PHENOTYPING INTRODUCED BREAD WHEAT GENOTYPES FOR RESISTANCE TO STEM RUST (*Puccinia graminis* f. sp. *tritici*) IN KENYA

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A Thesis Submitted to the Graduate School in Partial Fulfillment of the Requirements for the Master of Science Degree in Plant Breeding of Egerton University

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DECLARATION AND RECOMMENDATION

Declaration

This thesis is my original work and has not been presented in this university or any other for the award of a degree.

16-11-2021

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Recommendation

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DEDICATION

To my late mother, Antonina Naswa Wafula, and father, Hudson Wasike, who taught me the value of education and my sisters, Lilian, Pascalia, and Maureen and brothers, Noah, Kevin, and James, for their unreserved love and struggle in my upbringing.

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ABSTRACT

Stem rust (*Puccinia graminis* f. sp. *tritici*) causes high yield losses in wheat (*Triticum aestivum* L.). Ameliorating this challenge requires a multifaceted approach, the most plausible being genetic resistance. The objective of this study was therefore to determine genotypic variation for adult and seedling resistance to stem rust, grain yield (GY) and agronomic performance. Sixty-two introduced Australian wheat genotypes and two controls, Cacuke and Kenya Robin, were planted in a field experiment over two seasons in a partially balanced *lattice*-square design with three replicates. Adult plant resistance (APR) was assessed based on area under disease progress curve (AUDPC), coefficient of infection (CI) and final disease severity (FDS) with genotypes scoring ≤ 300, ≤ 20 and ≤ 30, respectively, being resistant. APR was identified in seven genotypes. Effect due to genotype, season and genotype-by-season interaction was significant ($p \leq 0.05$) for AUDPC, CI, FDS, GY, 1000-kernel weight (TKW) and test weight (TW). The range for GY, TKW and TW was 0.26-3.37 t ha^{-1}, 8.9-28.3 g and 41.4-74.5 kg hL^{-1}, respectively. In the greenhouse experiment, genotypes were inoculated with isolates *TTKSK* and *TTKTT* at the 2-leaf stage and infection types (ITs) scored after fourteen days. Eleven genotypes were identified for seedling resistance (ITs $\leq$ 2+) to both isolates. Genotypes Sunguard, Lancer and Gauntlet were uncovered for APR, seedling resistance, high yield performance and stability in resistance and, therefore, should be considered for inclusion in breeding programmes for resistance to stem rust and candidates for national performance trials for potential release to farmers.

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LIST OF ABBREVIATIONS AND ACCRONYMNS

APR	Adult Plant Resistance
AUDPC	Area Under Disease Progress Curve
Avr	Avirulent
Bdv	Barley yellow dwarf virus
BGRI	Borlaug Global Rust Initiative
cd sr m^{-2}	*Candela Steradian* per square metre
CI	Coefficient of Infection
CIMMYT	International Maize and Wheat Improvement Centre
DGGW	Delivering Genetic Gain in Wheat project
FAO	Food and Agriculture Organization of the United Nations
FAS	Foreign Agricultural Services
FCRI	Food Crops Research Institute
GDP	Gross Domestic Product
HI	Harvest Index
HPRs	Host plant reactions
ITs	Infection types
KIPPRA	Kenya Institute for Public Policy Research and Analysis
KNBS	Kenya National Bureau of Statistics
Lr	Leaf rust
Ltn	Leaf tip necrosis
masl	metres above sea level
NB-LRR	Nucleotide-Binding Leucine-Rich-Repeats
NPT	National Performance Trials
pa	per annum
Pm	Powdery mildew
QTL	Quantitative Trait Loci
REML	Restricted Maximum Likelihood
RH	Relative Humidity
Sr	Stem rust
USDA	United States Department of Agriculture
Yr	Yellow rust

