2.91 ± 1.14	15.21 ± 0.55	22.15 ± 0.29

3.4.2 Analysis of variance and genotype × season interaction

Highly significant ($p \leq 0.001$) variations were noted among the seasons, genotypes tested over seasons and the interaction between genotype × season for plant height, a thousand kernel weight, and harvest index. There were significant ($p \leq 0.01$) effects due to seasons and genotypes for spike length, days to maturity, leaf rust infection and grain filling period, biomass, yield, respectively. Effects due to seasons were significant ($p \leq 0.05$) for hectoliter weight and stem rust infection. There were no significant variations noted for grain

filling period between seasons however, there were significant ($p \leq 0.001$) effects due to genotypes and genotype × season for grain filling period (Table 3.2).

Table 3. 2. Mean squares of wheat genotypes evaluated for agronomic traits, yield, gain quality, leaf rust, stem rust and yellow rust reactions over two seasons at KALRO, Njoro.

Source of variation	Df	Expected mean squares	Height (cm)	Spike length (cm)	Grain filling period (days)	Biomass (tonnes ha^{-1})	Maturity (days)
Season	1	$\delta^2_\varepsilon+288\delta^2_b+3456\delta^2_r+10368\delta^2_s$	25323.95***	851.11**	38.37	112736.88***	6164.66**
Rep within Season	4	$\delta^2_\varepsilon+288\delta^2_b+3456\delta^2_r$	111.02	6.06	179.80	391.30	79.35
Block within Rep×Season	66	$\delta^2_\varepsilon+288\delta^2_b$	80.33	0.86	28.95	121.28	3.20
Genotype	143	$\delta^2_\varepsilon+72\delta^2_{sg}+144\delta^2_g$	852.40***	5.44***	140.79***	511.43**	696.24
Genotype × Season	143	$\delta^2_\varepsilon+72\delta^2_{sg}$	108.49***	1.27***	86.52***	308.78***	601.34***
Error	506	δ^2_ε	6.21	0.72	4.90	7.67	5.07
R^2			0.95	0.89	0.76	0.89	0.94
Cv %			6.74	7.26	13.94	24.61	4.60

*, **, ***, significant at ($P \leq 0.05$), ($P \leq 0.01$) and ($P \leq 0.001$), respectively. *Cv* - coefficient of variation.Test H= Season × Genotype and Blocks within replicates and seasons E= Random error; H=Genotype E= Season × Genotype; Test H=Replicates within season E= Blocks within replicates and Seasons; Test H=Season E= Replicates within season.

Table 3.2 ***continued…***

Source of variation	df	Expected mean squares	Yield (tonnes.ha^{-1})	Thousand kernel weight (g)	Hectoliter weight (kg.hl^{-1})	Harvest Index	Area under Disease Progress curve		
							Leaf rust	Stem rust	Yellow rust
Season	1	$\delta^2_\varepsilon+288\delta^2_b+3456\delta^2_r+10368\delta^2_s$	767.72***	347.09***	1178.29	0.01***	137937.05**	108014.01*	682420.32***
Rep within Season	4	$\delta^2_\varepsilon+288\delta^2_b+3456\delta^2_r$	7.45	1.80	116.19	0.01	4346.28	7066.35	548.98
Block within Rep×Season	66	$\delta^2_\varepsilon+288\delta^2_b$	0.96	0.61	68.82	0.00	314.61	332.41	288.98
Genotype	143	$\delta^2_\varepsilon+72\delta^2_{sg}+144\delta^2_g$	7.85**	6.22***	343.57***	0.01***	3867.02***	4103.06***	1655.90
Genotype × Season	143	$\delta^2_\varepsilon+72\delta^2_{sg}$	5.16***	1.35***	164.57***	0.00***	916.29***	964.28***	1602.60***
Error	506	δ^2_ε	0.50	0.57	6.55	0.07	15.04	15.05	15.51
R^2			0.96	0.91	0.80	0.82	0.89	0.89	0.91
Cv %			26.86	15.29	12.36	43.25	20.30	19.00	27.57

*, **, ***, significant at ($P \leq 0.05$), ($P \leq 0.01$) and ($P \leq 0.001$), respectively. *Cv* - coefficient of variation. Test H= Season × Genotype and Blocks within replicates and seasons E= Random error; H=Genotype E= Season × Genotype; Test H=Replicates within season E=Blocks within replicates and Seasons; Test H=Season E= Replicates within season.

There was significant ($p \leq 0.05$) difference of means for yield and yield components between seasons except for the grain filling period and harvest index. The plants grown during July to November season were taller (30.90%) and took longer days to mature (4.74%) than the February to July season. In addition, these plants had longer spikes (17.97%), higher biomass (53.61%), TKW (29.13%), hectoliter weight (4.32%) and yellow rust disease (99.80%) than in February-July (off-season). However, the plants took longest number of days to fill the grains (1.19%) during February to July season. Moreover, the plants possessed higher harvest index, leaf rust disease and stem rust disease than July to November season by 12.5%, 43.88% and 19.18% respectively (Table 3.3).

Table 3.3. Summary of means of disease and agronomic traits wheat genotypes evaluated against leaf rust disease at Njoro over two seasons.

Season	Plant height(cm)	Spike length(cm)	GFP (days)	Biomass (tonnes. ha^{-1})	Maturity (days)	Yield (tonnes. ha^{-1})	TKW (g)	Hectolitre weight (kg hI^{-1})	Harvest index	AUDPC		
										Leaf Rust	Stem rust	Yellow rust
Feb-Jul	75.48b	8.90b	35.32a	296.53b	107.41b	0.19b	3.09b	51.85b	0.08a	202.47a	184.93a	0.53b
Jul-Dec	109.24a	10.85a	34.90a	639.21a	112.75a	2.80a	4.36a	54.19a	0.07b	113.63b	149.46b	268.65a
$LSD_{(0.05)}$	0.83	0.10	0.65	1.03	0.68	0.07	0.08	0.88	0.01	2.01	2.01	2.07

Means followed by the same letters down the column are not significantly different at $p \leq 0.05$;AUDPC-Area under Disease Progress Curve;TKW-Thousand Kernel Weight.

3.4.3 Variation of kernel weight with yield

The Pearson correlation coefficient analysis showed that yield displayed significantly different positive correlation with a thousand kernel weight (r=0.74***) and hectoliter weight (r=0.40***). The TKW showed a significant positive correlation with hectoliter weight (r=0.56***), however, yield, TKW and hectoliter weight displayed significantly negative correlation with leaf rust (r=-0.27***, r=-0.30***, r=-0.19***) and stem rust (r=-0.19***, r=-0.22***, r=-0.19***). This is shown in Table 3.4 below.

Table 3.4. Correlation coefficient (r) among leaf rust and the traits of interest for wheat genotypes evaluated for leaf rust resistance at KALRO, Njoro, 2016.

***, significance at ($p \leq 0.001$)

			Area under Disease Progress Curve	
	Thousand Kernel Weight	Hectoliter Weight	Leaf Rust	Stem Rust
Yield	0.74***	0.40***	-0.27***	-0.19***
Thousand Kernel Weight	-	0.56***	-0.30***	-0.22***
Hectoliter Weight		-	-0.19***	-0.19***
AUDPC Leaf Rust			-	0.24***
AUDPC Stem Rust				-

3.4.4 Response of wheat genotypes for leaf rust severity and grain yield

The mean yield and AUDPC for leaf rust for the best 20 genotypes, the check variety and the least yielding wheat genotypes evaluated are presented in Table 3.5. Considering how the seasons differentiated performance of genotypes, the first season (4.76 t ha^{-1}) had lower yield than the second season. Genotypes *R1301* and *R1305* ranked the highest with; 6.51 t ha^{-1} and 5.86 t ha^{-1} mean yields across seasons, respectively. The most susceptible genotype *Marquis* had 0.06 t ha^{-1}, while the susceptible check *K. Chiriku* had 1.55 t ha^{-1}. Based on AUDPC means, genotypes *R1301* and *R1305* had lowest with means of 42.00 and 42.00, respectively.

Table 3.5. The mean yield and AUDPC for leaf rust for the best 20, the least yielder and the check of wheat (Triticum aestivum) genotypes evaluated across the two seasons at KALRO, Njoro in 2016.

		Yield (tonnes ha^{-1})			Area under Disease Progress Curve		
Genotype	Pedigree	Mean	Season 1	Season 2	mean	Season 1	Season 2
R1301	KSW/5/2*ALTAR 84/AE.SQUARROSA (221)//3*BORI95/3/URESJUN/KAUZ/4/WBLLI	6.51	0.15	12.87	42.00	28.00	56.00
R1305	KSW/5/2*ALTAR 84/AE.SQUARROSA (221)//3*BORI95/3/URESJUN/KAUZ/4/WBLLI	5.86	1.10	10.62	42.00	28.00	56.00
K. Kingbird	TAM200/TUI/6/PVN//CAR422/ANA/5/BOW/CROW//BUC/PVN/3/YR/4/TRAP#1	4.64	1.57	7.71	52.52	46.62	58.42
R1309	KFA/5/REH/HARE//2*BCN/3/CROC-I/AE.SQUARROSA(213)//PGO/4/HUITES/6/REH/HARE//2*BCN/3/CROC-I/AE.SQUARROSA(213)//PGO/4/HUITES	4.56	0.17	8.95	46.47	36.46	56.48
Means			1.25	6.01		49.61	85.85
Cv%			26.86			20.30	
$LSD_{(0.05)}$ [a]			0.56			2.01	
$LSD_{(0.05)}$ [b]			0.07			17.06	

R: Introduction, [a]: LSD for comparing means within seasons, [b]: LSD for comparing means between seasons.

Table 3.5. Continued...

Genotype	Pedigree	Yield (tonnes ha^{-1})			Area under Disease Progress Curve		
		Mean	Season 1	Season 2	mean	Season 1	Season 2
R1476	-	4.25	1.71	6.79	45.75	33.07	60.83
K. Tai	ND643/2*WBLL1	4.17	1.18	7.16	42.00	28.00	56.00
Eagle10	EMB16/CBRD//CBRD	4.11	1.02	7.20	53.30	39.49	67.11
	FROCOR*2/4/COMETA/3/ NEWTHATCH// MENTANA/	3.71	1.04	6.38	60.48	56.49	64.47
CI 14393	MENKEMEN						
	PRINIA/3/ALTAR84/AE.SQ//2*OPATA/4/CHEN/AEGILOPS	3.65	0.99	6.31	122.82	28.00	217.64
R1244	SQUARROSA (TAUS)//BCN/3/BAV92						
Means			1.25	6.01		49.61	85.85
Cv%			26.86			20.30	
$LSD_{(0.05)}$ [a]			0.56			2.01	
$LSD_{(0.05)}$ [b]			0.07			17.06	

R: Introduction, [a]: LSD for comparing means within seasons, [b]: LSD for comparing means between seasons.

Table 3.5.Continued...

Genotype	Pedigree	Yield (tonnes ha^{-1})			Area under Disease Progress Curve		
		Mean	Season 1	Season 2	mean	Season 1	Season 2
R1474	-	3.65	1.68	5.62	88.99	71.55	106.43
Ibis	KWALE/DUMA	3.61	2.25	4.97	117.05	92.78	141.32
ET-12-D4	MAMBA/UQ105	3.51	2.69	4.33	54.31	34.76	73.86
K. Nyangumi	TEZANOS-PINTOS-PRECOZ//SELKIRK-ENANO*6/LERMA-ROJO-64/3/AFRICA-MAYO-48/4/KENYA-SWARA/K-4500-6	3.41	1.20	5.62	61.60	33.07	90.13
K. Nyoka	CI-8154/2*FEDERATION//3*ROMANY	3.32	1.67	4.97	70.63	64.02	77.24
Verde	MN-7663/SBY-354-A	3.28	2.37	4.19	42.00	28.00	56.00
Zabadi	CORRECAMINOS/INIA-67//K-4500-2/3/KENYA-SWARA//TOBARI-66/CIANO-67	3.27	1.48	5.06	55.35	47.68	63.02
Means			1.25	6.01		49.61	85.85
Cv%			26.86			20.30	
$LSD_{(0.05)}$[a]			0.56			2.01	
$LSD_{(0.05)}$[b]			0.07			17.06	

R: Introduction, [a]: LSD for comparing means within seasons, [b]: LSD for comparing means between seasons.

Table 3.5.Continued...

Genotype	Pedigree	Yield (tonnes ha^{-1})			Area under Disease Progress Curve		
		Mean	Season 1	Season 2	mean	Season 1	Season 2
R1317	KSW/7/CAL/NH/H567.71/3/SERI/4/CAL/NH//H567.71/5/2*KAUZ/6/PASTOR/8/CAL/NH//H567.71/3/SERI/4/CAL/NH//H567.71/5/2*KAUZ/6/PASTOR	3.25	0.79	5.71	50.10	28.00	72.20
Tama	YAKTANA-54/LERMA-52	3.23	0.80	5.66	65.36	70.14	60.58
Kanga	-	3.18	1.64	4.72	68.93	56.09	81.77
Katar	COOK/VEE''S''//DOVE''S''/SERI/3/BJY''S''	3.12	0.98	5.26	57.73	47.61	67.85
Marquis	HARD-RED-CALCUTTA	0.06	0.00	0.12	145.63	107.29	183.97
K. Chiriku	KTB/(SIB)CARPINTERO	1.55	1.04	2.06	103.82	86.34	121.30
Means			1.25	6.01		49.61	85.85
Cv%			26.86			20.30	
$LSD_{(0.05)}$ [a]			0.56			2.01	
$LSD_{(0.05)}$ [b]			0.07			17.06	

R: Introduction, [a]: LSD for comparing means within seasons, [b]: LSD for comparing means between seasons

3.4.5 Field tests for adult plant resistance

Adult plant reactions showed a range of response level of the tested wheat genotypes to leaf rust disease. Plant reactions of the genotypes which were considered to be resistant and the check are presented in Table 3.6. It is worth to note that seven genotypes (*K. Tai, K. Korongo, Fletcher, Verder, R1244, R1305, R1301*) showed resistance response at adult stage for the two seasons.Twenty two genotypes (*K. Page, Lenana, Romany, Bounty, Plume, Sungura, Tobari 66, K. Paka, K. Tembo, K. Kingbird, Marquillo, 1061.K.4, Era, Mcvey, Morris, PWThatcher, Fronthatch, Polk, Angus, Norm, R1475, R1309*) were resistant only during the second season while, 5 genotypes (*K. Fahari, K. Wren, Minnpro, R1336, R1317*) showed resistance infection type during the first season. The remaining genotypes showed susceptibility that ranged between 5S to 90S at adult plant stage.

Table 3.6. Seedling and adult plant infection type to leaf rust (Puccinia triticina) for wheat (Triticum aestivum) genotypes that were considered resistant and a check as evaluated in greenhouse and field at KALRO, Njoro.

		Season 1				Season 2			
Genotype	Pedigree	Ist score	2nd score	FDS	AUDPC	Ist score	2nd score	FDS	AUDPC
K. Tai	ND643/2*WBLL1	0	0	0	0.0	0	0	0	0.0
K.Koron	BABAX/LR42//BABAX*2/4/SNI/TRAP#1/3/KAU								
go	Z*2/TRAP//KAUZ	0	0	0	0.0	0	0	0	0.0
	II-55-10/4/PEMBINA/II-52-329/3/II-53-388/III-58-								
Fletcher	4//II-53-546	0	0	0	0.0	0	0	0	0.0
Verder	MN-7663/SBY-354-A	0	0	0	0.0	TR	TR	TR	17.5
	PRINIA/3/ALTAR84/AE.SQ//2*OPATA/4/CHEN/								
	AEGILOPS SQUARROSA	0	0	0		0	0		
R1244	(TAUS)//BCN/3/BAV92				0.0			0	0.0
	KSW/5/2*ALTAR 84/AE.SQUARROSA								
R1305	(221)//3*BORI95/3/URESJUN/KAUZ/4/WBLLI	0	0	0	0.0	0	0	0	0.0
R1301	KSW/5/2*ALTAR 84/AE.SQUARROSA								
	(221)//3*BORI95/3/URESJUN/KAUZ/4/WBLLI	0	0	0	0.0	0	0	0	0.0

0=Immune, R= Resistant, MR=moderately resistant, MS=moderately susceptible, S=Susceptible, TR=trace resistant, MSS= moderately susceptible and susceptible (Johnston and Browder, 1966) AUDPC=Area under Disease Progress Curve; SIT=Seedling Infection Type, FDS= Final Disease Severity.0, 0;, 1, 2 = resistance response, 3 and 4 = susceptibility response

Table 3.6 continued...

Genotype	Pedigree	Season 1				Season 2			
		Ist Score	2nd score	FDS	AUDPC	Ist score	2nd score	FDS	AUDPC
K. Page	MENTANA/KENYA-58//BAGE/3/KENYA-184-P	5MS	20S	40S	437.5	0	0	0	0.0
Lenana	YAQUI- 48 / KENTANA- 48	5S	20S	40S	437.5	0	0	0	0.0
Romany	COLOTANA 261-51 / YAKTANA 54A	5MS	10S	20S	227.5	0	0	0	0.0
Bounty	TIMSTEIN/2*KENYA//BONZA	TR	5S	10S	108.5	0	0	0	0.0
Plume	MIDA/MCMURACHY//EXCHANGE/3/KENYA-184-P	0	5S	5S	70.0	0	0	0	0.0
Sungura	ID 1877/MORRIS	0	10S	15S	175.0	0	0	0	0.0
Fronthatch	FRONTANA / KENYA58 // NEWTHATCH	0	5S	5S	70.0	0	0	0	0.0
Polk	THATCHER / SUPREZA /3/ KENYA 58 / NEWTHATCH // FRONTANA	0	15S	15S	210.0	0	0	0	0.0
Norm	MN-73167/MN-81070	0	5S	5S	70.0	0	0	0	0.0
Tobari 66	TEZANOS-PINTOS-PRECOZ/SONORA-64-A	0	10S	10S	140.0	TR	TR	TR	17.5
R1475	-	TR	30S	30S	423.5	0	0	0	0.0

0=Immune, R= Resistant, MR=moderately resistant, MS=moderately susceptible, S=Susceptible, TR=trace resistant, MSS= moderately susceptible and susceptible (Johnston and Browder, 1966) AUDPC=Area under Disease Progress Curve; SIT=Seedling Infection Type, FDS= Final Disease Severity.0, 0;, 1, 2 = resistance response, 3 and 4 = susceptibility response

Table 3.6. Continued...

		Season 1				Season 2			
Genotype	Pedigree	Ist score	2nd score	FDS	AUDPC	Ist score	2nd score	FDS	AUDPC
Angus	THATCHER/2*SUPREZA/3/FRONTANA//KENY58/NEWTHATCH/7/PEMBINA//FRONTANA/5*THATCHER/6/MIDA//KENYA-117-A/2*THATCHER/3/FRONTANA/4*THATCHER/4/MN-III-58-4/5/KENYA-58/NEWTHATCH//3*LEE	0	0	5S	35.0	0	0	0	0.0
R1309	KFA/5/REH/HARE//2*BCN/3/CROC-I/AE.SQUARROSA(213)//PGO/4/HUITES/6/REH/HARE//2*BCN/3/CROC-I/AE.SQUARROSA(213)//PGO/4/HUITES	5MS	5MS	5MS	87.5	0	0	0	0.0
K. Paka	WISCONSIN-245/II-50-17//CI-8154/2*TOBARI-66	5MS	15S	30S	332.5	TR	TR	TR	17.5
K.Kingbird	TAM200/TUI/6/PVN//CAR422/ANA/5/BOW/CROW//BUC/PVN/3/YR/4/TRAP#1	0	20S	20S	280.0	TR	TR	TR	17.5

0=Immune, R= Resistant, MR=moderately resistant, MS=moderately susceptible, S=Susceptible, TR=trace resistant, MSS= moderately susceptible and susceptible (Johnston and Browder, 1966) AUDPC=Area under Disease Progress Curve; SIT=Seedling Infection Type, FDS= Final Disease Severity.0, 0;, 1, 2 = resistance response, 3 and 4 = susceptibility response

Table 3.6. Continued…

		Season 1				Season 2			
Genotype	Pedigree	Ist Score	2nd score	FDS	AUDPC	Ist score	2nd score	FDS	AUDPC
Marquillo	MARQUIS/(TR.DR)IUMILLO	0	5S	5S	70.0	TR	TR	TR	17.5
Era	II-55-10/4/PEMBINA/II-52-329/3/II-53-388/III-58-4//II-53-546	0	5S	5S	70.0	0	TR	TR	14.0
Mcvey	NING-8331/MN-87029//MN-89068	0	5S	10S	105.0	TR	TR	TR	17.5
Morris	THATCHER//KENYA-117 A/MIDA/3/FRONTANA/4*THATCHER/4/THATCHER/5/FRONTANA/4*THATCHER	0	5S	10S	105.0	0	TR	TR	14.0
PWThatcher	THATCHER/AGENT	5MS	10S	20S	227.5	TR	TR	TR	17.5
K. Fahari	TOBARI-66/3/SRPC-527-67//CI-8154/2*FROCOR	0	0	0	0.0	0	0	20MS S	140.0
K.wren	THELIN#2/TUKURU	0	0	0	0.0	5S	5S	5S	87.5
Minnpro	MN-72299/MN-74115	0	0	0	0.0	5S	5S	5S	87.5
R1336	BABAX/LR42//BABAX*2/3/TUKURU	0	0	0	0.0	5S	10S	10S	157.5
K. Chiriku	KTB/(SIB)CARPINTERO	10S	30S	50S	595.0	10MS	40S	40S	595.0

0=Immune, R= Resistant, MR=moderately resistant, MS=moderately susceptible, S=Susceptible, TR=trace resistant, MSS= moderately susceptible and susceptible (Johnston and Browder, 1966) AUDPC=Area under Disease Progress Curve; SIT=Seedling Infection Type, FDS= Final Disease Severity.0, 0;, 1, 2 = resistance response, 3 and 4 = susceptibility response

Table 3.6. Continued…

Genotype	Pedigree	Season 1				Season 2			
		Ist score	2nd score	FDS	AUDPC	Ist score	2nd score	FDS	AUDPC
R1317	KSW/7/CAL/NH/H567.71/3/SERI/4/CAL/NH//H567.71/5/2*KAUZ/6/PASTOR/8/CAL/NH//H567.71/3/SERI/4/CAL/NH//H567.71/5/2*KAUZ/6/PASTOR	0	0	0	0.0	5S	5S	5S	87.5

0=Immune, R= Resistant, MR=moderately resistant, MS=moderately susceptible, S=Susceptible, TR=trace resistant, MSS= moderately susceptible and susceptible (Johnston and Browder, 1966) AUDPC=Area under Disease Progress Curve; SIT=Seedling Infection Type, FDS= Final Disease Severity.0, 0;, 1, 2 = resistance response, 3 and 4 = susceptibility response

3.4.6 Stepwise multiple regression analysis

From the stepwise regression analysis, leaf rust had highest contribution to hectoliter weight (R^2=0.085) reduction. In addition, leaf rust infection contributed to the grain yield (R^2=0.218) and TKW (R^2=0.133) reduction. However, yellow rust infection was detected as a major cause of grain yield (R^2=0.136) reduction. On the other hand, stem rust infection had the greatest effect on reduction on TKW (R^2=0.084) (Table 3.7).

Table 3.7. Stepwise multiple regression analysis showing effects of leaf rust, stem rust and yellow rust on grain yield, thousand kernel weight and Hectoliter weight on wheat genotype tested at KALRO, Njoro in 201

	Variable	Parameter	Standard error	*C(P)*	Partial R^2	Model R^2
Yield	Intercept	2.88509	0.16829			
	AUDPC Yellow Rust	-0.00299	0.00071	25.3588	0.13640	0.13640
	AUDPC Leaf rust	-0.00201	0.00056	11.8719	0.08140	0.21780
	AUDPC Stem rust	-0.00149	0.00047	4.0000	0.05190	0.26960
TKW	Intercept	4.40726	0.15966			
	AUDPC Stem rust	-0.00141	0.00045	13.4171	0.08420	0.08420
	AUDPC Leaf rust	-0.00135	0.00053	7.3556	0.04840	0.13260
	AUDPC Yellow rust	-0.00156	0.00067	4.000	0.03220	0.16480
Hectoliter weight	Intercept	57.40115	1.24031			
	AUDPC Leaf rust	-0.01312	0.00411	9.4308	0.08470	0.08470
	AUDPC Stem rust	-0.00913	0.00349	3.7879	0.04710	0.13190
	AUDPC Yellow rust	-0.00699	0.00522	4.0000	0.01100	0.14290

AUDPC-Area under Disease Progress Curve

3.5 Discussion

The significant variation due to season for most of the parameters suggests environmental variations between the two seasons when the experiment was conducted. This significant difference could be attributed to variability in availability of temperature, and moisture among other environmental factors. The present results agree with those of Milan et al. (2015) who reported that the season was mainly responsible for variation of the agronomic traits in two-rowed winter malting barley. The significant effects due to genotype for agronomic traits, yield and yield components as well as rust diseases implies that these traits are affected by the genetic make-up of a given genotype either directly or indirectly. The results are in tandem with Yan *et al*. (2010) who did a different research on soybean and reported that genotypic effects were significant for all agronomic traits. Similarly, significant effects due to the interaction between season and genotype for all the parameters could be an indication that the genotypes used were not consistent between seasons probably due to environmental influence to the genotypes for given specific trait. This is in consistency with Bhatta (2015) who reported that interaction between season and genotype effects explained the variation in grain yield, hectoliter weight, days to heading, plant height, harvest index, and TKW on winter wheat.

Despite the heavy leaf rust disease pressure in the field during the two seasons, some lines remained resistant. Among the 144 wheat genotypes screened, 7 genotypes (K. Tai, K. Korongo, Fletcher, Verder, R1244, R1305, R1301) exhibited adult plant resistance during season one and season two. The avirulence of the leaf rust at adult plant stage in these genotypes revealed the presence of minor resistance genes. Parlevliet (2001) found out that seedling resistance is under the control of major genes which provides resistance at all stages of plant growth while adult plant resistance is under control of minor genes. Variations in the expression of resistance genes in adult plant stages could suggest that there was presence of gene diversity among evaluated genotypes. The results are in agreement with Newcomb *et al*. (2013) who did a different research on stem rust. Eleven genotypes showed trace infection responses at adult stage for leaf rust. The trace reaction could be associated with hypersensitive reaction whereby fungal infection signals a defense mechanism leading to cell collapse which restricts further disease spread as reported by Rubiales and Nicks, 2000.

Slow rusting has been shown to be more durable than major seedling resistance according to Singh *et al*. (2001) and a combination of adult plant resistant gene Lr34 and several minor genes have resulted in a high level of non-specific resistance in some cultivars (Navabi *et al*., 2005). These results may add a depth of their resistance to be exploited as

good source of resistance. Furthermore, resistance expression depends on the environmental conditions, plant growth stage, host-parasite interaction, and the interaction between resistance genes in wheat genome (Kolmer, 2005). The genes in the resistant genotypes may be deployed singly or in combination into high yielding genotypes to develop high yielding and resistant wheat genotypes. Additionally, new sources of resistance in wheat genotypes could be incorporated into wheat to improve the diversity of the existing gene pool for leaf rust resistance. Durable rust resistance mechanism in wheat is achieved through introgression of resistant minor genes which seems to be more appropriate solution for sustainable wheat production (Singh *et al*., 2000).

The significant variation due to season for the means of agronomic traits, yield, grain quality, leaf rust, stem rust and yellow rust severity suggested seasonal variations between the two seasons in which the field experiment was conducted. The warm moist conditions experienced during season one favored stem rust and leaf rust infection hence, the high AUDPC for the two diseases. The effects of leaf rust on grain yield varied across seasons. For instance, in the first season, leaf rust infection contributed to the higher reduction of grain yield and TKW compared to the second season when leaf rust infection was minimal. In a different study on barley, Ochoa and Parlevliet (2007) found out that yield loss due to leaf rust was related to AUDPC. Some wheat genotypes with high yellow rust disease severity had low leaf rust severities in this study. A report by Bancal et al. (2007) also highlighted that, due to the reduced photosynthetic area for stem rust fungus infection and spread, some wheat lines with high yellow rust disease severity tended to show low stem rust severities.

Inverse relation was present between the disease level and grain yield and this implies that, leaf rust disease directly affects the kernel quality leading to shriveling of wheat grains; for example, Marquis which had the least TKW and grain yield value was totally susceptible to the leaf rust. Marquis had very shriveled kernels in the field and in some plants there were no kernels at all implying that leaf rust negatively affected the kernel quality and quantity. These results are consistent with those of Nzuve *et al*. (2012) who did a research on resistance of bread wheat to stem rust.

The positive correlation between grain yield, TKW and hectoliter weight is an indication that the yield components is largely responsible for the determination of grain yield in individual plants. Similarly, in a different study on rice (*Oryza sativa* L.) Mirza *et al*. (1992) found that the number of grains per panicle was positively correlated with panicle length, TKW and grain yield. It was observed that TKW was affected by leaf rust infection

and could be used to estimate loss in yield due to leaf rust infection. Such results are in agreement with those of Draz *et al*. (2015).

Grain weight is a crucial trait and of primary importance in determining wheat yield. Genotypes with larger grain weight value tend to have longer grain filling period, resulting in higher assimilate accumulation and heavier grain weight. Thus, genotype R1301 had the highest grain weight among the evaluated genotypes and it possessed longest grain filling period as opposed to Marquis which had the least grain weight and shortest grain filling period. Grain weight is determined by the source capacity (photosynthetic leaves) to supply assimilate during the ripening period, and by sink capacity (developing grain) to accumulate the imported assimilate (Ntanos and Koutroubas, 2002).The significant variation due to season for most of the parameters suggests environmental variations between the two seasons when the experiment was conducted. This significant difference could be attributed to variability in availability of temperature, and moisture among other environmental factors. The present results agree with those of Milan *et al*. (2015) who reported that the season was mainly responsible for variation of the agronomic traits in two-rowed winter malting barley.

The significant effects due to genotype for agronomic traits, yield and yield components as well as rust diseases implies that these traits are affected by the genetic make-up of a given genotype either directly or indirectly. The results are in tandem with Yan *et al*. (2010) who did a different research on soybean and reported that genotypic effects were significant for all agronomic traits. Similarly, significant effects due to the interaction between season and genotype for all the parameters could be an indication that the genotypes used were not consistent between seasons probably due to environmental influence to the genotypes for given specific trait. This is in consistency with Bhatta, (2015) who reported that interaction between season and genotype effects explained the variation in grain yield, hectoliter weight, days to heading, plant height, harvest index, and TKW on winter wheat.

Despite the heavy leaf rust disease pressure in the field during the two seasons, some lines remained resistant. Among the 144 wheat genotypes screened, 7genotypes (*K. Tai, K. Korongo, Fletcher, Verder, R1244, R1301, R1305*) exhibited adult plant resistance during the two seasons. The avirulence of the leaf rust at adult stage in these genotypes revealed the presence of minor resistance genes. Parlevliet, 2001 found out that adult plant resistance is under control of minor genes. Variations in the expression of resistance genes in adult stages could suggest that there was presence of gene diversity among evaluated genotypes. The results are in agreement with Newcomb *et al*. (2013) who did a different research on stem rust.

Cultivars lacking leaf rust seedling resistance genes may have additional additive minor genes that contribute to low disease pressure in the field (Hysing *et al*., 2006). Slow rusting has been shown to be more durable than major seedling resistance according to Singh *et al*. (2001) and a combination of adult plant resistant gene *Lr34* and several addition minor genes have resulted in a high level of non-specific resistance in some cultivars (Navabi *et al*., 2005). These results may add a depth of their resistance to be exploited as good source of resistance. Furthermore, resistance expression depends on the environmental conditions, plant growth stage, host-parasite interaction, and the interaction between resistance genes in wheat genome (Kolmer, 2005). The genes in the resistant genotypes may be deployed singly or in combination into high yielding genotypes to develop resistant high-yielding wheat genotypes. In addition, new sources of resistance in wheat genotypes could be incorporated into wheat to improve the diversity of the existing gene pool for leaf rust resistance. Durable rust resistance mechanism in wheat is achieved through introgression of several minor genes which are resistant thereby, affering a more sustainable wheat production method (Singh *et al*., 2000).

The significant variation due to season for the means of agronomic traits, yield, grain quality, leaf rust, stem rust and yellow rust severity suggested seasonal variations between the two seasons in which the field experiment was conducted. The warm moist conditions experienced during season one favored stem rust and leaf rust infection hence, the high AUDPC for the two diseases. The effects of leaf rust on grain yield varied across seasons. For instance, in the first season, leaf rust infection contributed to the higher reduction of grain yield and TKW compared to the second season when leaf rust infection was minimal. In a different study on barley, Ochoa and Parlevliet. (2007) found out that yield loss due to leaf rust was related to AUDPC. Some wheat genotypes with high yellow rust disease severity had low leaf rust severities in this study. A report by Bancal *et al*. (2007) also highlighted that, due to the reduced photosynthetic area for stem rust fungus infection and spread, some wheat lines with high yellow rust disease severity tended to show low stem rust severities.

Inverse relation was present between the disease level and grain yield and this implies that, leaf rust disease directly affects the kernel quality leading to shriveling of wheat grains; for example, *Marquis* which had the least TKW and grain yield value was totally susceptible to the leaf rust. Marquis had very shriveled kernels in the field and in some plants there were no kernels at all implying that leaf rust negatively affected the kernel quality and quantity. These results are consistent with those of Nzuve *et al*. (2012) who did a research on resistance of bread wheat to stem rust.

The positive correlation between grain yield, TKW and hectoliter weight is an indication that the yield components is largely responsible for the determination of grain yield in individual plants. Similarly, in a different study on rice (*Oryza sativa* L.) Mirza *et al*. (1992) found that the number of grains per panicle was positively correlated with panicle length, TKW and grain yield. It was observed that TKW was affected by leaf rust infection and could be used to estimate loss in yield due to leaf rust infection. Such results are in agreement with those of Draz *et al*. (2015).

Grain weight is a crucial trait and of primary importance in determining wheat yield. Genotypes with larger grain weight value tend to have longer grain filling period, resulting in higher assimilate accumulation and heavier grain weight. Thus, genotype *R1301* had the highest grain weight among the evaluated genotypes and it possessed longest grain filling period as opposed to *Marquis* which had the least grain weight and shortest grain filling period. Grain weight is determined by the source capacity (photosynthetic leaves) to supply assimilate during the ripening period, and by sink capacity (developing grain) to accumulate the imported assimilate (Ntanos and Koutroubas, 2002).

The results based on stepwise regression underline the effect of the three rust diseases in explaining grain yield, TKW and hectoliter weight variability in wheat. The three rust diseases contributed in reduction of yield components differently, for instance, leaf rust was the disease that reduced hectoliter weight most while, stem rust and yellow rust were leading in reduction of TKW and grain yield respectively. These results imply that, the observed reduction in wheat yield is attributed not only to leaf rust disease but also the other foliar diseases like stem and yellow rust. Similarly, in different study on septoria leaf blotch Mojerlou *et al*. (2009) showed that AUDPC explained 95% variation against yield loss in wheat.

3.5 Conclusion

Despite the high disease pressure in regard to the most susceptible genotype level of resistance (90%), this study identified potential sources of adult plant resistance such as *K. Tai, K. Korongo, Fletcher, Verder, R1244, R1305 and R1301* against leaf rust disease. Furthermore, *R1301* and *R1305* had the least leaf rust severity and ranked the best yielders across seasons. Such genotypes could be used for breeding wheat genotypes with higher levels of resistance and negligible yield losses. The genotypes identified with combination of good agronomic traits and elite sources of resistance to leaf rust should urgently be integrated in the wheat breeding programmes to improve on leaf rust resistance. This could be achieved through introgression of the genes from the identified resistant genotypes into the adapted but susceptible Kenyan wheat genotypes through intercrosses with other genotypes containing minor genes. In addition, the outstanding genotypes can be evaluated in other locations to determine their disease and yield stability before release.

CHAPETR FOUR

EVALUATION OF KENYAN WHEAT (*Triticum aestimum*. L) GENOTYPES FOR LEAF RUST (*Puccinia triticina* Erik) AT SEEDLING STAGE

4.1 Abstract

Leaf rust caused by *Puccinia triticina* Eriks. is one of the main diseases of wheat (*Triticum aestivum* L.) in Kenya, causing up to 70% of yield losses. The objective of this study was to determine genotypic variation for resistance to leaf rust among Kenyan wheat genotypes at seedling stage. One hundred and forty-four wheat genotypes were planted and inoculated in the greenhouse at Kenya Agricultural and Livestock Research Organization (KALRO), Njoro. The test genotypes were inoculated with leaf rust urediniospores and evaluated for infection types at two leaf stage. Among the genotypes tested, 79 exhibited seedling resistance response rated "0" to "2" infection types while the remaining genotypes exhibited susceptible response with infection types "3" and "4". The identified sources of resistance in wheat genotypes could be incorporated into wheat to increase the diversity of the existing gene pool for leaf rust resistance.

Key words; Wheat resistance, *Puccinia triticina,* Seedling stage

4.2 Introduction

Wheat (Triticum aestivum L.) is a host for three rust diseases, stripe, leaf and stem rust. Leaf rust disease is considered the most common and widely distributed of the three wheat rusts and has become more serious problem of wheat causing great losses in grain yield (Huerta-Espino *et al*., 2011). Genetic resistance is the most economic and effective means of reducing yield losses caused by the disease. However, breeding genotypes for disease resistance is a continuous process and plant breeders need to add new effective sources to their breeding materials. Genetic diversity of plants determines their potential for improved efficiency and hence their use for breeding, which eventually may result in enhanced food production (Ormoli *et al*., 2015). Most resistance genes in sime wheat genotypes that are effective in seedling plants remain effective throughout the adult plant stage. For instance, *Lr1*, *Lr10*, *Lr21* and *Lr42* are good examples of race specific resistance genes that are effective both at seedling and adult stage (Huerta-Espino *et al*., 1998). Knowledge on the identity of such resistance genes in released cultivars is essential for the incorporation of effective resistance into breeding programmes to maintain a diversity of resistance genes in commonly grown wheat cultivars (Purnhauser *et al*., 2011).

Resistance gene, *Lr34* is among the adult plant resistance (APR) genes that have been isolated, characterized and is the most essential both in terms of stability and prevalent (Kolmer, 1996). This gene often can interact with seedling resistance genes in seedling plants to provide lower than the expected infection types (Zang *et al*., 2008). Major genes such as *Lr24/Sr24, Sr26* and polygenic resistance have been used successfully to control cereal rust diseases in Australia (Park, 2008). Combining race-specific and race-nonspecific genes in one genotype ensures more durable resistance than that based on single seedling resistance gene. The use of resistant wheat genotypes is the most economical and known to be environmentally friendly method of controlling the disease, besides the reduction of costs of fungicides applied (Martinez *et al*., 2001). However, host resistance conferred by a single or a few genes could be easily overcome by emergence of new races (McDonald and Linde, 2002).

Virulence in the pathogen population has been evolving rapidly following the deployment of many of these resistance genes, thus, necessitating a constant search and transfer of the new and effective sources of rust resistance. Most of the 60 catalogued leaf rust resistance genes confer race-specific resistance in a gene-for-gene manner (McIntosh *et al*., 2007). However, wheat varieties relying on race-specific resistance often lose effectiveness within a few years by imposing selection for virulent leaf rust races. A number of genes such as *Lr9, Lr19* and *Lr24*, are effective against most of the pathotypes of leaf rust, and are available in the improved genotypes, but sometimes, these resistant genes lack durability (Purnima *et al*., 2012). Thus, the short lived nature of race-specific hypersensitive response has created the necessity to search for more durable type of resistance. The objective of this study was to determine genotypic variation for resistance to leaf rust among Kenyan wheat genotypes at seedling stage.