CHAPTER ONE
INTRODUCTION

1.1 Background information

Wheat (*Triticum aestivum* L.), rice (*Oryza sativa* L.) and maize (*Zea mays* L.) are the most important cereal grains globally (FAO, 2022). Wheat is a cereal fruit (caryopsis) (Mauseth, 2017) which was first domesticated in the Fertile Crescent (Levant) at around 9600 BC but is now cultivated globally (Lev-Yadun *et al*., 2000; Maeda *et al*., 2016). In 2020, world production of wheat was about 760.9 million tonnes (t) from nearly 219.0 million hectares (ha), translating to a yield of 3.5 t ha^{-1} (FAOSTAT, 2022). The largest wheat producers are the European Union (155 million t; 20.2%), China (134.3 million t; 17.5%), India (107.6 million t; 14.1%), the Russian Federation (85.3 million t; 11.1%), the United States (49.7 million t; 6.5%), Canada (35.2 million t; 4.6%) and Australia (30 million t; 3.9%) (USDA-FAS, 2022). Wheat is cultivated on ~17% of all crop area across a wide range of environments from 67º N in Scandinavia and Russia to 44º S in Argentina and Chile, including tropics and sub-tropics, up to 4,570 metres above sea level (masl) in Tibet (Ecocrop, 2022; Hodson & White, 2007).

Wheat consumption has more than tripled since the advent of the Green Revolution (Godfray *et al*., 2010; Pingali, 2012) and, with a volume of > 187.5 million t in 2020 (FAO, 2022), wheat accounts for more than a third of the world trade in grains by financial value (> $50 billion) (Enghiad *et al.*, 2017). On average, its demand increases by 1.7% per annum (pa) and it is projected that 60% more wheat will be needed by 2050 (McKenzie & Williams, 2015). In Kenya, average annual production and consumption of wheat from 2015 through 2019 stood at 263,900 t and 1,943,200 t, respectively, implying that > 85% was imported (KNBS, 2020). This is a worrying trend considering the fact that agriculture accounts directly and indirectly for a combined 61.2% of the Gross Domestic Product (GDP) (KIPPRA, 2020).

Stem (syn. black) rust (*Puccinia graminis* Pers. f. sp. *tritici* Eriks. and E. Henn.), stripe (syn. yellow) rust (*Puccinia striiformis* Westend. f. sp. *tritici* Eriks.) and leaf (syn. brown) rust (*Puccinia triticina* Eriks. f. sp. *tritici*) are the major foliar diseases of wheat, with stem rust being the most destructive (Chen, 2020; Lewis *et al*., 2018; Szabo *et al*., 2014). In an effort to manage the stem rust disease, the Kenyan wheat breeding programme in collaboration with the International Maize and Wheat Improvement Centre (CIMMYT), Mexico have released a number of stem rust resistant varieties which are high yielding. However, the stem rust disease still poses a major challenge to wheat farming both locally

and globally. This is due to the rapid emergence of new and more virulent races (variants) of the pathogen arising from mutation thereby rendering genotypes that previously were resistant to existing races to be vulnerable (Lewis *et al*., 2018; Saunders *et al*., 2019). The detection of the highly virulent *Ug99* race in Uganda in 1999 demystified the earlier conception by wheat scientists that stem rust was a 'conquered' disease (Pretorius *et al*., 2000). This race 'broke down' the stem rust (sr) resistance gene *Sr31* on translocation 1BL.1RS [*Sr31*/*Yr9*/*Lr26*/*Pm8*] from rye (*Secale cereale* L., 2*n* = 2*x* = 14, RR) cv. Petkus which was the key gene conferring resistance in commercial varieties for more than 30 years (Evanega *et al*., 2014; Mago *et al*., 2005; Yediay *et al*., 2010). The *Ug99* race comprises several variants and possesses virulence to a large number of other genes which are important for resistance to stem rust in widely grown cultivars hence preventing sustainable production of wheat because only a few genes are effective (Rahmatov *et al*., 2019; Wessels *et al*., 2019). Stem rust races *TTKSK*, *PTKTK*, *TTHST*, *TTKST*, *TTTSK*, *PTKSK*, *PTKST*, *TTKTT*, *TTKTK*, *TTTTF*, *TKTTF*, *TTHSK* and *TTKTT*+ [*TTKTT*+*Sr8155-B1*] (detected in Kenya) and *TTKSP*, *TTKSF* and *TTKSF*+ differ in virulence, aggressiveness and fitness to survive (Olivera *et al*., 2017; Patpour *et al*., 2016). These races are virulent to deployed stem rust resistance genes *Sr5*, *Sr6*, *Sr7a*, *Sr7b*, *Sr8a*, *Sr9a*, *Sr9b*, *Sr9f*, *Sr10*, *Sr15*, *Sr16*, *Sr18*, *Sr19*, *Sr20*, *Sr23*, *Sr24*, *Sr30*, *Sr36* and *Sr41* from *T. aestivum*; *Sr9d*, *Sr9e*, *Sr9g*, *Sr11*, *Sr12* and *Sr17* from *T. turgidum*; *Sr21* from *T. monococcum*; *Sr34* from *T. comosum* and *Sr38* from *T. ventricosum* (Singh *et al*., 2015).

The stem rust pathogen mutates and spreads rapidly and, currently, sixteen (16) single-step mutation variants have been identified in thirteen (13) countries in Africa and the Middle East and is likely to move further into breadbaskets of Asia and beyond (Bhavani *et al*., 2019; Sharma *et al*., 2013). More than 80% of worldwide wheat germplasm tested in Kenya exhibit inadequate levels of resistance to stem rust (Bhavani *et al*., 2019; Macharia & Ngina, 2017). A conservative estimation by Reynolds and Borlaug (2006) put the total wheat area affected by this disease globally at more than 50 million ha. To date, > 90% of cultivated wheat is susceptible to stem rust, therefore, over 150 million t of wheat is at risk of being destroyed annually in the absence of resistance (Braun, 2011; Pardey *et al*., 2013). This risk is greatest in less-developed countries in which approximately 2.5 billion people directly and indirectly depend on wheat for sustenance (Singh *et al*., 2015).

1.2 Statement of the problem

Among rust diseases of wheat, stem rust (*Puccinia graminis* f. sp. *tritici*) is the most important. It causes economic losses of up to 100% by interrupting essential physiological processes in wheat, including photosynthesis and nutrient mobilization. Instantaneous emergence and spread of new variants having broad and complex virulence characteristics has rendered ≥ 90% of Kenyan wheat cultivars susceptible to this disease. This has allowed large populations of stem rust races to proliferate, therefore, creating a reservoir of this pathogen. Despite numerous mitigation efforts, stem rust races continue to evolve making the existing resistance to be short-lived. Deployment of resistant cultivars has been effectively used against stem rust, however, the emergence of virulent variants through mutation, recombination and selection has posed a challenge to the use of host plant resistance as a control strategy. Farmers are compelled to rely on fungicides for a profitable crop, whose frequent use not only poses adverse effects to human health and the environment but also increases the cost of production. This is a disincentive particularly to small-scale farmers. The use of resistance genes from diverse sources has the potential to confer adult plant resistance and ease the disease burden. Therefore, there is need for continuous search for additional sources of stem rust resistance genes for introgression into adapted cultivars. Such a strategy enhances resistance to stem rust through pyramiding of genes. Subsequently, accumulation of durable resistance genes alongside selection for grain yield could culminate into deployment of new varieties to improve food security and livelihood of Kenyan farmers.

1.3 Objectives

1.3.1 Broad objective

To contribute to improved food security and economic livelihoods in Kenya through development of wheat cultivars that are resistant to stem rust disease.

1.3.2 Specific objectives

i. To determine genotypic variation for resistance to stem rust at adult plant stage, grain yield and agronomic performance among the introduced wheat genotypes.
ii. To determine genotypic variation for resistance to stem rust at seedling stage among the introduced wheat genotypes.

1.4 Hypotheses

i. There is no genotypic variation among introduced wheat genotypes for resistance to stem rust at adult plant stage, grain yield and agronomic performance.
ii. There is no genotypic variation among the introduced wheat genotypes for resistance to stem rust at seedling stage.