4.3 Materials and methods

3.3.1 Experimental site

The study on virulence of leaf rust disease to different wheat genotypes at seedling stage was conducted in the greenhouse at Kenya Agricultural and Livestock Research Organization (KALRO)-Njoro (0° 20´S, 35° 56´E), 2185 meters above sea level.

3.3.3 Wheat genotypes

The plant materials (144 wheat genotypes) used in adult plant test in the field (Chapter three) were evaluated for seedling resistance in the greenhouse.

4.3.4 Experimental procedure

a) Collection of rust samples and inoculation of seedlings

The bulk inoculum was collected randomly from infected leaves from the International wheat screening nursery, Njoro. Five seeds from each of the 144 wheat genotypes were planted in square pots measuring 5 × 5 cm filled with about 46 g of vermiculite. Urediniospores of leaf rust were suspended in a solution of distilled water and approximately 1mg L^{-1} *Tween 20* (surfactant).

The solution with inoculum was sprayed onto the seedlings at GS12 (two leaves stage) from a distance of 50-80 cm as a fine mist using a hand sprayer. The inoculated plants were then placed in the dew chamber for 24 hours at a temperature ranging from 16 °C–18 °C. Seedlings were transferred to growth chamber maintained at a temperature ranging from 18 °C–25 °C and 80-100% relative humidity (RH) until the disease was set on the seedlings. The seedlings were evaluated for the infection type after 14 days post-inoculation.

b) Assessment of leaf rust disease

Response of seedlings to leaf rust infection were evaluated two weeks post inoculation based on the infection types (ITs) expressed on each entry. The infection types of *Puccinia triticina* were quantified using a standard 0 to 4 scale, Where: 0 = no uredinia or other macroscopic signs of infection; 0; = no uredinia, but hypersensitive necrotic or chlorotic flecks present; 1 = small uredinia surrounded by necrosis; 2 = small to medium uredinia surrounded by chlorosis or necrosis; 3 = medium-sized uredinia that may be associated with chlorosis; 4 = large uredinia without chlorosis or necrosis; X= heterogeneous infection types; + = slightly larger uredinia than expected for the infection type; - = slightly smaller uredinia than expected for the infection type (Johnston and Browder,1966).

4.4 Results

A range of infection types showing resistance and susceptibility were observed on seedlings of wheat genotypes (Table 4). Seventy-nine out of 144 genotypes exhibited resistance response rated ";","1" and "2" infection types at seedling stage. Among resistant genotypes, 5 genotypes *Africa Mayo, Eagle 10, Gabrino, R1244* and *R1336* were marked as having high level of resistance (";") while 74 genotypes had resistance infection types of "1","1^{-}", "1^{+}", "2", "2^{-}" ,"2^{+}" (or the combinations of either two).The remaining genotypes including the standard check variety K. *Chiriki* exhibited susceptible response with infection types "3" and "4".

4.5 Discussion

The disease infection types varied from the resistance to susceptible at seedling growth stage.The variations in the expression of resistance genes in seedling could suggest that there was presence of gene diversity among evaluated genotypes. The results are in agreement with Newcomb *et al*. (2013) who did a different research on stem rust. Seventy nine of the genotypes which displayed resistant infection types at seedling stages indicated that, the major genes were present. Parlevliet (2001) found out that seedling resistance is under the control of major genes which provides resistance at all stages of plant growth. Five of the resistant genotypes showed fleck infection responses leaf rust. The fleck reaction could be associated with hypersensitive reaction whereby fungal infection signals a defense mechanism leading to cell collapse which restricts further disease spread as determined by Rubiales and Nicks (2000).

Resistance shown by genotypes at seedling stage meant that most of the genotypes evaluated had major genes (Singh *et al*., 2013). Cultivars lacking leaf rust seedling resistance genes may have additional additive minor genes that would contribute to low disease pressure when evaluated in the field (Hysing *et al*., 2006). Although Adult plant resistance has been shown to be more durable than seedling resistance (Singh *et al*., 2001), a combination of adult plant resistant genes for example, *Lr34* and several addition minor genes have resulted in a high level of non-specific resistance in some cultivars (Navabi *et al*., 2005). The genes in the resistant genotypes may be deployed into high yielding genotypes to develop resistant high-yielding wheat genotypes. The present study identified new sources of resistance that can be incorporated into wheat to escape heavy yield losses caused by the leaf rust disease.

4.6 Conclusion

It could be concluded that, 54.86% of the evaluated genotypes were resistant at seedling stage, however, *Africa Mayo, Eagle 10, Gabrino, R1244* and *R1336* had highest level of resistance. Cultivation of such resistant genotypes can be of paramount importance in reduction of yield losses caused by leaf rust. Furthermore, team work between plant breeders' and pathologists should be encouraged as well as accounted for to continuously monitor rust situation and evolve resistant varieties to ensure food security of Kenya.

Table 4.1 Evaluation of 144 Kenyan wheat genotypes for seedling plant resistance against leaf rust in Njoro over two seasons.

GENOTYPE		PEDIGREE	SEEDLING INFECTION TYPES
Kentana 48	(1948)	KENYA-C-9906/MENTANA	3^{+} 4
Rhodesian sabanero	(1949)	(S)SABANERO	1^{-} 2^{-}
Kenya -184-P	(1951)	RELIANCE/KENYA-73-D	3^{+} 4^{+}
Africa Mayo	(1960)	AFRICA/MAYO-48	;
Mbega	(1963)	BONANZA/YECORA-70/3/F-35-75//KALYANSONA/BLUEBIRD	1^{-} 2^{-}
Tama	(1963)	YAKTANA-54/LERMA-52	1^{-}
K. Page	(1963)	MENTANA/KENYA-58//BAGE/3/KENYA-184-P	1^{-} 2^{-}
Lenana	(1963)	YAQUI- 48 / KENTANA- 48	3^{+}
Kenya Civet	(1966)	CI 12632 /3* KENYA 354	3^{+} 4
Kudu	(1966)	KENYA-131/KENYA-184-P	4^{-}
K. Leopard	(1966)	LAGAEDINHI /3* KENYA 381P // CI 12632 /3* KENYA 354P	3^{-} 4
Romany	(1966)	COLOTANA 261-51 / YAKTANA 54A	1^{-}
Token-Ken	(1966)	TIMSTEIN/2*KENYA//YAQUI-50	1^{-}
Bounty	(1966)	TIMSTEIN/2*KENYA//BONZA	3^{-} 4^{-}
Tobari 66	(1966)	TEZANOS-PINTOS-PRECOZ/SONORA-64-A	3^{+} 4
Plume	(1966)	MIDA/MCMURACHY//EXCHANGE/3/KENYA-184-P	3^{-} 4^{-}
Grange	(1966)	KENYA-360-F/GRANADERO-KLEIN	3^{+}
Trophy	(1968)	TIMSTEIN/2*KENYA-RF-324//2*YAQUI-50	1^{-}
Sungura	(1969)	ID 1877/MORRIS	1^{+} 2^{+}
Nyati	(1973)	AFRICA-MAYO/2*ROMANY	2^{-}
Enkoy	(1974)	HEBRAND SEL/WISCONSIN 245/SUPRESA/3/2*FROCOR//FRONTANA/YAQUI/4/AGUILERA, KENYA 4500 L6A4	1^{+} 2^{+}
K. Paka	(1975)	WISCONSIN-245/II-50-17//CI-8154/2*TOBARI-66	3^{+} 4
K. Nyoka	(1975)	CI-8154/2*FEDERATION//3*ROMANY	1^{-} 2^{-}
K. Tembo	(1975)	WISCONSIN-245/II-50-17//CI-8154/2*TOBARI-66	3^{+} 4
K. Kifaru	(1976)	WIS.245/II-50-17//CI8154/2*FR/3/3*TOB66	1^{+}

Table 4.1 continued

GENOTYPE		PEDIGREE	SEEDLING INFECTION TYPES
K. Nyangumi	(1979)	TEZANOS-PINTOS-PRECOZ//SELKIRK-ENANO*6/LERMA-ROJO-64/3/AFRICA-MAYO-48/4/KENYA-SWARA/K-4500-6	1^-
K. Fahari	(1977)	TOBARI-66/3/SRPC-527-67//CI-8154/2*FROCOR	2^+
Zabadi	(1979)	CORRECAMINOS/INIA-67//K-4500-2/3/KENYA-SWARA//TOBARI-66/CIANO-67	1^+ 2^+
K. Kongoni	(1981)	CI-8154/2*FROCOR//3*ROMANY/4/WISCONSIN-245/II-50-17/CI-8154//2*FROCOR/3/TOBARI-66	3^+ 4
K. Popo	(1982)	KLEIN-ATLAS/TOBARI-66//CENTRIFEN/3/BLUEBIRD/4/KENYA-K. FAHARI	3^+ 4
KKBB	(1982)	KAVKAZ/KALYANSONA/BLUEBIRD	2^+
Kenya Tumbili	(1984)	KTB/GIZA-155//NADADORES-63/T-238-1-5-8-17-10/3/KLEIN-ATLAS/TOBARI-66//CENTRIFEN/BLUEBIRD	1^+ 2^+
Kwale	(1987)	KAVKAZ/3/SONORA 64/CIANO F 67//INIA F 66/4/MAYA 74//BLUEBIRD/INIA F 66	1^+ 2^+
Mbuni	(1987)	ZARAGOZA-75/3/LD-357-E/THATCHER//GALLO	1^- 2^-
Pasa	(1989)	BUCK BUCK/CHAT	2^-
K. Tai	(1969)	ND643/2*WBLL1	1^- 2^-
Ngamia	(1993)	BUCKY/MAYA-74/4/BLUEBIRD//HD-832/OLESENS DWARF/3/CIANO 67/PENJAMO 62	3^- 4
Duma	(1993)	AURORA/UP301//GALLO/SUPER X/3/PEWEE/4/MAIPO/MAYA 74//PEWEE	1^-
K. Chiriku	(1989)	KTB/(SIB)CARPINTERO	3^+ 4
Heroe	(1998)	MBUNI/SRPC-64//YRPC-1	1^-
Yombi	(1998)	MBUNI/SRPC-64//YRPC-5	2^+
Simba	(2000)	PARULA/VEERY #6//MYNA/VULTURE	3^+ 4^-
Njoro Bw II	(2007)	IAS-58/4/KALYANSONA/BLUEBIRD//CAJEME-F-71/3/ALONDRA/5/BOBWHITE	3^- 4^-

Table 4.1 continued

GENOTYPE		PEDIGREE	SEEDLING INFECTION TYPES
Ibis	(2008)	KWALE/DUMA	3^{+} 4
Eagle10	(2011)	EMB16/CBRD//CBRD	;
Robin	(2011)	BABAX/LR42//BABAX*2/3/TUKURU	1^{-}
K. Sunbird	(2012)	ND643/2*WBLL1	3^{+}
K.wren	(2012)	THELIN#2/TUKURU	1^{-}
K..Kingbird	(2012)	TAM200/TUI/6/PVN//CAR422/ANA/5/BOW/CROW//BUC/PVN/3/YR/4/TRAP#1	1^{-} 2^{-}
K.Korongo	(2012)	BABAX/LR42//BABAX*2/4/SNI/TRAP#1/3/KAUZ*2/TRAP//KAUZ	1^{-}
Kenya-294-B-2 A-3	(-)	AUSTRALIAN-26-A/KENYA-117-A	1^{-} 2^{-}
Kenya 155	(-)	-	1^{-}
Reliance 261M	(-)	RELIANCE / KENYA 68	3
Kenya-318-AJ-4 A-1	(-)	KENYA-112/CERES	3^{-}
Kenya 6820	(-)	-	2^{+}
Cheetah	(-)	WARIGO/STERLING	3^{-}
Kanga	(-)	-	1^{+}
Kenya 8	(-)	-	1^{+} 2^{+}
Kenya-122	(-)	MARQUIS/AGUILERA 8	1^{+}
K.hawk	(-)		1^{-}
Morocco	(-)		3^{+} 4
Marquis		HARD-RED-CALCUTTA	3^{+} 4
Marquillo	(1926)	MARQUIS/(TR.DR)IUMILLO	3^{+} 4
Thatcher	(1934)	MARQUIS/(TR.DR)IUMILLO//MARQUIS/KANRED	3^{+} 4
Regent	(1939)	H44/REWARD	3^{+}
Newthatch	(1944)	HOPE/THATCHER//2*THATCHER	1^{+}
Yaqui 50	(1950)	NEWTHATCH/MARROQUI-588	3^{-}
Yaktana 54A	(1954)	YAQUI-48/KENTANA-48//FRONTANA	3^{+}

Table 4.1 continue…

GENOTYPE		PEDIGREE	SEEDLING INFECTION TYPES
Justin	(1962)	CONLEY/ND-40-2	3^- 4^-
Gabrino	(1963)	KENTANA/RIO-NEGRO//GABO-54	;
Bonza	(1963)	YAQUI-50/KENTANA-48	3^+ 4
Menco	(1963)	MENTANA / KENYA // FRONTANA / CINCO	1^+ 2^+
Salmayo	(1963)	SALLES/MCMURACHY//MAYO-48	2^-
Catcher	(1963)	THATCHER/SANTA-CATALINA//FROCOR	3^-
Frontana	(1963)	FRONTEIRA/MENTANA	2^+
Tama	(1963)	YAKTANA-54/LERMA-52	3^-
Gem	(1964)	BT908 / FRONTANA // CAJEME 54	3^+
Fronthatch	(1964)	FRONTANA / KENYA58 // NEWTHATCH	2^-
Pewter	(1964)	PW-327,USA/5*THATCHER	1^-
Fury	(1964)	FROCOR/MENTANA/KENYA-2/MCMURACHY/YAQUI-50	1^- 2^-
Chris	(1965)	FRONTANA/3*THATCHER/3/KENYA-58/NEWTHATCH//2*THATCHER	3^+ 4
Bailey	(1966)	FRONTANA/4*THATCHER/3/THATCHER//KENYA58/NEWTHATCH/4/THATCHER/5/FRONTANA/4*THATCHER	1^- 2^-
Goblet	(1967)	GABO-54/LERMA-52//GABO/3/KENYA/GENERAL-URQUIZA	1^+ 2^+
Ciano F67	(1967)	PITIC-62/(SIB)CHRIS//SONORA-64	1^-
II-50-17	(1967)	FRONTANA//KENYA-58/NEWTHATCH	1^+ 2^+
Kalyanosona	(1967)	FRONTANA // KENYA 58/ NEWTHATCH/3/NORIN 10 /BREVOR/4/ GABO 55	1^+ 2^+
Beacon-Ken	(1968)	Frontana / Kenya 58 // Newthatch /3/3* Bonza	3^- 4^-
Waldron	(1968)	JUSTIN/ND-81	1^-
Polk	(1968)	THATCHER / SUPREZA /3/ KENYA 58 / NEWTHATCH // FRONTANA	1^-

Table 4.1 continued…

GENOTYPE		PEDIGREE	SEEDLING INFECTION TYPES
1010 F3 SEL. 7	(1969)	II-50-17/KENYA-184-P	3^-
90 F4 SEL.D.1	(1969)	KENYA-360-H//2*MARQUIS/AGROPYRON ELONGATUM	$1^- 2^-$
1012 B.1. (L)	(1969)	MENTANA/KENYA//BAGE/3/KENYA-184-P	3^+
1061.K.4	(1969)	MIDA // McMURACHY / EXCHANGE /3/ RIO NEGRO	$3^- 4^-$
1010 F3 SEL. 4	(1969)	II-50-17/KENYA-184-P	$1^- 2^-$
Santa Elena	(1969)	SANTA-CATALINA-6/THATCHER//FROCOR	$3^+ 4$
Bonanza	(1969)	PITIC-62/(SIB)CHRIS//SONORA-64	1^-
Fletcher	(1970)	II-55-10/4/PEMBINA/II-52-329/3/II-53-388/III-58-4//II-53-546	1^-
Penjamo 62	(1972)	FKN/NORIN 10 BREVOR	3^+
Borah	(1974)	NO-58/THATCHER//THATCHER/KENYA-FARMER/3/MN-III-58-1//FRONTANA/3*THATCHER	4^+
Zaragoza 75	(1975)	MENGAVI/II-8156	$3^+ 4$
Era	(1970)	II-55-10/4/PEMBINA/II-52-329/3/II-53-388/III-58-4//II-53-546	1^-
Inia66	(1971)	LERMA ROJO 64/SONORA 64	3^-
CI 14393	(1975)	FROCOR*2/4/COMETA/3/ NEWTHATCH// MENTANA/ MENKEMEN	1^-
Sonora63	(1975)	YAKTANA-54//NORIN-10/BREVOR/3/2*YAQUI-54	$3^+ 4$
Bobwhite	(1977)	AVRORA//KALYANSONA/BLUEBIRD/3/(SIB)WOODPECKER	$3^+ 4$
Angus	(1978)	THATCHER/2*SUPREZA/3/FRONTANA//KENY58/NEWTHATCH/7/PEMBINA//FRONTANA/5*THATCHER/6/MIDA//KENYA-117-A/2*THATCHER/3/FRONTANA/4*THATCHER/4/MN-III-58-4/5/KENYA-58/NEWTHATCH//3*LEE	$1^- 2^-$
ET-12-D4	(1981)	MAMBA/UQ105	$3^- 4^-$
Marshall	(1982)	ERA/WALDRON	1^-
Pavon 76	(1982)	VICAM 571//CIANO F67/SIETE CERROS T	$1^- 2^-$

Table 4.1 continued …

GENOTYPE		PEDIGREE	SEEDLING INFECTION TYPES
Paa	(1982)	KVZ/3/CNO/CHRIS//0N	1^-
Gara	(1984)	AVRORA//KALYANSONA/BLUEBIRD/3/(SIB)WOODPECKER	3^- 4^-
Batu	(1984)	GALLO/CUCKOO//KAVKAZ/SUPER X	1^-
Dashen	(1984)	KAVKAZ/BUHO//KALYANSONA/BLUEBIRD	4^-
Minnpro	(1990)	MN-72299/MN-74115	3^- 4^-
Norm	(1992)	MN-73167/MN-81070	2^-
Verde	(1995)	MN-7663/SBY-354-A	1^-
Bacup	(1996)	NUY-BAY/PIONEER-2375//MARSHALL,USA	1^+ 2^-
Tusie	(1997)	COOK/VEERY//DOVE/SERI M82	2^+
Abola	(1997)	BOBWHITE/BUCKBUCK	-
Shina	(1998)	GOLDEN-VALLEY(GOV)/AZTECA-67//MUSALA/3/R-37/GHL-121//KALYANSONA/BLUEBIRD/4/ANI	1^+ 2^+
Dodota	(2001)	BLUEJAY/COCORAQUE F 75//PARULA/BOBWHITE	3^-
Sirbo	(2001)	VS73.600/MRL/3/BOBWHITE//YECORA F 70/TRIFON	3^+ 4
Bobicho	(2002)	PEREGRINE/PF70354/KALYANSONA/BLUEBIRD/ALONDRA/3/MARINGA	3^- 4^-
Mcvey	(1999)	NING-8331/MN-87029//MN-89068	1^- 2^-
Katar	(1999)	COOK/VEE''S''//DOVE''S''/SERI/3/BJY''S''	1^- 2^-
Wabe	(-)	MIRLO/BUCKBUCK	1^+
Fanfare	(-)	-	3^+ 4
Impala	(-)	-	3^-
Morris	(-)	THATCHER//KENYA-117 A/MIDA/3/FRONTANA/4*THATCHER/4/THATCHER/5/FRONTANA/4*THATCHER	2^-
PW Thatcher	(-)	THATCHER/AGENT	1^+
291 J.1.I.1	(-)	AUSTRALIA 26 / KENYA 58	3^+
R1476			1^- 2^-
R1475			4

Table 4.1 continued…

GENOTYPES	PEDIGREE	SEEDLING INFECTION TYPES
R1244	PRINIA/3/ALTAR84/AE.SQ//2*OPATA/4/CHEN/AEGILOPS SQUARROSA (TAUS)//BCN/3/BAV92	;
R1336	BABAX/LR42//BABAX*2/3/TUKURU	;
R1271	PBW343*2/KUKUNA*2//YANAC	1^{+} 2^{+}
R1286	QUAIU/3/PGO/SERI/BAV92	3^{+} 4
R1317	KSW/7/CAL/NH/H567.71/3/SERI/4/CAL/NH//H567.71/5/2*KAUZ/6/PASTOR/8/CAL/NH//H567.71/3/S ERI/4/CAL/NH//H567.71/5/2*KAUZ/6/PASTOR	1^{-}
R1474		3^{+} 4
R1305	KSW/5/2*ALTAR 84/AE.SQUARROSA (221)//3*BORI95/3/URESJUN/KAUZ/4/WBLLI	3^{-} 4^{-}
R1301	KSW/5/2*ALTAR 84/AE.SQUARROSA (221)//3*BORI95/3/URESJUN/KAUZ/4/WBLLI	3^{+}
R1309	KFA/5/REH/HARE//2*BCN/3/CROC-I/AE.SQUARROSA(213)//PGO/4/HUITES/6/REH/HARE//2*BCN/3/CROC-I/AE.SQUARROSA(213)//PGO/4/HUITES	1^{-} 2^{-}

0=Immune, R= Resistant, MR=moderately resistant, MS=moderately susceptible, S=Susceptible, TR=trace resistant, MSS= moderately susceptible and susceptible (Johnston and Browder, 1966); AUDPC=Area under Disease Progress Curve; SIT=Seedling Infection Type, FDS= Final Disease Severity.0, 0;, 1, 2 = resistance response, 3 and 4 = susceptibility response

CHAPTER FIVE

ANALYSIS OF WHEAT (*Triticum aestivum* L.) LEAF RUST (*Puccinia triticina* Eriks.) VIRULENCE IN KENYA.