1.5 Justification of the study

Wheat is one of the most widely consumed cereal grains worldwide and therefore important for food security. However, stem rust remains a devastating disease to adapted Kenyan wheat cultivars due to the transient nature of their resistance. It causes up to 100% yield losses in highly susceptible cultivars if conditions are favourable to its infection and development highlighting the need for scientific interventions. Although mitigation measures including host plant resistance have been undertaken, the stem rust disease has persisted mainly due to rapid evolution of the pathogen. Whilst these measures could be convenient in managing the disease, justifiable economic returns cannot be demonstrated. Nevertheless, cultivation of resistant cultivars, particularly those possessing adult plant resistance, remains a cost effective and sustainable strategy for the management of stem rust. The continuous search for new sources of resistance is held as affective against current and emerging races and a useful genetic resource for wheat improvement. Subsequently, introgression of novel genes into adapted Kenyan wheat cultivars from diverse sources followed by selection is a promising approach confers durable resistance to stem rust hence contributing to reduced production costs and improved yield among small-scale farmers.

CHAPTER TWO
LITERATURE REVIEW

2.1 Introduction

Wheat (*Triticum* spp.) is one of the eight crops which formed the basis for human societies' transition from hunting and gathering to a sedentary agrarian lifestyle during the Neolithic period (Avni *et al*., 2017; Bilgic *et al*., 2016; Zohary *et al*., 2012). Bread wheat (*Triticum aestivum* L.) is a self-pollinated annual grass in tribe triticeae of family poaceae and is staple food to a large proportion of the world population (Clayton *et al*., 2015; Shiferaw *et al*., 2013). It has been grown in Kenya since the early 1900s and its improvement through introductions, hybridization and selection started around 1906 (Dixon, 1960; Hurd *et al*., 1969; Pinto & Hurd, 1970). The origin of wheat in Kenya is traced to Australian founder lines followed by Egyptian, Italian and Canadian germplasm (Dixon, 1960; Hurd *et al*., 1969; Macharia & Ngina, 2017; Pinto & Hurd, 1970). Cultivation of wheat was confined to large-scale farms in highland areas of high (Mau Narok and Timau) and low (lower Narok and Laikipia) rainfall and acidic soils (Uasin Gishu and Trans Nzoia) (Wanyera & Wanga, 2016). Currently, it is grown on ~150,000 ha but commercial production is still limited to medium-to-high-altitude areas of 1,500 to 3,000 masl, respectively, and 800-2000 mm of rainfall annually (Macharia & Ngina, 2017). Wheat is produced both by large-scale (> 2.5 ha; 20%) and small-holder (≤ 2.5 ha; 80%) farmers accounting for 80 and 20% of the produce, respectively. This is due to expensive farm inputs, especially, fungicides (Alemu & Mideksa, 2016).

Since antiquity, rusts (*Puccinia* spp.) have caused high yield losses in wheat (Bhavani *et al*., 2019; Szabo *et al*., 2014). This is due to the narrow genetic base of resistance in the elite wheat breeding gene pools resulting from strong selective breeding (Cavanagh *et al*., 2013; Voss-Fels *et al*., 2015). In Kenya, the problem of rust diseases is as old as the crop has been in cultivation (Macharia *et al*., 2015) with a severe devastation occurring during 1906-1917 (Pinto & Hurd, 1970). Recurrent epidemics of rusts, particularly stem rust, makes the country to be heavily dependent on importation of wheat which is highly vulnerable to price fluctuation in the world market (Macharia, 2015). This is against the backdrop of increasing consumption needs conservatively estimated in 2020 to be > 150% of local production (FAOSTAT, 2022). The situation is further exacerbated by the emergence and spread of new races of the pathogen (Lewis *et al*., 2018; Saunders *et al*., 2019). Their broad virulence combinations is a threat to wheat production (Sharma *et al*., 2013). Over the past few years,

yield losses of 20-70% have been reported in Kenya with a possibility of total loss of the crop during epidemic years (Singh *et al*., 2015; Wanyera & Wanga, 2016). Globally, more than 5,000 species of rust pathogens are known to attack plants. However, stem rust (*P. graminis*), stripe rust (*P. striiiformis*) and leaf rust (*P. triticina*) are of the most economic importance to wheat due to the magnitude of induced losses (Chen, 2020; Marsalis & Goldberg, 2016). To enhance efficiency in breeding for durable resistance to stem rust, it is necessary that the genetic basis of the disease is understood. To date, more than 60 stem genes have been catalogued in wheat as part of the International Wheat Genetics Symposium (IWGS) gene catalog (McIntosh *et al*., 2017; Randhawa *et al*., 2018).

2.2 Origin and evolution of wheat

Earliest cultivated forms of wheat, diploid einkorn (*Triticum monococcum* L., 2*n*=2*x*=14) and tetraploid emmer (*T. dicoccum* L., 2*n*=4*x*=28) are landraces which originated from south eastern parts of the present-day Turkey (Faris, 2014; Zhu *et al*., 2019; Zohary *et al*., 2012). Modern wheat cultivars primarily comprise of two polyploid species: hexaploid bread wheat (*T. aestivum* L., 2*n*=6*x*=42) (95%) and tetraploid hard or the durum-type of wheat (*T. turgidum* L. subsp. *durum* (Desf.) Husnot, 2*n*=4*x*=28, AABB) (5%) (Belderok *et al*., 2000; Maccaferri *et al*., 2019). The latter is largely used for making *macaroni* and low-rising bread. Wheat is also classified either as spring (3-4 months; 65%) or winter/facultative (6-11 months; 35%) based on its growth habit (Braun & Sãulescu, 2002). Cultivation of wheat reached the Near East 9000 years ago when hexaploid bread wheat appeared (Faris, 2014; Zohary *et al*., 2012). Wheat has a 96% chance of being self-pollinated, therefore, genetic diversity resides in her wild relatives, global germplasm collections and in more than 25,000 cultivars worldwide (Jovovic *et al*., 2020; Vikram *et al*., 2016).

Primitive forms of wheat had hulled grains and brittle ears that disarticulate at maturity into individual spikelets, with each spikelet having a wedge-shaped rachis internode at its base and an arrow-like device that inserts the seed in the ground (Pourkheirandish *et al*., 2018; Zohary *et al*., 2012). In the course of its domestication, farmers selected for desirable traits like yield, loss of shattering of spikes at maturity and free-threshing forms in which glumes do not adhere tightly to grains (Golan *et al*., 2015; Haas *et al*., 2019; Sharma *et al*., 2019). Ease of harvest and suitability for storage were critical for domestication (Haas *et al*., 2019; Lev-Yadun *et al*., 2000). In these cultivated forms, ears were not brittle and remained intact after maturation, therefore, relying on farmers for sowing, reaping and threshing

(Sharma *et al*., 2019). Non-shattering, a trait which is controlled by mutation at the *Brittle Rachis 1* (*TtBtr1*) locus (Nalam *et al.,* 2006; Pourkheirandish *et al*., 2018), ensures that seeds are not dispersed in their natural habitat. Non-brittleness and nakedness of cultivated forms is controlled by the *Q* locus (Luo *et al*., 2000) on chromosome 5 of genome A and is thought to have arisen through a series of mutations of gene *q* in hulled cultivars (Simons *et al*., 2006). In addition, this gene controls spike length, plant height and grain yield (Kowalski *et al*., 2016).

2.3 Botany and genetics of wheat

Wheat (*Triticum aestivum* L.) is an annual flowering, vascular and monocotyledonous grass in the family poaceae, subfamily pooideae, tribe triticeae, subtribe triticinae and genus triticum (Clayton *et al*., 2015). Wheat is propagated by seed and is predominantly self-pollinated where it undergoes sexual reproduction. The seed utilises available moisture and carbohydrate reserves to develop a primary root and a coleoptile that grows into a shoot (Junaidi *et al*., 2018; Lafon-Placette & Köhler, 2014). Wheat develops seminal (main) and nodal (crown) root systems in the top 30 cm of soil for absorption of water and nutrients (Junaidi *et al*., 2018; Zhang *et al*., 2003). The plant develops a central stem with leaves emerging at opposite sides (Setter & Carlton, 2000). Its shoot is made up of a series of phytomers, each having a node, a hollow internode, a leaf and a tiller bud at leaf axils (Kirby, 2002). The leaf sheath wraps the stem to provide support and the stem terminates in the ear(s) (Bowden *et al*., 2007). Leaves have an epidermal layer that is enclosed in epicuticular wax and the mesophyll is transected by vascular tissues. There is also a membranous ligule and a pair of hairy projections (auricles) at the base of each leaf blade (Kirby, 2002).