5.1 Abstract

Leaf rust caused by *Puccinia triticina* Eriks. is one of the most important foliar diseases of wheat (*Triticum aestivum* L.) worldwide. The objective of this study was to determine the virulence of *Puccinia triticina* on North American and Australian differential sets. Leaf rust urediniospores collected from infected wheat genotypes in the International Screening Nursery (ISN) at Kenya Agricultural and Livestock Research Organization, Njoro in 2016 were inoculated on seedlings in the greenhouse and analyzed for virulence. Leaf rust differentials were used to determine the races that exist in Kenya. Seedlings were evaluated for infection types based on North American and Australian leaf rust differential sets. Varied disease infection types observed ranged from '0' to '3^{+}'. On both differential sets, leaf rust genes *Lr1, Lr2a, Lr2c, Lr3, Lr9, Lr16, Lr19, Lr24, Lr26, Lr3ka, Lr11, Lr17, Lr30, Lr3bg, Lr15, Lr18, Lr10, Lr23,* and *Lr27+Lr31* were avirulent to the races of Kenya. For North American differential sets, virulence was observed for *Lr10, Lr3ka* and *Lr3* while, for Australian differential sets, virulence was observed on resistant gene, *Lr13*. In addition to resistant genes identified in 20 differential sets (North American and Australian), resistant genes; *Lr2b, Lr3a, Lr12, Lr14, Lr20, Lr21, Lr22a, Lr25, Lr28, Lr29, Lr32, Lr34, Lr35, Lr36* and *LrB* were also identified among 91 differential lines (44 and 47 from North America and Australia, respectively) tested for leaf rust virulence. These leaf rust genes could be valuable source of resistance to leaf rust.

Key words: Leaf rust, Differential lines, Virulence

5.2 Introduction

Leaf rust caused by *Puccinia triticina* Eriks. is among the most important rust diseases of wheat (*Triticum aestivum* L.). It is globally distributed with diverse race structures that continuously evolve and form novel virulent races (Bolton *et al*., 2008). Leaf rust occurs more regularly and in more worldwide regions than stem (*Puccinia graminis*) and yellow rust (*Puccinia striiformis*) (Melvin *et al*., 2008). This disease attacks the leaf blades, although it can also infect the leaf sheath and glumes in highly susceptible cultivars (Huerta-Espino *et al*., 2011). Virulence of pathotypes can be characterized based on host seedling differential set. A

nomenclature system for designating virulence combinations of *Puccinia triticina* isolates in North America was accepted by the North American wheat leaf rust research workers committee in 1986 (Long and Kolmer, 1989; Kolmer *et al*., 2004). The identification of pathotypes involves infecting seedlings of a set of near-isogenic lines of wheat each carrying a different known leaf rust resistance gene, with a field collected sample of rust. The ability or inability of the rust isolate to infect each line allows the pathotype or pathotypes present to be identified (McIntosh *et al*., 1995; Park, 2016). These differential lines have been extremely valuable for conducting analysis of virulence variation in *Puccinia triticina* populations, genetics of leaf rust resistance in wheat and genetics of host–parasite relationship between wheat and *Puccinia triticina*. Currently, over 70 races of this pathogen are detected each year in North America where it persists through reproduction from asexual urediniospores (Kolmer *et al*., 2007).

Production of wheat in Kenya is highly affected by rusts; stem rust, yellow rust and leaf rust. Leaf rust disease is considered the most common and widely distributed of the three wheat rusts and has become more serious problem of wheat causing great losses in grain yields (MacIntosh *et al*., 1995; Huerta-Espino *et al*., 2011). This disease is capable of reducing wheat yields drastically depending on genotype susceptibility and stage of infection (Hollaway, 2014). Leaf rust reduces weight and number of kernels per spike in a wheat crop leading to yield losses ranging from 5% to 16% on average and up to 40% in epidemic years (Knott, 1989; Bolton *et al*., 2008). *Puccinia triticina* is now recognized as an important pathogen in wheat production worldwide, causing significant yield losses over large geographical areas (Roelfs *et al*., 1992; Marasas *et al*., 2004). Like the other two rusts, due to the long-distance dissemination of leaf rust races, leaf rust can spread very fast creating an epidemic in a very short duration of time in presence of humidity and relatively warm temperatures (Kolmer, 2005; Hanzalova and Bartos, 2014).

Highly effective durable resistance to leaf rust has been difficult to achieve due to the high degree of virulence variation in the *Puccinia triticina* population and the rapid selection of races with virulence to effective *Lr* genes in wheat genotypes (Jin *et al*., 2007). This high degree of specificity has made durable rust resistance in wheat difficult to achieve because the virulence of leaf rust against wheat resistance genes is highly diverse resulting in the existence of many different pathogenic races (Kolmer, 2005). For instance, the novel race BBG/BN and its variant BBG/BP overcame the resistance of widely adapted durum cultivars in northwestern Mexico

which had been effective and stable for more than twenty five years (Singh *et al* 2004; Huerta-Espino *et al*., 2009a). However, introgressing and pyramiding of genes that confer partial resistance is an outstanding method for developing wheat genotypes with durable resistance to leaf rust (Kolmer, 1996; Chu *et al*., 2009; Hollay, 2014). For instance, the rust-resistance gene *Lr41* from *T. tauschii* has been introgressed into chromosome 2D of several wheat cultivars that are currently under commercial production (Xiaochun *et al*., 2008). In addition, combining race-specific and race-nonspecific resistance genes such as *Lr16* (race-specific resistance gene) and *Lr34* (race-nonspecific resistance gene) in a single genotype could significantly improve both durability and the level of resistance (Kolmer *et al*., 2008; Zhang *et al*., 2008).

Seedling resistant genes together with adult plant resistant determine the host resistance to leaf rust (Kolmer *et al*., 2003). However, host resistance conferred by a single or a few genes could be easily overcome by the appearance of rust races with new combinations of virulence genes (McIDonald and Linde, 2002). Survey of wheat leaf rust using seedling differentials are very useful in describing virulence pathotypes , and how leaf rust phenotypes change in response to host selection (Rattu *et al*., 2009). In addition, virulence surveys are important for studying the evolution of new races and forecasting the virulence shifts in a physiologic races population (Admassu *et al*., 2009). This enhances, monitoring dynamic changes of rust pathogen populations to identify new virulent races, and deploying resistance genes to defeat the new pathogen race.

Numerous genes conferring resistance to wheat leaf rust have been identified and used in wheat breeding, however, several of these genes have been rendered ineffective due to the emergence of new virulent races (Kolmer *et al*., 2008). For instance, the results obtained from the survey conducted in Egypt during 2012-2014 growing seasons showed a significant variability in pathotypes which were different from season to season. A total of 118, 166 and 61 physiologic races were identified in 2011/2012, 2012/2013 and 2013/2014, respectively with the most frequent pathotypes designated as STTST and TKTTT (each with 2.54%) in 2011/2012; PKTST (6.63%), TTTTT (7.83%) and TTTST (10.24%) in 2012/2013 as well as FKTTT (4.92%) and PTTTT (11.47%) in 2013/2014 (Walid *et al*., 2015). This was attributed to host-pathogen interaction in wheat where virulence shifts in the pathogen populations, and hence, reduce the effectiveness of a number of leaf rust resistance genes (Johnson, 2000). Wheat varieties that rely on race-specific resistance often lose effectiveness within a few years by

imposing selection for virulent leaf rust races because, most leaf rust resistance genes confer race-specific resistance in a gene-for-gene manner (Singh *et al*., 2000; McIntosh *et al*., 2007). In Kenya leaf rust disease of wheat has received less attention with the presence of stem and yellow rusts which are the most aggressive. Therefore, the limited information is available on leaf rust hence, the objective of the present study was to identify the virulence of wheat leaf rust in Kenya using leaf rust differential sets.

5.3 Materials and methods

5.3.1 Experimental site

Evaluation of wheat leaf rust differential lines for leaf rust virulence in Kenya was conducted in the greenhouse at Kenya Agricultural and Livestock Research Organization (KA*LR*O)-Njoro (0° 20´S, 35° 56´E). This site is situated in the low highlands III (LH_3) Agro ecological zone, in Nakuru Kenya and elevated at approximately 2,185 masl (Jaetzold *et al*., 2012). This area experiences an average minimum and maximum temperature of 8 ± 2 °C and 25 ± 2 °C, respectively and an average annual precipitation of 996.4 ± 4.2 mm (KALRO Meteorological station No. 903502 (1), 2013).

5.3.2 Differential hosts

The experiment was conducted under greenhouse conditions where virulence of leaf rust urediniospores collected from International Screening Nursery, Njoro was determined on 91 differential lines. Forty four of the differential lines were acquired from CIMMYT and 47 from Australia. Among these 44 and 47 differential lines, 20 differential sets were selected and used in the first experiment while a total of 91 differential lines were used in the second experiment.

5.3.3 Virulence analysis of *Puccinia triticina*

Response of differential seedlings to leaf rust infection was assessed following the procedure described in section 4.3.4 a and b of chapter four. Host differential lines were grouped into sets of four (Table 5.1), and a total of 91 differential lines were presented in Table 5.2.

5.4 Results

Generally, there were limited variations due to responses to leaf rust infection on wheat differential sets from North America and Australia (Table 5.1). However, there were variable infection types among the differential sets. All *Lr* genes evaluated for leaf rust infection showed resistance except *Lr3 (*Tc*6/Democrat), *Lr3ka* (Tc*6/Klein Aniversario) and *Lr10* (Tc*6/Exchange) from North America and *Lr13* in *Egret* background from Australia. In 2016, leaf rust disease infection types ranged from "0" to "3^{+}" on differential sets. The leaf rust population was virulent to *Lr3, Lr3ka* located on chromosome *6BL* and *Lr10* located on chromosome *1A* with ITs "3", "3^{-}3" and "2^{+}3", respectively while differential sets possessing; *Lr1, Lr2a, Lr2c, Lr3bg, Lr9, Lr11, Lr13, Lr15, Lr16, Lr17, Lr18, Lr19, Lr23, Lr24, Lr26, Lr30,* and *Lr27+Lr31* genes from North America showed resistance with ITs ranging from "0" to "2". On Australian differential sets, the leaf rust population was virulent to *Lr13* which exhibited an infection type reaction of "2" to "3" and avirulent to resistant genes; *Lr1, Lr2a, Lr2c, Lr3a, Lr3bg, Lr3ka, Lr9, Lr10, Lr11, Lr15, Lr16, Lr17, Lr18, Lr19, Lr23, Lr24, Lr26, Lr30,* and *Lr27+Lr31* with ITs ranging from "0" to "2" .

Of the twenty pairs of wheat differential sets inoculated, sixteen pairs including *Lr1, Lr2a, Lr2c, Lr3bg, Lr9, Lr11, Lr15, Lr16, Lr17, Lr18, Lr19, Lr23, Lr24, Lr26, Lr30* and *Lr27 + Lr31* located on chromosome *5DL, 2DS,2DS, 6BL, 6BL, 2A, 2DS, 4B, 2AS, 5BL, 7DL, 2BS, 3DL, IR, 4AL* and *3BS*, respectively displayed the same infection type pattern. Infection types on genes *Lr3a* and *Lr3ka* in *Thatcher* background from North America showed different response to leaf rust population compared to *Lr3* in *Democrat* background and *Lr3ka* in *Thatcher* background from Australia located on chromosome arm *6BL*. Similarly, ITs on *Lr13* in *Egret* background from Australia ranged from "2" to "3^{-}" while ITs on *Lr13* in *Manitou* background from North America ranged from ";" to "2^{+}" despite of being located on chromosome arm *2BS*. Although, gene *Lr10* in *Thatcher* background from Australia and *Lr10* in *Thatcher* background from North America are located at the same locus in chromosome arm *IA*, they showed varied infection types.

Additionally, of the 91 differential lines tested for seedling reaction, showed diverse infection types (Table 5.2). Among 44 North American lines tested, lines possessing *Lr3* (Tc*6/Exchange), *Lr3ka* (Tc*6/Klein), *Lr10* (Tc*6/Exchange) and *Lr33* (Tc*6/PI58548) exhibited susceptible response of "$33^{+"}$, "$3^{+"}$, "3" and "$3^{-"}$, respectively. Similarly, among 47

Australian differential lines tested, lines having genes *Lr2a + Lr3a*, *Lr13* in *Egret* background, *Lr16* in *Thatcher* background, and *Lr37* in *Sunlin* background exhibited susceptible response ranging from "3^{-}" to "3^{+}" infection types. Differential line *Egret* from Australia possessing *Lr13* had high ITs of '3^{-}' to the leaf rust population tested, similar to that of near isogenic lines (NILs) containing *Lr3* (Tc*6/Democrat), *Lr3ka* (Tc*6/Klein Aniversario) and *Lr10* (Tc*6/Exchange) from North American origin. Differential lines possessing; *Lr16* in *Thatcher* background, *Lr37* in *Sunlin* background and *Lr2a + Lr3a* in *Meditrranean* background from Australia had high ITs of "3^{-}", "3^{-}" and "3^{+}", respectively to *Puccinia triticina* population tested. The rest of the differential lines exhibited resistance infection types ranging from "0" to "2". Leaf rust population from ISN, Njoro was virulent for *Lr16* in *Thatcher* background and *Lr37* in *Sunlin* background from Australia but avirulent for *Lr16 i*n *Thatcher* background and *Lr37* in *Thatcher* background from North America despite of being located on chromosome *4B* and *2AS*, respectively.

Table 5.1. Virulence analysis of leaf rust (Puccinia triticina) population from International Screening Nursery, Njoro using the seedling infection types (ITs) on 20 North American and 20 Australian wheat differential sets.

Host line	Pedigree	R.L.NO	Origin	*Lr* gene	Chromosome	Infection types Set 1	Set 2	Set 3
Set 1								
Tarsa		R.L.6003	Australian	*Lr1*	5DL	; 1	;	;
Nil-Thatcher-*Lr*1-Ctr	Tc*6/Centenario	R.L.6003	N. America	*Lr1*	5DL	1 2	; 1	1 2
Thatcher +*Lr*2a		R.L.6019	Australian	*Lr2a*	2DS	; 2	; 1	1^-
Nil-Thatcher-*Lr*2a-Wst	Tc*6/Webster	R.L.6016	N. America	*Lr2a*	2DS	; 1	; 1	0 ;
Thatcher+*Lr*2c		R.L.6022	Australian	*Lr2c*	2DS	1 2	1 2	; 1^-
Nil-Thatcher-*Lr*2c-Loros	Tc*6/Loros	R.L.6047	N. America	*Lr2c*	2DS	1 2^+	; 1	; 1
Democrat		R.L.6002	Australian	*Lr3a*	6BL	1 2^+	; 1	-
Nil-Thatcher-*Lr*3-Democrat	Tc*6/Democrat	R.L.6002	N. America	*Lr3*	6BL	3	3	3
Set 2								
Thatcher+*Lr*9		R.L.6010	Australian	*Lr9*	6BL	; 1	0 ;	0 ;
Nil-Thatcher-*Lr*9-Tranfer	Transfer/Tc*6	R.L.6010	N. America	*Lr9*	6BL	; 1	; 1	; 1

Table 5.1: Continued…

Exchange		R.L.6005	Australian	*Lr16*	4B	; 1	;	;
Nil-Thatcher-*Lr*16-Ex	Tc*6/Exchange	R.L.6005	N. America	*Lr16*	4B	1 2^{+}	; 1	1 2^{+}
Agent		R.L.6064	Australian	*Lr24*	3DL	; 1	;	1
Nil-Thatcher-*Lr*24-Agent	Tc*6/Agent	R.L.6064	N. America	*Lr24*	3DL	; 1	; 1	; 1
Thatcher+*Lr*26		R.L.6078	Australian	*Lr26*	1R	2^{+}	1	2^{+}
Nil-Thatcher-*Lr*26-St-1-25	Tc*6/St-1-25	R.L.6078	N. America	*Lr26*	1R	; 1	; 1	0 ;
Set 3								
Thatcher+*Lr*3ka		R.L.6010	Australian	*Lr3ka*	6BL	; 1	; 1	; 1
Il-Thatcher-*Lr*3ka-Aiv	Tc*6/Klein Aniversario	R.L.6007	N. America	*Lr3ka*	6BL	3^{+}	3^{-}	3
Thatcher+*Lr*11		R.L.6048	Australian	*Lr11*	2A	; 1	;	1^{-}
Hussar-*Lr*11	Tc*2/Hussar	RL6053	N. America	*Lr11*	2A	; 1	; 1	; 1
Songlen		R.L.6041	Australian	*Lr17*	2AS	1 2	; 1	2^{-}
Nil-Thatcher-*Lr*17-Kllu	K.Lucero/Tc*6	R.L.6008	N. America	*Lr17*	2AS	1 2^{+}	1 2	2^{+}
Thatcher+*Lr*30		R.L.6049	Australian	*Lr30*	4AL	1 2	; 1	1

Table 5.1: Continued….