Tillers emanate from the main stem to produce leaves and potentially ears (Setter and Carlton, 2000). The ear (spike) has two rows of spikelets which are made up of florets on either side of a central rachis (Reale *et al*., 2017; Setter & Carlton, 2000). Each floret (later kernel) is enclosed by a lemma and a palea, where the top of the lemma may form an awn (Li *et al*., 2010; Reale *et al*., 2017). They consist of carpel (ovary and stigma) and stamen (three anthers and a filament), with each anther having four nutritive layers (loculi) that enclose pollen grains (microspores) (Kirby, 2002; Reale *et al*., 2017). The kernel usually measures ~ 8 mm in length and ~35 mg in weight (Faltermaier *et al*., 2014). It is either elliptical or oval in shape with short or long brush hairs and is composed of 80-85% endosperm, 13-17% bran and 2-3% germ based on dry matter (Jaaskelainen *et al*., 2013; Ndung'u *et al*., 2016).

Genera triticum and aegilops comprise of 13 diploid and 18 allopolypoid species developed through hybrid speciation (Borrill *et al*., 2015; Faris, 2014). They possess the same number of base chromosomes (*n*=7) in 3 ploidy levels (diploid; 2*n*=2*x*=14, tetraploid; 2*n*=4*x*=28 and hexaploid; 2*n*=6*x*=42), therefore, the genomic sequence of bread wheat is extraordinarily large (15,961 base pairs) (Zimin *et al*., 2017). However, only 1-5% of DNA represent genes and 83-90% is repetitive (Zhu *et al*., 2019). Bread wheat (*Triticum aestivum* L.) (2*n*=6*x*=42, BBAADD) is an allohexaploid species whose donors include *Aegilops speltoides* (2*n*=2*x*=14; BB), *Triticum urartu* (2*n*=2*x*=14; AA) and *Aegilops tauschii* (2*n*=2*x*=14; DD) (Ling *et al*., 2018; Luo *et al*., 2017; Tang *et al*., 2018) and it forms 21 pairs of homeologous chromosomes during meiosis (Glémin *et al*., 2019). The three ancestors are related by descent and ancestral hybridization (Marcussen *et al*., 2014) but have undergone ancient polyploidization events and reverted back to their diploid status (Glémin *et al*., 2019; Pont & Salse, 2017). The interaction of multiple genomes in a single cell enables wheat to buffer the loss of chromosomes (Li *et al*., 2015).

Mujeeb-Kazi *et al*. (2013) classified wheat-related species into distinct gene pools based on their ability to cross with hexaploid wheat. Compared to other crops, the genetics of wheat is complex because while some species are diploid, a majority are stable polyploids with 4 (tetraploid) or 6 (hexaploid) sets of chromosomes (Zhu *et al*., 2019; Zohary *et al*., 2012). Einkorn wheat has two sets of chromosomes (diploid, 2*n*=14) (Belderok *et al*., 2000). On the other hand, tetraploid species (emmer and durum) originate from wild emmer (*T. turgidum* subsp. *dicoccoides*, 2*n*=4*x*=28, BBAA), which resulted from hybridization of two diploid wild grasses, *T. urartu* and *A. searsii* (wild goat grass) (Avni *et al*., 2017; Ling *et al*., 2018; Maccaferri *et al*., 2019; Zhu *et al*., 2019). The unknown grass is yet to be identified among present-day wild grasses, but her closest relative is *A. speltoides* (Glémin *et al*., 2019). The hybridization which resulted in wild emmer is thought to have taken place in the wild, through natural selection, long before domestication **(**Glémin *et al*., 2019; Voss-Fels *et al*., 2015; Zhu *et al*., 2019). Hexaploid wheat species evolved in farmers' fields, where hybridization between either domesticated emmer or durum wheat with diploid *A. tauschii* (wild grass) resulted in hexaploid wheat, spelt wheat and bread wheat (Dvorak *et al*., 2012; Luo *et al*., 2017; Maccaferri *et al*., 2019; Zhu *et al*., 2019). Although bread wheat is an allohexaploid, it often behaves like a diploid at meoisis with normal disomic inheritance due to *Ph1* gene (Glémin *et al*., 2019; Pont & Salse, 2017; Sidhu *et al*., 2008).

2.4 Phenology of wheat

The growth cycle of wheat is partitioned into developmental stages which vary with genotype, temperature, photoperiod sensitivity and sowing date (Laitinen & Nikoloski, 2019; Ullah *et al*., 2019). The exposure to low temperatures (vernalization) accelerates flowering while photoperiodic responses regulate the transition between vegetative and reproductive apices and physiological maturity (González *et al*., 2014; Whittal *et al*., 2018). These processes influence the adaptation of wheat and, therefore, their genetic manipulation enhances yield and adaptability (Bailey *et al*., 2019; Dube *et al*., 2019; Laitinen & Nikoloski, 2019).

Anthesis occurs 3-10 days after ear emergence at temperatures of 9.5 °C (minimum) and 18-24 °C (optimum), however, temperatures of < 9.5 °C and > 31 °C are harmful (Ullah *et al*., 2019). On the other hand, optimum and maximum temperatures for grain development are 19.3-22.1 °C and 33.4-37.4 °C, respectively (Ullah *et al*., 2019). Growth stages are standardized on Zadoks *et al*. (1974) decimal scale, where main developmental stages are delineated as 1 to 9 from seedling to ripening of kernels, respectively, while subdivisions within these stages are coded by a second digit as shown in Appendix 1. The growth stages are seedling, tillering, stem elongation, booting, ear emergence, flowering, milk, dough and ripening (Bowden *et al*., 2007; Herbek & Lee, 2009). This scale align farmers' and scientists' understanding of growth and development of wheat.

2.5 Environmental requirements for wheat

Wheat is grown in a wide range of environments and has the broadest adaptation of any cereal crop (Dube *et al*., 2019). Currently, it covers more than 15.4% of all arable land in the world (Fischer *et al*., 2014). It is a C_3 grass and, therefore, has evolved in cool and wet environments. Nonetheless, it grows in areas receiving 250-1750 mm of precipitation annually and minimum, optimum and maximum temperatures of 3-4 °C, 25 °C and 30-32 °C, respectively (Ecocrop, 2022; Ullah *et al*., 2019). Wheat flourishes in varying agro-ecological zones from the equator to within the Arctic Circle, but most suitably between latitudes 30 °N and 60 °N and 27 °S and 40 °S (Lantican *et al*., 2016). It grows in well drained and aerated soils from the sea level up to 4,570 masl (Ecocrop, 2022; Hodson & White, 2007).

In the tropics, the crop is grown at high elevations during cooler months. Wheat requires ≥ 0.5% organic matter, adequate levels of essential nutrients (especially nitrogen and phosphorus) and an optimum *pH* of 5.5-7.5 (Ghaly & Ramakrishnan, 2013). However, the

crop is sensitive to soil salinity. Cultivation of semi-dwarf, fertilizer responsive and early maturing cultivars in the last 50 years significantly improved yield and reduced losses resulting from lodging (Berry & Spink, 2012; Kamran *et al*., 2013; Kowalski *et al*., 2016). Wide adaptation in wheat is due to the complex nature of its genome which provides plasticity (Laitinen & Nikoloski, 2019; Zimin *et al*., 2017).

2.6 Production and economic importance of wheat

Wheat is one of the most important cereals in the world, being first in terms of area under cultivation followed by maize (201.9 million ha) and rice (164.2 million ha) and second to maize (1,162.4 million t) in terms of production (FAOSTAT, 2022). It is highly diverse in terms of ecological range of cultivation and is grown at any time of the year depending on the genotype (Macharia, 2015). Over time, it has become inseparable from cultures of different societies in the world (Macharia & Ngina, 2017). Wheat is the dominant grain of world commerce (33%) and its significance is projected to double by 2050 (Burkitbayeva, 2013). The annual increase in demand is highest in eastern and southern Africa (5.8%), western and central Africa (4.7%) and in southern Asia and the Pacific (4.3%) (Shiferaw *et al*., 2013). In 2019, the most important exporters of wheat were the Russian Federation (31.9 million t), the United States (27.1 million t), Canada (22.8 million t), France (20 million t) and Australia (9.6 million t) while largest importers were Indonesia (11 million t), Egypt (10.4 million t), Algeria (6.8 million t), Brazil (6.6 million t) and Japan (5.3 million t) (FAOSTAT, 2022). Wheat also accounts for the largest share of emergency food aid globally (Dixon *et al*., 2009).