Nil-Thatcher-*Lr*30-Tzio	Tc*6/Terenzio	R.L.6049	N. America	*Lr30*	4AL	; 1	1	; 1
Set 4								
Mantana		R.L.6042	Australian	*Lr3bg*	6BL	2^{+}	1 2	1 2
Nil-Thatcher-*Lr*3bg-Bage	Bage/Tc*8	R.L.6042	N. America	*Lr3bg*	6BL	2^{+}	2^{+}	; 1
Egret			Australian	*Lr13*	2BS	3^{-}	2 3^{-}	3^{-}
Manitou-*Lr*13			N. America	*Lr13*	2BS	2+	2^{+}	; 1
K1483	-	R.L.6052	Australian	*Lr15*	2DS	1 2^{+}	;	1 2^{-}
Nil-Thatcher-*Lr*15-K1483	Tc*6/Kenya W1483	R.L.6052	N. America	*Lr15*	2DS	; 1	; 1	0
Thatcher+*Lr*18		R.L.6009	Australian	*Lr18*	5BL	1 2	; 1	2
Nil-Thatcher-*Lr*18-Af43	Tc*7/Africa43	R.L.6009	N. America	*Lr18*	5BL	; 1	; 1	0 ;
Set 5								
Thatcher+*Lr*10		R.L.6146	Australian	*Lr10*	1A	2^{-}	; 1	;
Nil-Thatcher-*Lr*10-Ex	Tc*6/Exchange	R.L.6004	N. America	*Lr10*	IA	3	2^{+}	3
Thatcher+*Lr*19		R.L.6040	Australian	*Lr19*	7DL	0 ;	;	0

Table 5.1: Continued…

Nil-Thatcher-*Lr*19-Tr	Tc*7/Tr.4 A.elong	RL6040	N. America	*Lr19*	7DL	0 ;	0 ;	0
Thatcher+*Lr*23		R.L.6012	Australian	*Lr23*	2BS	1 2	; 1	1^{-}
	Lee		N. America	*Lr23*	2BS	1 2	; 1	; 1
	310/Tc*6							
Nil-Thatcher-*Lr*23-Lee310		R.L.6012						
Sun 6B	-	-	Australian	*Lr1, Lr2a ,Lr27+Lr31*	-	1 2	; 1	1 2
	Gatcher			*Lr27 +Lr 31*	3BS	; 1	; 1	;
Gatcher-*Lr*27+*Lr*31	[W3021]	W3021	N. America					

*Leaf rust genes observed to be virulence; 0= no uredinia or flecks visible, 0; = very faint hypersensitive flecks; = hypersensitive flecks, 1 = small uredinia surrounded by necrosis, 2 = small uredinia surrounded by chlorosis, 3 = moderate size uredinia without chlorosis, + = slightly larger uredinia than expected for the infection type, - = slightly smaller uredinia than expected for the infection type (Johnston and Browder, 1966); #some = chromosome.

Table 5.2. Seedling infection types (ITs) on 44 North American and 47 Australian wheat differential lines inoculated with leaf rust (Puccinia triticina) urediniospores.

Host line	Pedigree	Source	*Lr* gene	Chromosome	SIT
Thatcher		N. America	-		1^-
Thatcher		Australian	-		; 1
Nil-Thatcher-*Lr*1-Ctr	Tc*6/Centenario	N. America	*Lr1*	5DL	1 2
TarSa		Australian	*Lr1*	5DL	;1
Thatcher+*Lr*1		Australian	*Lr1*	5DL	0 ;
Nil-Thatcher-*Lr*2a-WSt	Tc*6/WebSter	N. America	*Lr2a*	2DS	; 1
WebSter		Australian	*Lr2a*	2DS	; 1^-
Thatcher+*Lr*2a		Australian	*Lr2a*	2DS	0 1^-
Mediterranean		Australian	*Lr2a, Lr3a*	2DS	3^+
Nil-Thatcher-*Lr*2b-Carina	Tc*6/Carina	N. America	*Lr2b*	2DS	1-
Thatcher+*Lr*2b		Australian	*Lr2b*	2DS	1^- 2^-
Nil-Thatcher-*Lr*2c-LoroS	Tc*6/Loros	N. America	*Lr2c*	2DS	; 1
Thatcher+*Lr*2c		Australian	*Lr2c*	2DS	1 2
Nil-Thatcher-*Lr*3-Democrat	Tc*6/Democrat	N. America	*Lr3*	6B	3 3^+
Democrat		Australian	*Lr3a*	6B	1 2^+

Table 5.2 Continue…

Thatcher+*Lr*3a		Australian	*Lr3a*	6B	1^{+} 2^{+}
Il-Thatcher-*Lr*3ka-Aiv	Tc*6/Klein	N. America	*Lr3ka*	6B	3^{+}
Klein Titan		Australian	*Lr3ka*	6B	0
Thatcher+*Lr*3ka		Australian	*Lr3ka*	6B	;1
Nil-Thatcher-*Lr*3bg-Bage	Bage/Tc*8	N. America	*Lr3bg*	6B	2^{+}
Mantana	-	Australian	*Lr3bg*	6B	2^{+}
Nil-Thatcher-*Lr*9-Tranfer	Transfer/Tc*6	N. America	*Lr9*	6BL	; 1
Thatcher+*Lr*9		Australian	*Lr9*	6BL	; 1
Nil-Thatcher-*Lr*10-Ex	Tc*6/Exchange	N. America	*Lr10*	1A	3
HuSSar-*Lr*11	Tc*2/Hussar	N. America	*Lr11*	2A	; 1
Thatcher+*Lr*10		Australian	*Lr10*	1A	2^{-}
Thatcher+*Lr*11		Australian	*Lr11*	2A	; 1
Nil-Thatcher-*Lr*12-Ex	Exchange/Tc*6	N. America	*Lr12*	4B	0 ;
Manitou-*Lr*13	Tc*6/Frontana	N. America	*Lr13*	2BS	2^{+}
Thatcher+*Lr*13		Australian	*Lr13*	2BS	1^{-} 2^{-}

Table 5.2. Continue…

Egret		Australian	*Lr13*	2BS	3^{-}
Naparoo		Australian	*Lr13, Lr24*	-	-
Nil-Thatcher-*Lr*14a-Sk	Selkirk/Tc*6	N. America	*Lr14a*	7B	1^{-}
Spica		Australian	*Lr14a*	7B	; 1^{-}
Nil-Thatcher-*Lr*14b-Me	Tc*6/M. EScobar	N. America	*Lr14b*	7B	1^{-} 2^{-}
Nil-Thatcher-*Lr*15-K1483	Tc*6/Kenya	N. America	*Lr15*	2D	; 1
Thatcher+*Lr*15		Australian	*Lr15*	2D	0 ;
K1483		Australian	*Lr15*	2D	1^{-} 2^{+}
Nil-Thatcher-*Lr*16-Ex	Tc*6/Exchange	N. America	*Lr16*	4B	1 2^{+}
Exchange		Australian	*Lr16*	4B	; 1^{-}
Thatcher+*Lr*16		Australian	*Lr16*	4B	3^{-}
Nil-Thatcher-*Lr*17-Kllu	K.Lucero/Tc*6	N. America	*Lr17*	2AS	1 2^{+}
Songlen		Australian	*Lr17*	2AS	$1^{-}2^{-}$
Thatcher+*Lr*17a		Australian	*Lr17a*	2AS	0 ;
Harrier		Australian	*Lr17b*	2AS	2^{-}
Nil-Thatcher-*Lr*18-Af43	Tc*7/Africa43	N. America	*Lr18*	5BL	; 1
Thatcher+*Lr*18		Australian	*Lr18*	5BL	1 2

Table 5.2. Continue…

Nil-Thatcher-*Lr*19-Tr	Tc*6/Jimmer	N. America	*Lr19*	7DL	0;
Thatcher+*Lr*19		Australian	*Lr19*	7DL	;
Agatha		Australian	*Lr19*	7DL	0
Thew-*Lr*20	Tc*6/Jimmer	N. America	*Lr20*	7AL	0
Thew		Australian	*Lr20*	7AL	; 1^{-}
Norka		Australian	*Lr1, Lr20*	-	0 ;
Nil-Thatcher-*Lr*21-Rl5406	Tc*6/RL5406 Tetra	N. America	*Lr21*	1DL	0 ;
Thatcher+*Lr*21		Australian	*Lr21*	1DL	-
Nil-Thatcher-*Lr*22a	Tc*6/RL 5404 TetraC	N. America	*Lr22a*	2DS	0
Nil-Thatcher-*Lr*23-Lee310	Lee 310/Tc*6	N. America	*Lr23*	2BS	1 2
Gaza		Australian	*Lr23*	2BS	; 1^{-}
Thatcher+*Lr*23		Australian	*Lr23*	2BS	; 1^{-}
Nil-Thatcher-*Lr*24-Agent	Tc*6/Agent	N. America	*Lr24*	3DL	; 1
Agent		Australian	*Lr24*	3DL	; 1
TranSec (Awned)-*Lr*25	Tc*6/TranSec	N. America	*Lr25*	4BS	0 ;

Table5.2. Continue…

Thatcher+*Lr*25		Australian	*Lr25*	4BS	-
Nil-Thatcher-*Lr*26-St-1-25	Tc*6/St-1-25	N. America	*Lr26*	1B	; 1
MildreSS		Australian	*Lr26*	1B	-
Gatcher-*Lr*27+*Lr*31	Gatcher W3021	N. America	*Lr27+Lr31*	3BS	; 1
Thatcher+*Lr*26		Australian	*Lr26*	1B	$1\ 2^{+}$
Gatcher		Australian	*Lr27+31*	3BS	1^{-}
Sun 6B		Australian	*Lr1,Lr3a, Lr27+31*	-	$1\ 2^{-}$
CS2d-2m-*Lr*28	Tc*6/C-77-1	N. America	*Lr28*	4AL	0
CS 2a/2m		Australian	*Lr28*	4AL	0
Nil-Thatcher-*Lr*29-CS7ag11	Tc*6/CS7D-Ag#11	N. America	*Lr29*	7DS	0
Thatcher+*Lr*29		Australian	*Lr29*	7DS	0
Nil-Thatcher-*Lr*30-Tzio	Tc*6/Terenzio	N. America	*Lr30*	4AL	; 1
Thatcher+*Lr*30		Australian	*Lr30*	4AL	; 1
Nil-Thatcher-*Lr*32-Ae.Ta	Tc*6/Ae. Sq.	N. America	*Lr32*	3DS	; 1
Nil-Thatcher-*Lr*33-Pi58548	Tc*6/PI58548	N. America	*Lr33*	1BL	3^{-}

Table 5.2. Continue….

Nil-Thatcher-*Lr*34-Pi58548	Tc*6/PI58548	N. America	*Lr34*	7DS	1^{-}
Nil-MarquiS-*Lr*35-T.Sp	Tc*6/RL 5711	N. America	*Lr35*	2B	0
Thatcher+*Lr*37		Australian	*Lr37*	2AS	;
Nil-Thatcher-*Lr*37-Vpm	Tc*8/VPM1	N. America	*Lr37*	2AS	;
Sunlin	-	Australian	*Lr37*	2AS	3^{-}
Nil-Thatcher-*Lr*b-Carina	Tc*6/Carina	N. America	*LrB*	-	1^{-} 2^{-}
Wl 711		N. America	-	-	1^{-}
Gaza		N. America	-	-	0
Altar 84		N. America	-	-	1^{-}
Dw 7276		N. America	-	-	1^{-}
Iumillo-Sr9g,Sr12,+		N. America	-\\\	-	1^{-}
40SAtil*2/Local Red		N. America	-	-	0 ;
Morocco		Australian	*Lr73*	-	-
K.Chiriku (Check)		Kenya	-	-	3^{+} 4

*Leaf rust genes observed to be virulence; 0= no uredinia or flecks visible, 0; = very faint hypersensitive flecks; = hypersensitive flecks, 1 = small uredinia surrounded by necrosis, 2 = small uredinia surrounded by chlorosis, 3 = moderate size uredinia without chlorosis, + = slightly larger uredinia than expected for the infection type, - = slightly smaller uredinia than expected for the infection type (Johnston and Browder, 1966)

5.5 Discussion

In this study, *Lr1, Lr2a, Lr2c, Lr9, Lr16, Lr24, Lr26, Lr11, Lr17, Lr30, Lr3bg, Lr13, Lr15, Lr18, Lr19, Lr21, Lr23, Lr25* and *Lr27+Lr31* from North American differential sets showed effectiveness against leaf rust disease from the International Screening Nursery, Njoro. These results are in tandem with those of Wanyera *et al*. (2014) who found out that, leaf rust resistance genes; *Lr13, Lr15, Lr16, Lr17, Lr19, Lr21* and *Lr25* were resistant to the leaf rust isolate from this site in 2011. Resistance gene *Lr13* in *Egret* background from Australian origin was virulent to *Puccinia triticina* population in Njoro. The results are consistent with those of Oelke and Kolmer, (2004) who conducted their research on resistance in hard red spring wheat cultivars and found out that differential lines with the resistance gene *Lr13* were susceptible to *Puccinia triticina* in United States. Resistance genes *Lr16* and *Lr24* showed resistance against *Puccinia triticina* in Njoro over 2016 cropping season which is in agreement with results of Kolmer *et al*. (2003) who found out that *Lr16* and *Lr24* were resistant to leaf rust population in Midwest. The leaf rust disease from the International Screening Nursery was virulent to the resistant gene *Lr3* in *Thatcher* background from North American origin. This in agreement with findings reported in United States where, nearly all of the *Puccinia tritcina* isolates were virulent to *Lr3* (Oeke and Kolmer, 2004).

The variation of infection types expressed within and among North American and Australian differential sets in this study may be attributed to differences in the background or origin of the differential sets and entire host or pathogen genotype. For instance, leaf rust resistance genes designated as *Lr2a, Lr2c* and *Lr15* were mapped to a locus on chromosomes arm *2DS* (McIntosh and Baker, 1968), *Lr3a, Lr3ka* and *Lr3bg* are at a locus on chromosome arm *6BL* (Haggag and Dyck, 1973). These genes showed low infection types ranging from ";" to "2^{+}" but *Lr3 (Lr3a)* and *Lr3ka* from *Thatcher* background in North American origin were virulent with infection types "3" and "3^{+}", respectively despite of being at a locus in the same chromosome arm *6BL*. According to Long and Kolmer, (1989) infection types expressed by

differential sets vary depending on the entire host or pathogen genotype and environment. Genes *Lr3* and *Lr3ka* are all located in chromosome *6BL* but the infection types varied between differential sets from Australian and North American origin possessing these genes. This is an indication that, the variation was due to differences in background and source of the differential sets. Therefore, some differential sets may be more useful than others in some regions of the world depending on the leaf rust races present.

The wheat differential lines possessing leaf rust resistance genes, *Lr9 Lr17, Lr17b, Lr18, Lr19, Lr1+ Lr20, Lr21, Lr22a, Lr24, Lr25, Lr31, Lr28, Lr29, Lr34, Lr35,Lr37 Lr27+Lr31* exhibited seedling resistance during 2016 wheat growing season. The results are in agreement with those of Niazmand *et al*. (2010) who found that no virulence were detected on *Lr9, Lr19, Lr25* and *Lr28* resistance genes in Iran during 2007-2008 growing season. Resistance genes *Lr9, Lr19, Lr28* and *Lr34* were effective on pathogen population of *Puccinia triticina* in Njoro. These findings are consistent with those of Rattu *et al*. (2009) who reported the effectiveness of *Lr9, Lr19, Lr28* and *Lr34* on pathogen population of *Puccinia triticina* in Pakistan. The results demonstrated broad effectiveness of *Lr19, Lr21, Lr29* and *Lr34* against *Puccinia triticina* in Njoro which is consistent with findings by (McCallum and Seto-Gon, 2004) who made a determination which showed that *Lr19, Lr21, Lr29* and *Lr34* were effective against three hundred and sixty-two *Puccinia triticina* isolates collected across Canada during 2001. Genotypes with *Lr2a* combined with *Lr3a, Lr16*, and *Lr37* exhibited susceptible response ranging from "3^{-}" to "3^{+}" infection types. A study independently conducted by Negm *et al*. (2013) also detected that *Lr3* and *Lr16* were ineffective against most race groups tested during 2009/2010 and 2010/2011 growing seasons in Egypt.

Resistance gene *Lr1* has been shown to interact with *Lr20* to condition lower seedling infection types that either of the genes condition separately. Similar results were shown by the interaction of *Lr16* with *Lr34* and *Lr13* which conditioned lower seedling infection types than either of the resistance genes independently (German and Kolmer, 1992; Kolmer *et al*., 2010). This proves that, combination of adult plant *Lr* resistance genes such as *Lr34* with effective seedling genes can also provide good level of durable resistance. Therefore, pyramiding of several leaf rust resistance genes into a single genotype is of importance since the combined effects give the genotype a wider base of disease resistance (Roelfs *et al*., 1992; Chu *et al*., 2009).

5.6 Conclusion

The 2016 leaf rust samples from the International Screening Nursery, Njoro were avirulent for *Lr1, Lr2a, Lr2c, Lr9, Lr16, Lr24, Lr26, Lr11, Lr17, Lr30, Lr3bg, Lr15, Lr18, Lr19, Lr23,* and *Lr27+Lr31* on both North American and Australian leaf rust differential sets. In addition, wheat differential lines with resistant genes; *Lr2b, Lr3a, Lr12, Lr14, Lr20, Lr21, Lr22a, Lr25, Lr28, Lr29, Lr32, Lr34, Lr35, Lr36* and *LrB* exhibited good degree of seedling resistance to leaf rust. Therefore, these sources of resistance could be introgressed into wheat genotypes to diversify the existing gene pool for leaf rust. In addition, continued monitoring of leaf rust disease virulence is necessary for early detection of changes in pathogen population in Kenya. Furthermore, frequent and rigorous monitoring and continuous modeling of forecast should be established in the country for the identification of genes for resistance with concurrent knowledge of the changes occurring in the *Pucinia triticina* population.

CHAPTER SIX

6.0 GENERAL DISCUSSION, CONCLUSIONS AND RECOMMENDATIONS

6.1 General Discussion

There are various strategies employed to control leaf rust and yield loss on wheat which includes; the use of fungicides, incorporating genetic resistance into susceptible wheat genotypes and crop management. Genetic resistance is the primary tool to protect wheat crops from leaf rust disease. Despite the fact that it takes long time breeding for durable resistant wheat genotypes to leaf rust remains cost effective option of minimizing loss due to disease (Yuen *et al*., 2007). However, breeding efforts are challenged by rapidly mutating nature of leaf rust pathogen. It is therefore, very crucial to cross-check wheat genotypes at every growth stage. This ensures that novel sources of resistance to the emerging races and high yielding potential are identified and employed in various wheat improvement programmes.

In Kenya, there is knowledge gap on leaf rust virulence. Leaf rust has appeared sporadically and has not been a problem for the past 20 years, but recently it has emerged in the wheat grown fields and experimental plots including International Screening Nursery with severity of over 50%. Breeding of wheat genotypes with durable resistance to leaf rust continues to be a priority but also it is a challenge due to the complexity of interactions among resistance genes with newly evolved races. Leaf rust resistance is normally in two types; seedling and adult plant resistance. In the selection criteria, plant breeders mainly consider genotypes that exhibit both types of resistance as well as high yielding potential. This is because, the main aim is to increase yield production in crops.

To create awareness on virulence of leaf rust and contribute towards improvement of wheat production in Kenya, three experiments were carried out. The first experiment involved evaluation of wheat genotypes for response to infection at adult stage in the field. Second experiment, involved evaluation for the same genotypes for infection type at seedling stage in the greenhouse. The third experiment involved leaf rust virulence determination using 91 differential lines in the greenhouse.

Often, the resistance of wheat to leaf rust is determined by adult plant resistance genes in combination with seedling resistance genes. Leaf rust infections are greatly influenced by the compatibility with the genetic constitutions of the host in a given environment and prevalence of aggressive and virulent races of the pathogen. With this in perspective, the use of resistant wheat genotypes is the most economical and environmentally sustainable technique of controlling rust infections, and additionally, it greatly lowers the cost of

fungicides applied. The emergence of new races can easily overcome the host resistance as it is usually conferred by a single or a few genes.

5.2 Conclusion

1. There was variation in seedling infection type ranging from resistance to susceptible. Seventy-nine genotypes exhibited resistance (infection types of ";", "1", "2" or combinations of either two or three). The rest of the genotypes showed susceptible reactions ranging from "3" to "4".
2. The field experiment confirmed the existence of significant genetic variation among the wheat genotypes for resistance to leaf rust. It is worth to note that seven genotypes (*K. Tai, K. Korongo, Fletcher, Verder, R1244, R1305, and R1301*) showed resistance response at adult stage for the two seasons.
3. Leaf rust virulence analysis revealed varied disease infection types ranging from '0'to '3^{+}'. For both sets of differential lines, avirulence was observed for leaf rust genes *lr1, lr2a, lr2c, lr3, lr9, lr16, lr19, lr24, lr26, lr3ka, lr11, lr17, lr30, lr3bg, lr13, lr15, lr18, lr10, lr23, and lr27+lr31, lr2b, lr3a, lr12, lr14, lr20, lr21, lr22a, lr25, lr28, lr29, lr32, lr34, lr35, lr36* and *lrB.*

5.3 Recommendations

1. Genetic studies should be done on the identified 79 resistant genotypes to determine the number of responsible genes and the mode of action. In addition, diagnostic molecular markers should be used on these genotypes to confirm the phenotypically identified resistance genes
2. Considering the field disease reaction and yield performance for the genotypes across the seasons, genotypes K. Tai, K. Korongo, Fletcher, Verder, R1244, R1301 and R1305 ranked the best. These genotypes can be exploited in wheat breeding programmes for development of high yielding and leaf rust resistant wheat genotypes.
3. Differential lines, with leaf rust resistance genes; lr1, lr2a, lr2c, lr3, lr9, lr16, lr19, lr24, lr26, lr3ka, lr11, lr17, lr30, lr3bg, lr13, lr15, lr18, lr10, lr23, and lr27+lr31, lr2b, lr3a, lr12, lr14, lr20, lr21, lr22a, lr25, lr28, lr29, lr32, lr34, lr35, lr36 and lrB are potential lines to be used in wheat breeding programmes as well.

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APPENDICES

Appendix 1. Means of agronomic traits for wheat (*Triticum aestivum* L.) genotypes evaluated at KALRO, Njoro for two season in 2016.

					Area under Disease Progress Curve		
Genotype	Biomass (tonnes.ha^{-1})	Yield (tonnes.ha^{-1})	Thousand kernel weight (g)	Hectolitre weight(kg.hl^{-1})	Leaf rust	Stem rust	Yellow rust
Simba	27.00	1.56	3.65	50.14	92.35	246.17	43.75
Beacon-Ken	33.15	2.33	3.44	51.29	52.58	40.83	172.08
KkBB	21.01	0.41	2.11	42.01	159.24	180.25	373.33
Mbega	32.10	0.83	2.22	39.00	55.96	120.75	0.00
Tama	34.57	3.23	5.07	63.02	120.25	100.33	201.25
690 F4 Sel.D.1	28.65	1.10	4.20	55.33	216.74	68.83	282.92
Kenya-294-B-2 A3	17.41	1.11	4.23	54.59	226.87	339.50	358.75
Abola	18.11	1.04	3.17	50.59	96.53	200.08	292.25
Kenya 155	24.68	1.60	3.07	56.62	484.94	524.42	164.50
R. Sabanero	29.13	0.36	2.77	48.89	400.69	123.00	85.75
Marquillo	35.67	3.08	4.35	53.61	15.33	55.42	75.83
Zaragoza 75	33.28	1.38	4.15	55.10	280.45	81.57	632.92
Justin	44.15	0.31	1.62	51.93	183.26	144.08	131.83
Yaqui 50	27.56	2.34	3.85	54.70	210.42	257.25	140.00
Fletcher	56.01	0.71	2.68	44.70	0.00	30.33	102.08
Thatcher	28.36	0.28	1.35	24.02	563.98	126.00	282.92
Pewter	43.50	0.87	3.23	52.52	91.16	70.00	103.25
Era	44.59	0.77	2.92	48.28	13.50	49.00	326.67

Means followed by the same letters down the columns are not significantly different at $LSD_{0.05}$

Appendix 1. ***Continue……***

Genotype	Biomass (tonnes.ha^{-1})	Yield (tonnes.ha^{-1})	Thousand kernel weight (g)	Hectolitre weight(kg.hl^{-1})	Area under Disease Progress Curve		
					Leaf rust	Stem rust	Yellow rust
Dashen	23.71	1.24	3.09	44.30	436.47	123.67	245.00
Shina	14.46	0.89	3.07	52.05	201.25	190.17	266.00
Romany	34.78	2.76	4.49	56.87	65.33	117.25	292.25
Et-12-D4	29.72	3.51	3.97	56.05	34.54	113.75	178.50
Gabrino	49.73	2.19	4.30	51.17	218.33	84.58	181.42
Kentana 48	21.74	0.94	2.88	45.84	174.24	221.08	339.50
Regent	28.04	0.81	2.68	50.00	396.79	152.83	359.92
Kongoni	31.74	3.11	4.21	51.23	512.60	87.50	41.42
Kenya Civet	27.63	1.66	4.34	58.36	554.29	31.50	117.25
Bobicho	27.71	1.70	3.13	46.39	163.46	227.50	11.67
Gara	30.26	1.92	3.69	53.61	136.48	152.83	126.00
Chris	40.07	1.54	4.08	59.31	84.63	59.50	124.25
Kenya Tumbili	30.84	2.34	4.72	57.97	189.44	410.67	31.10
Pavon 76	22.09	2.29	3.96	54.14	125.43	61.25	247.92
Gem	36.18	1.88	4.52	58.22	266.42	103.25	274.17
Angus	52.18	2.15	3.34	42.81	2.92	12.25	176.75
1010 F3 Sel. 7	32.82	0.84	3.41	51.52	51.85	36.75	195.42
Tusie	28.73	1.55	3.09	49.72	123.83	252.58	42.00
Borah	34.78	0.65	2.24	57.52	346.65	70.00	333.08
Nyangumi	36.73	3.41	3.53	51.80	46.46	42.00	23.33
Ngiri	30.75	3.01	4.56	56.56	2.92	37.92	58.92
1012 B.1. (L)	43.32	0.92	3.50	58.16	93.72	26.25	312.08
Trophy	24.01	2.86	4.13	57.42	244.04	92.17	254.33

Means followed by the same letters down the columns are not significantly different at $LSD_{0.05}$

Appendix 1. ***Continue……***

Genotype	Biomass (tonnes.ha^{-1})	Yield (tonnes.ha^{-1})	Thousand kernel weight (g)	Hectolitre weight(kg.hl^{-1})	Area under Disease Progress Curve		
					Leaf rust	Stem rust	Yellow rust
Paka	24.58	2.73	3.36	57.16	71.93	172.65	88.67
Penjamo 62	23.84	2.14	4.55	55.39	83.48	399.58	158.68
Wabe	17.84	2.25	4.12	52.20	72.42	223.42	200.08
Norm	32.56	2.79	4.83	57.75	8.75	81.67	168.00
Bobwhite	27.35	1.89	3.30	56.57	19.74	90.42	306.83
Bonza	35.71	2.85	4.53	55.45	133.38	85.17	277.08
Sirbo	31.67	1.05	2.31	46.89	30.61	182.58	72.33
Kudu	35.33	2.59	3.36	50.33	530.86	215.83	14.58
Fanfare	29.02	1.69	4.48	57.42	172.57	58.92	300.42
Ngamia	24.74	2.55	4.28	55.04	42.64	116.67	236.25
Reliance 261M	26.54	0.63	2.47	53.42	225.04	277.08	152.25
Marquis	28.07	0.06	0.83	14.84	691.74	193.67	169.75
Leopard	34.83	1.29	4.15	36.78	170.76	99.17	144.67
Yombi	33.56	1.94	3.60	51.62	108.84	359.92	4.67
Bailey	53.07	0.32	2.65	35.14	43.09	25.07	84.00
Kenya -184-P	31.68	2.28	5.53	59.67	213.37	131.25	109.08
1061.K.4	42.96	0.94	2.93	53.57	17.58	146.42	148.75
Page	38.79	0.70	2.67	55.51	105.07	37.92	414.17
Menco	23.45	2.08	5.48	60.31	75.14	322.58	341.25
1010 F3 Sel. 4	43.51	0.45	2.80	46.35	57.29	200.83	230.42
Batu	21.54	1.25	3.04	43.42	131.73	224.58	263.08
Nyoka	32.60	3.32	4.74	58.32	107.06	178.50	126.00
Verde	28.93	3.28	4.08	59.78	3.67	75.83	221.67

Means followed by the same letters down the columns are not significantly different at $LSD_{0.05}$

Appendix 1. ***Continue*……**

Genotype	Biomass (tonnes.ha^{-1})	Yield (tonnes.ha^{-1})	Thousand kernel weight (g)	Hectolitre weight(kg.hl^{-1})	Area under Disease Progress Curve		
					Leaf rust	Stem rust	Yellow rust
Fahari	36.83	3.08	6.08	65.84	0.00	24.50	79.33
Mcvey	36.98	1.81	4.28	57.69	23.42	100.92	304.50
Newthatch	28.74	0.32	2.21	52.93	517.40	91.58	274.75
Njoro Bw II	25.72	2.30	3.74	58.81	157.54	82.83	247.92
K.318-AJ-4 A-1	35.31	0.11	1.56	24.47	378.71	63.83	105.58
Ci 14393	35.18	3.71	5.04	60.72	67.61	76.42	127.17
Paa	18.40	0.38	2.58	55.58	450.57	609.00	82.25
Impala	28.82	2.85	4.84	58.47	341.01	106.75	231.00
Fury	21.01	1.30	4.47	51.09	205.17	799.17	20.42
Salmayo	28.61	2.28	4.33	55.76	56.34	85.17	126.00
Minnpro	24.77	2.21	5.28	58.28	3.76	46.67	143.50
Fronthatch	45.30	0.73	2.84	53.59	8.75	43.75	35.00
Sonora63	9.03	0.29	2.18	41.89	316.44	602.00	429.33
Kenya 6820	35.32	2.59	4.85	61.19	61.31	73.50	46.67
Santa Elena	16.31	0.96	4.55	51.31	603.64	418.83	175.00
Cheetah	32.26	0.78	4.06	63.10	510.17	97.47	154.58
Duma	25.07	1.18	2.98	48.50	75.95	356.42	17.50
Inia66	19.26	1.92	4.12	53.03	258.69	95.08	193.67
Tembo	29.67	2.86	5.07	57.97	98.62	36.17	2.92
Ibis	34.66	3.61	4.51	59.92	404.52	71.17	2.92
Pasa	19.91	0.91	2.79	44.32	163.58	96.25	327.25
Bonanza	15.68	1.11	3.29	51.70	51.02	79.92	210.00
Dodota	31.38	1.76	3.26	45.43	94.08	504.00	169.17

Means followed by the same letters down the columns are not significantly different at $LSD_{0.05}$

Appendix 1. ***Continue*****……**

Genotype	Biomass (tonnes.ha^{-1})	Yield (tonnes.ha^{-1})	Thousand kernel weight (g)	Hectolitre weight(kg.hl^{-1})	Area under Disease Progress Curve		
					Leaf rust	Stem rust	Yellow rust
Nyati	27.17	2.26	4.33	53.85	71.97	33.83	93.92
Ii-50-17	31.74	1.05	4.37	59.37	71.63	82.83	87.50
Catcher	17.61	1.44	3.82	52.23	370.89	439.25	35.00
Bounty	39.74	2.72	4.31	53.42	33.82	43.75	35.00
Waldron	42.76	0.94	2.96	48.53	39.39	57.75	65.08
Sungura	37.88	1.48	3.09	53.36	55.20	49.00	49.58
Token-Ken	17.40	1.58	3.57	49.51	204.49	195.42	80.50
Enkoy	35.67	2.77	4.55	59.20	221.27	48.42	39.67
Zabadi	31.74	3.27	4.52	59.92	41.00	156.33	37.92
Africa Mayo	27.17	1.65	3.67	53.90	585.02	189.58	14.58
Goblet	27.31	2.08	3.89	60.92	164.40	69.42	265.42
Frontana	22.14	0.59	3.04	53.72	12.91	149.92	338.33
Sungura	49.38	1.92	2.90	64.82	46.67	35.00	10.50
Marshall	32.04	0.32	1.67	44.29	5.15	97.42	49.58
Heroe	24.75	0.78	2.57	48.83	83.75	210.00	134.75
Plume	48.31	1.72	4.44	56.77	11.67	93.33	53.08
Morris	46.09	0.55	2.66	48.22	19.33	20.42	157.50
Lenana	33.60	2.50	5.69	62.54	129.50	64.75	51.92
Popo	35.05	1.81	5.11	58.79	434.62	85.17	29.17
Kanga	28.55	3.18	4.55	60.07	92.00	93.33	71.75
Tama	34.68	3.10	5.24	65.33	63.75	102.08	163.33
Katar	26.46	3.12	3.74	52.11	53.85	284.08	89.25
Pw Thatcher	46.29	1.02	3.37	56.38	67.25	24.50	57.75
Yaktana 54A	16.68	0.57	2.82	48.38	202.18	349.42	438.08

Appendix 1. ***Continue*****……..**

Genotype	Biomass (tonnes.ha^{-1})	Yield (tonnes.ha^{-1})	Thousand kernel weight (g)	Hectolitre weight(kg.hl^{-1})	Area under Disease Progress Curve		
					Leaf rust	Stem rust	Yellow rust
Kenya Kifaru	38.63	0.96	4.11	47.91	291.11	127.17	333.08
Kalyanosona	10.91	0.44	3.17	44.16	164.00	484.17	417.67
Kenya 8	27.96	0.35	2.05	49.51	423.57	288.17	35.00
291 J.1.I.1	36.70	0.40	2.37	54.49	233.34	179.08	65.92
Kenya-122	31.23	0.07	1.23	22.85	399.31	350.00	201.83
Bacup	18.97	1.19	3.50	58.69	97.56	641.67	218.75
Polk	57.73	1.36	3.79	56.07	35.00	40.25	116.67
Ciano F67	16.84	1.96	3.88	57.05	159.27	345.92	126.58
Grange	37.41	0.89	2.79	44.92	106.04	166.83	382.08
K.Hawk	26.27	0.70	2.71	40.39	37.27	702.33	23.92
K.Sunbird	36.88	2.64	4.58	57.40	36.83	92.75	79.92
K.Tai	39.23	4.17	4.44	53.88	0.00	85.15	43.75
R1476	35.49	4.25	6.39	63.40	11.75	191.33	58.33
R1475	37.32	2.88	3.74	51.97	88.67	128.17	116.67
Kwale	24.77	1.48	3.05	47.97	212.27	50.75	186.67
Robin	29.01	0.74	2.56	44.49	114.07	637.00	0.00
K.Kingbird	26.62	4.64	5.25	58.49	49.67	39.67	1.75
K.Korongo	37.55	2.77	3.89	50.33	0.00	319.08	157.00
Eagle10	38.57	4.11	4.97	63.55	34.09	151.08	44.92
R1244	30.89	3.65	5.22	57.91	0.00	116.08	2.92
R1336	25.74	0.76	2.30	46.52	40.10	666.75	6.42
R1271	28.39	2.53	4.45	55.37	121.20	315.58	93.92
R1286	36.13	2.14	4.30	52.44	85.83	76.42	50.17
R1317	35.20	3.25	4.15	52.28	8.88	271.83	0.00

Means followed by the same letters down the columns are not significantly different at $LSD_{0.05}$

Appendix 1. ***Continue…..***

Genotype	Biomass (tonnes.ha^{-1})	Yield (tonnes.ha^{-1})	Thousand kernel weight (g)	Hectolitre weight(kg.hl^{-1})	Area under Disease Progress Curve		
					Leaf rust	Stem rust	Yellow rust
R1305	47.16	5.86	6.35	69.55	0.00	32.08	33.250
R1309	37.76	4.57	4.73	63.30	14.58	20.42	99.75
R1474	45.31	3.65	4.03	56.70	195.68	61.25	105.58
R1301	54.29	6.51	4.92	66.19	0.00	8.75	2.92
Morocco	6.41	0.19	1.27	42.21	27.86	1316.58	529.08
K. Chiriku	26.52	1.48	4.49	53.42	263.53	110.83	156.92

Means followed by the same letters down the columns are not significantly different at $LSD_{0.05}$

Appendix 2. Means of agronomic traits for wheat (*Triticum aestivum* L.) genotypes evaluated at KALRO, Njoro for main and off -season in 2016.

Genotype	Plant Height (cm)	Spike Length (cm)	Grain filling period(days)	Biomass (tonnes.ha^{-1})	Maturity (days)	Yield (tonnes.ha^{-1})	Thousand kernel weight(g)	Harvest Index
Simba	76.92	10.42	39.17	27.00	111.50	1.56	3.65	0.068
Beacon-Ken	102.18	10.14	29.00	33.15	106.33	2.33	3.44	0.078
KkBB	85.93	10.92	31.33	21.01	111.67	0.41	2.11	0.023
Mbega	81.53	11.71	33.17	32.10	115.00	0.83	2.22	0.040
Tama	99.13	8.88	43.33	34.57	110.50	3.23	5.07	0.087
690 F4 Sel.D.1	105.20	9.27	37.83	28.65	112.83	1.10	4.20	0.040
Kenya-294-B-2	101.34	10.12	37.67	17.41	108.50	1.11	4.23	0.085
Abola	80.61	10.34	36.83	18.11	110.00	1.04	3.17	0.067
Kenya 155	109.87	11.15	39.50	24.68	107.17	1.60	3.07	0.068
Sabanero	112.28	10.57	33.50	29.13	120.00	0.36	2.77	0.013
Marquillo	90.95	10.97	34.11	35.67	110.83	3.08	4.35	0.085
Zaragoza 75	72.61,,	8.94	34.33	33.28	117.67	1.38	4.15	0.040
Justin	104.08	9.27	32.50	44.15	123.00	0.31	1.62	0.005
Yaqui 50	100.76	9.80	31.17	27.56	103.83	2.34	3.85	0.090
Fletcher	80.62	9.39	28.83	56.01	118.00	0.71	2.68	0.015
Thatcher	90.53	8.64	14.00	28.36	59.33	0.28	1.35	0.008
Pewter	105.41	10.28	35.00	43.50	125.33	0.87	3.23	0.022
Shina	77.94	10.07	37.17	14.46	110.17	0.89	3.07	0.058
Era	75.66	9.51	34.17	44.59	125.00	0.77	2.92	0.018
Gabrino	98.81	9.39	42.50	49.73	119.00	2.19	4.30	0.047
Romany	100.76	9.45	34.33	34.78	108.17	2.76	4.49	0.102

Means followed by the same letters down the columns are not significantly different at $LSD_{0.05}$

Appendix 2. ***Continue……***

Genotype	Plant Height (cm)	Spike Length (cm)	Grain filling period(days)	Biomass (tonnes.ha^{-1})	Maturity (days)	Yield (tonnes.ha^{-1})	Thousand kernel weight(g)	Harvest Index
Et-12-D4	85.79	9.34	38.33	29.72	109.83	3.51	3.97	0.175
Kongoni	94.05	10.63	35.00	31.74	106.67	3.11	4.21	0.093
Kentana 48	100.18	9.17	32.67	21.74	113.67	0.94	2.88	0.062
Regent	91.89	10.57	33.17	28.04	124.67	0.81	2.68	0.030
Dashen	79.63	10.70	32.33	23.71	113.50	1.24	3.09	0.070
Kenya Civet	96.66	9.57	35.83	27.63	119.83	1.66	4.34	0.055
Bobicho	84.36	10.35	32.67	27.71	107.00	1.70	3.13	0.077
Gara	83.89	10.56	40.00	30.26	107.50	1.92	3.69	0.085
Chris	119.07	9.17	37.17	40.07	110.67	1.54	4.08	0.040
Tumbili	98.07	9.67	40.17	30.84	110.17	2.34	4.72	0.093
Pavon 76	83.04	10.22	37.00	22.09	109.17	2.29	3.96	0.152
Gem	113.23	10.16	34.67	36.18	115.50	1.88	4.52	0.057
Angus	85.38	11.03	38.67	52.18	126.00	2.15	3.34	0.047
1010 F3 Sel. 7	125.74	9.84	36.83	32.82	126.33	0.84	3.41	0.028
Tusie	85.38	9.39	36.17	28.73	111.17	1.55	3.09	0.065
Borah	74.74	10.45	31.21	34.78	114.32	0.65	2.24	0.015
Nyangumi	81.69	10.35	36.50	36.73	114.67	3.41	3.53	0.038
Ngiri	81.36	8.89	40.00	30.75	106.50	3.01	4.56	0.113
Sirbo	79.48	9.51	34.67	31.67	117.50	1.05	2.31	0.042
Penjamo 62	80.26	8.86	31.83	23.84	98.67	2.14	4.55	0.092
Ngamia	71.37	8.93	41.17	24.74	112.17	2.55	4.28	0.113
Bobwhite	72.46	9.34	34.00	27.35	105.67	1.89	3.30	0.072
Wabe	77.67	11.50	40.67	17.84	109.83	2.25	4.12	0.135

Means followed by the same letters down the columns are not significantly different at $LSD_{0.05}$

Appendix 2. ***Continue……..***

Genotype	Plant Height (cm)	Spike Length (cm)	Grain filling period(days)	Biomass (tonnes.ha^{-1})	Maturity (days)	Yield (tonnes.ha^{-1})	Thousand kernel weight(g)	Harvest Index
Ngamia	71.37	8.93	41.17	24.74	112.17	2.55	4.28	0.113
1012 B.1. (L)	100.56	10.07	33.83	43.32	125.00	0.92	3.50	0.022
Norm	85.70	8.70	38.67	32.56	110.67	2.79	4.83	0.082
Trophy	91.11 1	9.77	84.83	24.01	101.83(	2.86	4.13	0.122
Paka	81.39	9.92	40.17	24.58	106.67	2.73	3.36	0.133
Kudu	100.80	10.60	36.67	35.33	111.17	2.59	3.36	0.085
Bonza	101.94	9.41	33.17	35.71	108.33	2.85	4.53	0.088
Fanfare	97.61	9.38	31.17	29.02	102.50	1.69	4.48	0.077
Reliance 261M	104.03	10.67	32.67	26.54	110.17	0.63	2.47	0.023
Marquis	97.81	9.71	11.00	28.07	56.33	0.06	0.83	0.002
Leopard	103.20	9.81	36.00	34.83	120.33	1.29	4.15	0.037
Yombi	79.25	9.53	36.67	33.56	109.83	1.94	3.60	0.075
Bailey	108.30	9.59	35.30	53.07	111.60	0.32	2.65	0.005
Kenya -184-P	102.89	9.32	35.33	31.68	105.67	2.28	5.53	0.078
1061.K.4	106.39	11.19	29.17	42.96	116.83	0.94	2.93	0.020
Njoro Bw II	79.36	10.43	38.17	25.72	112.17	2.30	3.74	0.102
Paa	88.04	7.64	36.17	18.40	104.50	0.38	2.58	0.025
Page	99.42	10.81	31.67	38.79	118.33	0.70	2.67	0.017
Menco	91.89	9.19	39.83	23.45	106.67	2.08	5.48	0.118
1010 F3 Sel. 4	109.96	10.29	33.33	43.51	124.50	0.45	2.80	0.008

Means followed by the same letters down the columns are not significantly different at $LSD_{0.05}$

Appendix 2. ***Continue……***

Genotype	Plant Height (cm)	Spike Length (cm)	Grain filling period(days)	Biomass ($tonnes.ha^{-1}$)	Maturity (days)	Yield ($tonnes.ha^{-1}$)	Thousand kernel weight(g)	Harvest Index
Fronthatch	103.20	10.44	30.67	45.30	123.67	0.73	2.84	0.017
Verde	82.25	8.67	40.83	28.93	112.67	3.28	4.08	0.125
Batu	73.23	10.62	38.00	21.54	110.67	1.25	3.04	0.065
Nyoka	93.57	8.96	35.83	32.60	104.00	3.32	4.74	0.108
Ci 14393	105.63	8.02	42.50	35.18	111.00	3.71	5.04	0.093
Mcvey	98.91	8.92	36.83	36.98	113.83	1.81	4.28	0.055
Newthatch	91.44	8.55	31.75	28.74	108.79	0.32	2.21	0.012
Fury	87.54	9.47	39.83	21.01	102.67	1.30	4.47	0.087
Fahari	97.45	10.24	37.33	36.83	106.50	3.08	6.08	0.085
Kenya-318-AJ	95.99	8.94	14.17	35.31	61.33	0.11	1.56	0.003
Impala	86.96	8.58	41.33	28.82	111.00	2.85	4.84	0.095
Salmayo	100.50	7.71	38.00	28.61	104.33	2.28	4.33	0.082
Minnpro	84.31	9.53	37.00	24.77	106.83	2.21	5.28	0.118
Sonora63	73.02	8.71	30.50	9.03	96.00	0.29	2.18	0.037
Kenya 6820	110.13	10.10	28.83	35.32	103.67	2.59	4.85	0.073
Santa Elena	87.34	8.04	33.33	16.31	99.67	0.96	4.55	0.085
Cheetah	106.86	9.75	42.50	32.26	126.00	0.78	4.06	0.022
Frontana	112.03	10.77	35.67	22.14	119.17	0.59	3.04	0.028
Duma	87.17	11.46	38.33	25.07	108.33	1.18	2.98	0.052
Catcher	85.36	8.59	33.87	17.61	101.17	1.44	3.82	0.098
Dodota	85.54	9.97	37.17	31.38	106.17	1.76	3.26	0.065

Means followed by the same letters down the columns are not significantly different at $LSD_{0.05}$

Appendix 2. ***Continue……..***

Genotype	Plant Height (cm)	Spike Length (cm)	Grain filling period(days)	Biomass (tonnes.ha^{-1})	Maturity (days)	Yield (tonnes.ha^{-1})	Thousand kernel weight(g)	Harvest Index
Bounty	110.32	9.62	30.50	39.74	104.00	2.72	4.31	0.063
Inia66	90.81	8.74	33.17	19.26	97.67	1.92	4.12	0.100
Tembo	92.12	10.63	38.83	29.67	11.50	2.86	5.07	0.097
Ibis	77.26	9.90	36.83	34.66	110.50	3.61	4.51	0.110
Sungura	98.68	8.41	31.00	37.88	123.50	1.48	3.09	0.035
Waldron	108.68	9.36	35.50	42.76	122.50	0.94	2.96	0.022
Pasa	71.05	10.64	33.33	19.91	116.00	0.91	2.79	0.048
Bonanza	70.12	8.36	40.17	15.68	106.33	1.11	3.29	0.083
Ii-50-17	114.41	11.00	34.83	31.74	118.17	1.05	4.37	0.028
Mbuni	87.38	10.63	33.67	24.89	112.50	2.00	4.36	0.093
Nyati	91.17	8.72	34.67	27.17	103.83	2.26	4.33	0.095
Token-Ken	93.51	8.58	34.67	17.40	101.00	1.58	3.57	0.093
Enkoy	90.35	9.28	40.00	35.67	111.17	2.77	4.55	0.072
Zabadi	92.66	8.54	37.17	31.74	107.33	3.27	4.52	0.105
Tobari 66	73.3	8.83	40.00	22.06	106.00	2.26	4.44	0.128
K.Tai	92.04	11.79	35.67	39.23	110.00	4.17	4.44	0.098
Goblet	99.55	8.80	35.50	27.31	108.83	2.08	3.89	0.083
Sungura	95.62	8.39	33.07	49.38	107.96	1.92	2.90	0.035
Kalyanosona	66.46	8.79	33.67	10.91	106.33	0.44	3.17	0.048
Africa Mayo	109.86	10.04	29.17	27.17	103.83	1.65	3.67	0.053

Means followed by the same letters down the columns are not significantly different at $LSD_{0.05}$

Appendix 2. ***Continue……***

Genotype	Plant Height (cm)	Spike Length (cm)	Grain filling period(days)	Biomass (tonnes.ha^{-1})	Maturity (days)	Yield (tonnes.ha^{-1})	Thousand kernel weight(g)	Harvest Index
Yaktana 54A	93.19	9.22	33.50	16.68	107.83	0.57	2.82	0.040
Kanga	92.35	9.19	35.33	28.55	102.33	3.18	4.55	0.107
Tama	99.18	7.64	41.17	34.68	108.50	3.10	5.24	0.083
Katar	88.66	10.18	34.83	26.46	106.50	3.12	3.74	0.115
Pw Thatcher	117.06	9.85	36.18	46.29	111.52	1.02	3.37	0.027
Kenya 8	112.90	11.64	32.83	27.96	121.83	0.35	2.05	0.013
Kenya-122	107.69	10.74	15.17	31.23	59.83	0.07	1.23	0.002
Bacup	82.63	9.31	37.67	18.97	102.33	1.19	3.50	0.087
291 J.1.I.1	103.97	10.57	29.50	36.70	124.83	0.40	2.37	0.012
Kenya Kifaru	103.71	10.79	32.17	38.63	122.17	0.96	4.11	0.025
Polk	108.68	11.69	32.00	57.73	123.50	1.36	3.79	0.020
Ciano F67	66.44	8.19	34.00	16.84	98.83	1.96	3.88	0.145
Grange	106.54	10.60	31.67	37.41	125.83	0.89	2.79	0.023
K.Hawk	91.45	11.24	35.83	26.27	107.50	0.70	2.71	0.035
K.Sunbird	88.97	10.41	42.00	36.88	111.33	2.64	4.58	0.068
R1476	81.74	9.51	38.50	35.49	109.33	4.25	6.39	0.128
R1475	78.56	9.63	35.17	37.32	108.50	2.88	3.74	0.082
Kwale	78.61	11.20	32.33	24.77	114.00	1.48	3.05	0.090
R1336	89.29	11.75	34.00	25.74	105.83	0.76	2.30	0.035
K.Kingbird	76.44	8.94	37.00	26.62	105.55	4.64	5.25	0.172

Means followed by the same letters down the columns are not significantly different at $LSD_{0.05}$

Appendix 2. ***Continue…..***

Genotype	Plant Height (cm)	Spike Length (cm)	Grain filling period(days)	Biomass (tonnes.ha^{-1})	Maturity (days)	Yield (tonnes.ha^{-1})	Thousand kernel weight(g)	Harvest Index
K.Wren	90.94	12.29	39.50	34.69	110.17	2.46	3.78	0.073
Robin	87.73	11.83	29.50	29.01	101.67	0.74	2.56	0.027
R1286	90.34	11.74	37.83	36.13	116.33	2.14	4.30	0.092
R1271	85.37	9.14	38.33	28.39	107.50	2.53	4.45	0.075
K.Korongo	87.96	10.58	37.17	37.55	109.67	2.77	3.89	0.075
Eagle10	82.69	10.65	41.00	38.57	107.17	4.11	4.97	0.098
R1244	84.72	11.46	38.67	30.89	114.17	3.65	5.22	0.105
R1317	84019	11.23	39.67	35.20	111.67	3.25	4.15	0.078
R1474	77.18	9.97	39.17	45.31	113.67	3.65	4.03	0.090
R1305	84.70	9.96	39.50	47.16	111.33	5.86	6.35	0.103
R1301	93.20	10.80	45.17	54.29	115.17	6.51	4.92	0.085
R1309	89.22	10.89	38.67	37.76	111.50	4.57	4.73	0.085
Morocco	73.02	8.35	28.33	6.41	103.00	0.19	1.27	0.030
K. Chiriku	84.90	10.22	40.33	26.52	116.00	1.48	4.49	0.057

Means followed by the same letters down the columns are not significantly different at $LSD_{0.05}$

Appendix 3. Evaluation of 144 Kenyan wheat genotypes for seedling and adult plant resistance against leaf rust in Njoro over two seasons.

				SEASON 1				SEASON 2			
GENOTYPE		PEDIGREE	SIT	Ist score	2nd score	FDS	AUDPC	Ist score	2nd score	FDS	AUDPC
Kentana 48	(1948)	KENYA-C-9906/MENTANA	3^+ 4	5MS	20S	50S	507.5	10S	15S	15S	245.0
Rhodesian sabanero	(1949)	(S)SABANERO	1^- 2^-	5S	40S	50S	647.5	15MS	20S	20S	332.5
Kenya -184-P	(1951)	RELIANCE/KENYA-73-D	3^+ 4^+	5MS	30S	50S	577.5	20MS	25MSS	25MSS	420.0
Africa Mayo	(1960)	AFRICA/MAYO-48	;	10MS	40S	60S	735.0	20S	60S	60S	910.0
Mbega	(1963)	BONANZA/YECORA-70/3/F-35-75//KALYANSONA/BLUEBIRD	1^- 2^-	0	10S	10S	140.0	0	5MS	10MS	105.0
Tama	(1963)	YAKTANA-54/LERMA-52	1^-	10S	20S	30S	385.0	TR	TR	5MS	45.5
K. Page	(1963)	MENTANA/KENYA-58//BAGE/3/KENYA-184-P	1^- 2^-	5MS	20S	40S	437.5	0	0	0	0.0
Lenana	(1963)	YAQUI- 48 / KENTANA- 48	3^+	5S	20S	40S	437.5	0	0	0	0.0
Kenya Civet	(1966)	CI 12632 /3* KENYA 354	3^+ 4	0	50S	50S	700.0	20S	40S	40S	630.0
Kudu	(1966)	KENYA-131/KENYA-184-P	4^-	5MS	20S	50S	507.5	50MS	60S	60S	1015.0
K. Leopard	(1966)	LAGAEDINHI /3* KENYA 381P // CI 12632 /3* KENYA 354P	3^- 4	5MSS	30S	30S	437.5	10MS	10MS	10MS	175.0
Romany	(1966)	COLOTANA 261-51 / YAKTANA 54A	1^-	5MS	10S	20S	227.5	0	0	0	0.0
Token-Ken	(1966)	TIMSTEIN/2*KENYA//YAQUI-50	1^-	TR	40S	60S	703.5	10S	10S	10S	175.0
Bounty	(1966)	TIMSTEIN/2*KENYA//BONZA	3^- 4^-	TR	5S	10S	108.5	0	0	0	0.0
Tobari 66	(1966)	TEZANOS-PINTOS-PRECOZ/SONORA-64-A	3^+ 4	0	10S	10S	140.0	TR	TR	TR	17.5
Plume	(1966)	MIDA/MCMURACHY//EXCHANGE/3/KENYA-184-P	3^- 4^-	0	5S	5S	70.0	0	0	0	0.0
Grange	(1966)	KENYA-360-F/GRANADERO-KLEIN	3^+	5S	30S	50S	577.5	20S	50S	50S	770.0
Trophy	(1968)	TIMSTEIN/2*KENYA-RF-324//2*YAQUI-50	1^-	10MSS	30S	40S	525.0	10MS	10MS	10MS	175.0
Sungura	(1969)	ID 1877/MORRIS	1^+ 2^+	0	10S	15S	175.0	0	0	0	0.0
Nyati	(1973)	AFRICA-MAYO/2*ROMANY	2^-	0	5S	5S	70.0	0	10MS	10MS	140.0
Enkoy	(1974)	HEBRAND SEL/WISCONSIN 245/SUPRESA/3/2*FROCOR//FRONTANA/YAQUI /4/AGUILERA,KENYA 4500 L6A4	1^+ 2^+	10MS	15S	40S	420.0	10S	10S	10S	175.0
K. Paka	(1975)	WISCONSIN-245/II-50-17//CI-8154/2*TOBARI-66	3^+ 4	5MS	15S	30S	332.5	TR	TR	TR	17.5
K. Nyoka	(1975)	CI-8154/2*FEDERATION//3*ROMANY	1^- 2^-	TR	20S	40S	423.5	10MSS	10MSS	10MSS	175.0
K. Tembo	(1975)	WISCONSIN-245/II-50-17//CI-8154/2*TOBARI-66	3^+ 4	0	15S	50S	455.0	TR	TR	TR	17.5
K. Kifaru	(1976)	WIS.245/II-50-17//CI8154/2*FR/3/3*TOB66	1^+	5MS	50S	60S	787.5	20S	20S	20S	350.0

0=Immune, R= Resistant, MR=moderately resistant, MS=moderately susceptible, S=Susceptible, TR=trace resistant, MSS= moderately susceptible and susceptible (Johnston and Browder, 1966); AUDPC=Area under Disease Progress Curve; SIT=Seedling Infection Type, FDS= Final Disease Severity.0, 0;, 1, 2 = resistance response, 3 and 4 = susceptibility response

Appendix 3. ***Continue***

GENOTYPE		PEDIGREE	SIT	Ist score	2nd score	FDS	AUDPC	Ist score	2nd score	FDS	AUDPC
		TEZANOS-PINTOS-PRECOZ//SELKIRK-ENANO*6/LERMA-ROJO-64/3/AFRICA-MAYO-									
K. Nyangumi	(1979)	48/4/KENYA-SWARA/K-4500-6	1^-	0	5S	5S	70.0	10S	10MS	10S	175.0
K. Fahari	(1977)	TOBARI-66/3/SRPC-527-67//CI-8154/2*FROCOR	2^+	0	0	0	0.0	0	0	20MSS	140.0
		CORRECAMINOS/INIA-67//K-4500-2/3/KENYA-									
Zabadi	(1979)	SWARA//TOBARI-66/CIANO-67	$1^+ 2^+$	5MS	5S	20S	192.5	TR	5MS	5MS	73.5
K. Kongoni		CI-8154/2*FROCOR//3*ROMANY/4/WISCONSIN-									
(1981)		245/II-50-17/CI-8154//2*FROCOR/3/TOBARI-66	$3^+ 4$	10S	40S	40S	595.0	10S	50S	50S	735.0
		KLEIN-ATLAS/TOBARI-									
		66//CENTRIFEN/3/BLUEBIRD/4/KENYA-K.	$3^+ 4$	5MS	40S	50S		20S	40S		
K. Popo	(1982)	FAHARI					647.5			40S	630.0
KKBB	(1982)	KAVKAZ/KALYANSONA/BLUEBIRD	2^+	5MS	20S	40S	437.5	20S	20S	20S	350.0
		KTB/GIZA-155//NADADORES-63/T-238-1-5-8-17-10/3/KLEIN-ATLAS/TOBARI-									
Kenya Tumbili	(1984)	66//CENTRIFEN/BLUEBIRD	$1^+ 2^+$	10MS	15S	20S	280.0	20S	20S	20S	350.0
		KAVKAZ/3/SONORA 64/CIANO F 67//INIA F									
Kwale	(1987)	66/4/MAYA 74//BLUEBIRD/INIA F 66	$1^+ 2^+$	5MS	20S	50S	507.5	30S	30S	30S	525.0
Mbuni	(1987)	ZARAGOZA-75/3/LD-357-E/THATCHER//GALLO	$1^- 2^-$	0	20S	30S	350.0	15S	20S	20S	332.5
Pasa	(1989)	BUCK BUCK/CHAT	2^-	5MS	15S	30S	332.5	5S	5S	20MSS	192.5
K. Tai	(1969)	ND643/2*WBLL1	$1^- 2^-$	0	0	0	0.0	0	0	0	0.0
		BUCKY/MAYA-74/4/BLUEBIRD//HD-									
Ngamia	(1993)	832/OLESENS DWARF/3/CIANO 67/PENJAMO 62	$3^- 4$	5MS	5S	20S	192.5	5S	5S	5S	87.5
		AURORA/UP301//GALLO/SUPER									
Duma	(1993)	X/3/PEWEE/4/MAIPO/MAYA 74//PEWEE	1^-	TR	10S	20S	213.5	5S	10MSS	40MSS	367.5
K. Chiriku	(1989)	KTB/(SIB)CARPINTERO	$3^+ 4$	10S	30S	50S	595.0	10MS	40S	40S	595.0
Heroe	(1998)	MBUNI/SRPC-64//YRPC-1	1^-	0	20S	20S	280.0	0	0	10MS	70.0
Yombi	(1998)	MBUNI/SRPC-64//YRPC-5	2^+	0	20S	30S	350.0	20S	20S	20S	350.0
Simba	(2000)	PARULA/VEERY #6//MYNA/VULTURE	$3^+ 4^-$	0	0	5S	35.0	10S	15S	20S	280.0
		IAS-58/4/KALYANSONA/BLUEBIRD//CAJEME-									
Njoro Bw II	(2007)	F-71/3/ALONDRA/5/BOBWHITE	$3^- 4^-$	5MS	30S	40S	507.5	5S	10MS	10MS	157.5
Ibis	(2008)	KWALE/DUMA	$3^+ 4$	10MS	30S	50S	595.0	30S	40S	40S	665.0
Eagle10	(2011)	EMB16/CBRD//CBRD	;	5MS	10MS	10MS	157.5	5S	5S	5S	87.5
Robin	(2011)	BABAX/LR42//BABAX*2/3/TUKURU	1^-	0	5S	5S	70.0	15S	15S	20S	297.5
K. Sunbird	(2012)	ND643/2*WBLL1	3^+	0	10S	10S	140.0	5S	5S	5S	87.5
K.wren	(2012)	THELIN#2/TUKURU	1^-	0	0	0	0.0	5S	5S	5S	87.5

0=Immune, R= Resistant, MR=moderately resistant, MS=moderately susceptible, S=Susceptible, TR=trace resistant, MSS= moderately susceptible and susceptible(Johnston and Browder, 1966); AUDPC=Area under Disease Progress Curve; SIT=Seedling Infection Type, FDS= Final Disease Severity.0, 0;, 1, 2 = resistance response, 3 and 4 = susceptibility response

Appendix 3. ***Continue*……**

GENOTYPE		PEDIGREE	SIT	Ist score	2nd score	FDS	AUDPC	Ist score	2nd score	FDS	AUDPC
K..Kingbird	(2012)	TAM200/TUI/6/PVN//CAR422/ANA/5/BOW/CROW//BUC/PVN/3/YR/4/TRAP#1	1^{-} 2^{-}	0	20S	20S	280.0	TR	TR	TR	17.5
K.Korongo	(2012)	BABAX/LR42//BABAX*2/4/SNI/TRAP#1/3/KAUZ*2/TRAP//KAUZ	1^{-}	0	0	0	0.0	0	0	0	0.0
Kenya-294-B-2 A-3	(-)	AUSTRALIAN-26-A/KENYA-117-A	1^{-} 2^{-}	10MS	40S	40S	595.0	TR	TR	20S	150.5
Kenya 155	(-)	-	1^{-}	TR	40S	60S	703.5	30S	50S	50S	805.0
Reliance 261M	(-)	RELIANCE / KENYA 68	3	5MS	30S	50S	577.5	10S	10S	10S	175.0
Kenya-318-AJ-4 A-1	(-)	KENYA-112/CERES	3^{-}	5S	30S	40S	507.5	20MSS	20MSS	25MSS	385.0
Kenya 6820	(-)	-	2^{+}	5MS	10S	10S	157.5	5S	5S	5S	87.5
Cheetah	(-)	WARIGO/STERLING	3^{-}	5MS	40S	50S	647.5	20S	30S	30S	490.0
Kanga	(-)	-	1^{+}	5S	20S	20S	297.5	10MS	10MSS	10MSS	175.0
Kenya 8	(-)	-	1^{+} 2^{+}	10S	40S	50S	665.0	20MS	20MS	20MS	350.0
Kenya-122	(-)	MARQUIS/AGUILERA 8	1^{+}	10MS	40S	60S	735.0	5S	50S	50S	717.5
K.hawk	(-)		1^{-}	0	5S	5S	70.0	5S	10S	10S	157.5
Morocco	(-)		3^{+} 4	40S	80S	90S	1400.0	50S	60S	60S	1015.0
Marquis		HARD-RED-CALCUTTA	3^{+} 4	10S	50S	60S	805.0	10MS	40MS	40MS	595.0
Marquillo	(1926)	MARQUIS/(TR.DR)IUMILLO	3^{+} 4	0	5S	5S	70.0	TR	TR	TR	17.5
Thatcher	(1934)	MARQUIS/(TR.DR)IUMILLO//MARQUIS/KANRED	3^{+} 4	10S	40S	70S	805.0	30S	50S	50S	805.0
Regent	(1939)	H44/REWARD	3^{+}	10S	50S	50S	735.0	5S	20S	30S	367.5
Newthatch	(1944)	HOPE/THATCHER//2*THATCHER	1^{+}	5S	50S	70S	857.5	20S	30S	30S	490.0
Yaqui 50	(1950)	NEWTHATCH/MARROQUI-588	3^{-}	5S	50S	50S	717.5	5S	5S	5S	87.5
Yaktana 54A	(1954)	YAQUI-48/KENTANA-48//FRONTANA	3^{+}	5MS	30S	60S	647.5	5S	5S	5S	87.5
Justin	(1962)	CONLEY/ND-40-2	3^{-} 4^{-}	5MS	30S	40S	507.5	10MS	15MS	20MS	280.0
Gabrino	(1963)	KENTANA/RIO-NEGRO//GABO-54	;	10S	40S	40S	595.0	TR	TR	5S	45.5
Bonza	(1963)	YAQUI-50/KENTANA-48	3^{+} 4	5MS	20S	30S	367.5	TR	TR	10MS	80.5
Menco	(1963)	MENTANA / KENYA // FRONTANA / CINCO	1^{+} 2^{+}	5MS	15S	30S	332.5	0	0	10S	70.0
Salmayo	(1963)	SALLES/MCMURACHY//MAYO-48	2^{-}	5MS	10S	15S	192.5	10S	10S	10S	175.0
Catcher	(1963)	THATCHER/SANTA-CATALINA//FROCOR	3^{-}	5MS	40S	60S	717.5	20MS	20MS	20MS	350.0
Frontana	(1963)	FRONTEIRA/MENTANA	2^{+}	0	5S	5S	70.0	TR	TR	TR	17.5
Tama	(1963)	YAKTANA-54/LERMA-52	3^{-}	5MS	10S	10S	157.5	5S	5S	5S	87.5
Gem	(1964)	BT908 / FRONTANA // CAJEME 54	3^{+}	5MS	40S	50S	647.5	5S	5S	5S	87.5
Fronthatch	(1964)	FRONTANA / KENYA58 // NEWTHATCH	2^{-}	0	5S	5S	70.0	0	0	0	0.0

0=Immune, R= Resistant, MR=moderately resistant, MS=moderately susceptible, S=Susceptible, TR=trace resistant, MSS= moderately susceptible and susceptible (Johnston and Browder, 1966). AUDPC=Area under Disease Progress Curve; SIT=Seedling Infection Type, FDS= Final Disease Severity.0, 0;, 1, 2 = resistance response, 3 and 4 = susceptibility response

Appendix 3. ***Continue…….***

GENOTYPE		PEDIGREE	SIT	I^{st} score	2^{nd} score	FDS	AUDPC	I^{st} score	2^{nd} score	FDS	AUDPC
Pewter	(1964)	PW-327,USA/5*THATCHER	1^-	5MS	20S	30S	367.5	20MSS	20MSS	20MSS	350.0
Fury	(1964)	FROCOR/MENTANA/KENYA-2/MCMURACHY/YAQUI-50	$1^- 2^-$	0	20S	20S	280.0	15S	15S	15S	262.5
Chris	(1965)	FRONTANA/3*THATCHER/3/KENYA-58/NEWTHATCH//2*THATCHER	$3^+ 4$	0	10S	20S	210.0	5S	5S	5S	87.5
Bailey	(1966)	FRONTANA/4*THATCHER/3/THATCHER//KENYA58/NEWTHATCH/4/THATCHER/5/FRONTANA/4*THATCHER	$1^- 2^-$	0	10S	15S	175.0	5S	5S	5S	87.5
Goblet	(1967)	GABO-54/LERMA-52//GABO/3/KENYA/GENERAL-URQUIZA	$1^+ 2^+$	10S	30S	30S	455.0	10S	10S	10S	175.0
Ciano F67	(1967)	PITIC-62/(SIB)CHRIS//SONORA-64	1^-	0	40S	40S	560.0	5S	5S	5S	87.5
II-50-17	(1967)	FRONTANA//KENYA-58/NEWTHATCH	$1^+ 2^+$	0	10S	20S	210.0	5S	5S	5S	87.5
Kalyanosona	(1967)	FRONTANA // KENYA 58/ NEWTHATCH/3/NORIN 10 /BREVOR/4/ GABO 55	$1^+ 2^+$	5MS	30S	50S	577.5	0	0	TR	7.0
Beacon-Ken	(1968)	Frontana / Kenya 58 // Newthatch /3/3* Bonza	$3^- 4^-$	0	20S	20S	280.0	0	5S	5S	70.0
Waldron	(1968)	JUSTIN/ND-81	1^-	0	5S	15S	140.0	TR	TR	TR	17.5
Polk	(1968)	THATCHER / SUPREZA /3/ KENYA 58 / NEWTHATCH // FRONTANA	1^-	0	15S	15S	210.0	0	0	0	0.0
1010 F3 SEL. 7	(1969)	II-50-17/KENYA-184-P	3^-	0	10S	15S	175.0	5S	5S	5S	87.5
690 F4 SEL.D.1	(1969)	KENYA-360-H//2*MARQUIS/AGROPYRON ELONGATUM	$1^- 2^-$	5MS	30S	40S	507.5	5S	10S	10S	157.5
1012 B.1. (L)	(1969)	MENTANA/KENYA//BAGE/3/KENYA-184-P	3^+	0	15S	40S	385.0	10S	10S	10S	175.0
1061.K.4	(1969)	MIDA // McMURACHY / EXCHANGE /3/ RIO NEGRO	$3^- 4^-$	0	5S	15S	140.0	TR	TR	TR	17.5
1010 F3 SEL. 4	(1969)	II-50-17/KENYA-184-P	$1^- 2^-$	0	10S	10S	140.0	10S	10S	10S	175.0
Santa Elena	(1969)	SANTA-CATALINA-6/THATCHER//FROCOR	$3^+ 4$	20MS	50S	60S	840.0	5S	5S	5S	87.5
Bonanza	(1969)	PITIC-62/(SIB)CHRIS//SONORA-64	1^-	5S	5S	10S	122.5	5S	5S	5S	87.5
Fletcher	(1970)	II-55-10/4/PEMBINA/II-52-329/3/II-53-388/III-58-4//II-53-546	1^-	0	0	0	0.0	0	0	0	0.0
Penjamo 62	(1972)	FKN/NORIN 10 BREVOR	3^+	TR	15S	30S	318.5	5S	5S	5S	87.5
Borah	(1974)	NO-58/THATCHER//THATCHER/KENYA-FARMER/3/MN-III-58-1//FRONTANA/3*THATCHER	4^+	5S	40S	50S	647.5	40S	50S	50S	840.0

0=Immune, R= Resistant, MR=moderately resistant, MS=moderately susceptible, S=Susceptible, TR=trace resistant, MSS= moderately susceptible and susceptible (Johnston and Browder, 1966). AUDPC=Area under Disease Progress Curve; SIT=Seedling Infection Type, FDS= Final Disease Severity.0, 0;, 1, 2 = resistance response, 3 and 4 = susceptibility response

Appendix 3. ***Continue*****……**

GENOTYPE		PEDIGREE	SIT	Ist score	2nd score	FDS	AUDPC	Ist score	2nd score	FDS	AUDPC
Zaragoza 75	(1975)	MENGAVI/II-8156	3^{+} 4	5S	50S	60S	787.5	5S	5S	10MS	122.5
Era	(1970)	II-55-10/4/PEMBINA/II-52-329/3/II-53-388/III-58-4//II-53-546	1^{-}	0	5S	5S	70.0	0	TR	TR	14.0
Inia66	(1971)	LERMA ROJO 64/SONORA 64	3^{-}	10MS	40S	50S	665.0	10S	15MS	15MS	245.0
CI 14393	(1975)	FROCOR*2/4/COMETA/3/ NEWTHATCH// MENTANA/ MENKEMEN	1^{-}	5MS	15S	20S	262.5	5S	5S	5S	87.5
Sonora63	(1975)	YAKTANA-54//NORIN-10/BREVOR/3/2*YAQUI-54	3^{+} 4	10S	50S	60S	805.0	5S	5S	5S	87.5
Bobwhite	(1977)	AVRORA//KALYANSONA/BLUEBIRD/3/(SIB)WOODPECKER	3^{+} 4	0	5S	5S	70.0	0	5MS	5MS	70.0
Angus	(1978)	THATCHER/2*SUPREZA/3/FRONTANA//KENY58/NEWTHATCH/7/PEMBINA//FRONTANA/5*THATCHER/6/MIDA//KENYA-117-A/2*THATCHER/3/FRONTANA/4*THATCHER/4/MN-III-58-4/5/KENYA-58/NEWTHATCH//3*LEE	1^{-} 2^{-}	0	0	5S	35.0	0	0	0	0.0
ET-12-D4	(1981)	MAMBA/UQ105	3^{-} 4^{-}	0	5S	5S	70.0	10S	10S	10S	175.0
Marshall	(1982)	ERA/WALDRON	1^{-}	0	0	0	0.0	0	0	5MS	35.0
Pavon 76	(1982)	VICAM 571//CIANO F67/SIETE CERROS T	1^{-} 2^{-}	TR	15S	20S	248.5	10S	10S	10S	175.0
Paa	(1982)	KVZ/3/CNO/CHRIS//0N	1^{-}	10S	40S	50S	665.0	10S	40S	40S	595.0
Batu	(1984)	GALLO/CUCKOO//KAVKAZ/SUPER X	1^{-}	5MS	20S	35S	402.5	5S	5S	5S	87.5
Gara	(1984)	AVRORA//KALYANSONA/BLUEBIRD/3/(SIB)WOODPECKER	3^{-} 4^{-}	0	20S	20S	280.0	0	10MS	10MS	140.0
Dashen	(1984)	KAVKAZ/BUHO//KALYANSONA/BLUEBIRD	4^{-}	10S	50S	50S	735.0	20S	25S	30S	455.0
Minnpro	(1990)	MN-72299/MN-74115	3^{-} 4^{-}	0	0	0	0.0	5S	5S	5S	87.5
Norm	(1992)	MN-73167/MN-81070	2^{-}	0	5S	5S	70.0	0	0	0	0.0
Verde	(1995)	MN-7663/SBY-354-A	1^{-}	0	0	0	0.0	TR	TR	TR	17.5
Bacup	(1996)	NUY-BAY/PIONEER-2375//MARSHALL,USA	1^{+} 2^{-}	0	30S	30S	420.0	10S	10S	10S	175.0
Tusie	(1997)	COOK/VEERY//DOVE/SERI M82	2^{+}	5MS	20S	30S	367.5	TR	TR	TR	17.5
Abola	(1997)	BOBWHITE/BUCKBUCK	-	TR	20S	30S	353.5	TR	5MS	5MS	73.5
Shina	(1998)	GOLDEN-VALLEY(GOV)/AZTECA-67//MUSALA/3/R-37/GHL-121//KALYANSONA/BLUEBIRD/4/ANI BLUEJAY/COCORAQUE F	1^{+} 2^{+}	5MS	40S	50S	647.5	5S	5S	5S	87.5
Dodota	(2001)	75//PARULA/BOBWHITE	3^{-}	5MS	20S	20S	297.5	10S	10S	10S	175.0

0=Immune, R= Resistant, MR=moderately resistant, MS=moderately susceptible, S=Susceptible, TR=trace resistant, MSS= moderately susceptible and susceptible (Johnston and Browder, 1966). AUDPC=Area under Disease Progress Curve; SIT=Seedling Infection Type, FDS= Final Disease Severity.0, 0;, 1, 2 = resistance response, 3 and 4 = susceptibility response

Appendix 3. C*ontinue*……

GENOTYPE		PEDIGREE	SIT	Ist score	2nd score	FDS	AUDPC	Ist score	2nd score	FDS	AUDPC
Sirbo	(2001)	VS73.600/MRL/3/BOBWHITE//YECORA F 70/TRIFON	3^+ 4	0	10S	10S	140.0	5S	5S	5S	87.5
Bobicho	(2002)	PEREGRINE/PF70354/KALYANSONA/BLUEBIRD/ALONDRA/3/MARINGA	3^- 4^-	TR	15S	40S	388.5	20MS	30MS	30MS	490.0
Mcvey	(1999)	NING-8331/MN-87029//MN-89068	1^- 2^-	0	5S	10S	105.0	TR	TR	TR	17.5
Katar	(1999)	COOK/VEE''S''//DOVE''S''/SERI/3/BJY''S''	1^- 2^-	5S	5S	10S	122.5	5S	5S	5S	87.5
Wabe	(-)	MIRLO/BUCKBUCK	1^+	TR	15S	30S	318.5	0	0	5MS	35.0
Fanfare	(-)	-	3^+ 4	5MS	30S	30S	437.5	10MS	10MS	10MS	175.0
Impala	(-)	-	3^-	10MSS	50S	60S	805.0	40S	50S	50S	840.0
Morris	(-)	THATCHER//KENYA-117 A/MIDA/3/FRONTANA/4*THATCHER/4/THATCHER/5/FRONTANA/4*THATCHER	2^-	0	5S	10S	105.0	0	TR	TR	14.0
PW Thatcher	(-)	THATCHER/AGENT	1^+	5MS	10S	20S	227.5	TR	TR	TR	17.5
291 J.1.I.1	(-)	AUSTRALIA 26 / KENYA 58	3^+	0	40S	40S	560.0	10S	10S	10S	175.0
R1476			1^- 2^-	0	5S	5S	70.0	TR	TR	TR	17.5
R1475			4	TR	30S	30S	423.5	0	0	0	0.0
R1244		PRINIA/3/ALTAR84/AE.SQ//2*OPATA/4/CHEN/AEGILOPS SQUARROSA (TAUS)//BCN/3/BAV92	;	0	0	0	0.0	0	0	0	0.0
R1336		BABAX/LR42//BABAX*2/3/TUKURU	;	0	0	0	0.0	5S	10S	10S	157.5
R1271		PBW343*2/KUKUNA*2//YANAC	1^+ 2^+	5MS	20S	40S	437.5	5S	5S	5S	87.5
R1286		QUAIU/3/PGO/SERI/BAV92	3^+ 4	10S	10S	20S	245.0	5S	5S	5S	87.5
R1317		KSW/7/CAL/NH/H567.71/3/SERI/4/CAL/NH//H567.71/5/2*KAUZ/6/PASTOR/8/CAL/NH//H567.71/3/SERI/4/CAL/NH//H567.71/5/2*KAUZ/6/PASTOR	1^-	0	0	0	0.0	5S	5S	5S	87.5
R1474			3^+ 4	5MS	20S	40S	437.5	5MS	10MSS	15MSS	192.5
R1305		KSW/5/2*ALTAR 84/AE.SQUARROSA (221)//3*BORI95/3/URESJUN/KAUZ/4/WBLLI	3^- 4^-	0	0	0	0.0	0	0	0	0.0
R1301		KSW/5/2*ALTAR 84/AE.SQUARROSA (221)//3*BORI95/3/URESJUN/KAUZ/4/WBLLI	3^+	0	0	0	0.0	0	0	0	0.0
R1309		KFA/5/REH/HARE//2*BCN/3/CROC-I/AE.SQUARROSA(213)//PGO/4/HUITES/6/REH/HARE//2*BCN/3/CROC-I/AE.SQUARROSA(213)//PGO/4/HUITES	1^- 2^-	5MS	5MS	5MS	87.5	0	0	0	0.0

0=Immune, R= Resistant, MR=moderately resistant, MS=moderately susceptible, S=Susceptible, TR=trace resistant, MSS= moderately susceptible and susceptible (Johnston and Browder, 1966) . AUDPC=Area under Disease Progress Curve; SIT=Seedling Infection Type, FDS= Final Disease Severity.0, 0;, 1, 2 = resistance response, 3 and 4 = susceptibility response