The Green Revolution has led to an increase in production and yield of wheat from 222 million t and 1.2 t ha^{-1} in 1961 to about 760.9 million t and 3.5 t ha^{-1} in 2020, respectively (FAOSTAT, 2022). This was occasioned by increased adoption of high yielding varieties with high responsiveness to fertilizers, improved agronomic practices and sustainable agricultural policies (Baum *et al*., 2015). However, KNBS (2020) projects that Kenya would remain a net importer of wheat unless domestic production is significantly stepped up (Figure 2.1a). In Kenya, the area under cultivation and average yield has stagnated at between 100,000 and 120,000 ha, and $\leq$1 and 2.3 t ha^{-1}, respectively (Figure 2.1b). Practicing monoculture has introduced extreme levels of uniformity on a huge spatial-temporal scale therefore narrowing the genetic profile of cultivated wheat (Cavanagh *et al*., 2013). Together with the effects of climate change, this has aggravated the vulnerability of

the crop to biotic and abiotic stresses (Asseng *et al*., 2017; Gammans *et al*., 2017; Savary *et al*., 2019; Zabel *et al*., 2014). Nevertheless, more than 180 wheat varieties with varying levels of resistance to stem rust have been released to Kenyan farmers since 1906 (Macharia *et al*., 2016).

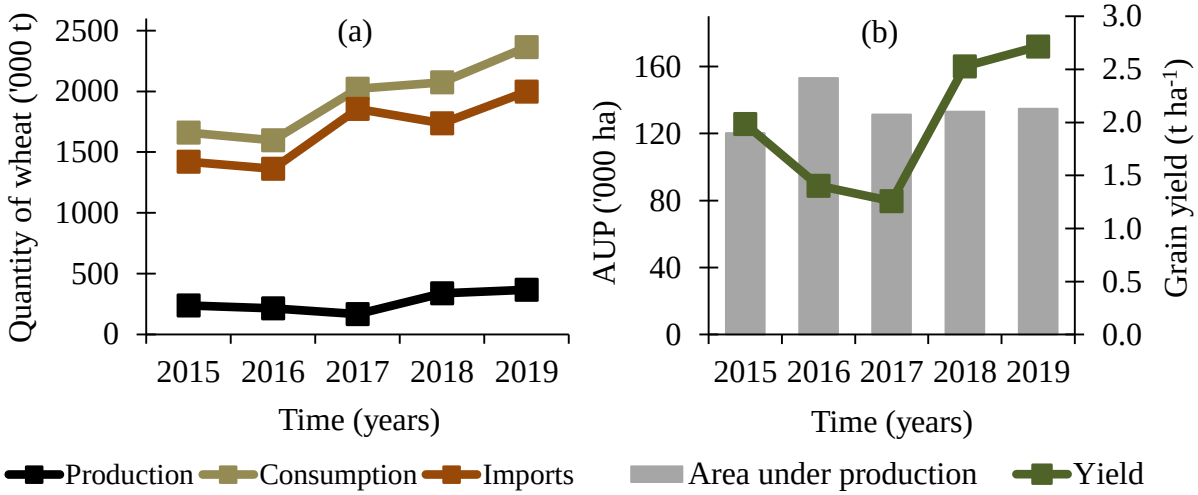


Figure 2.1 (a) Quantity of wheat produced, consumed and imported (’000 t), and (b) area under production (AUP) (’000 ha) and grain yield (t ha^{-1}) of wheat in Kenya, 2015-2019.
Source: KNBS (2020).

Globally, approximately 67% of wheat is consumed as human food, 17% as animal feeds while the rest is either used for industrial purposes or as seed (Wrigley, 2017). In developing countries, the demand for wheat is projected to increase by > 60% in 2050 against a 29% drop in production (Braun, 2011; Ray *et al*., 2013). Its significance is attributed to wide adaptability, storability, nutritional value and the numerous food products derived from it (Morris, 2004). Wheat is a major staple to 4.5 billion people in 94 developing countries and serves as the primary source of calories (21%) and vegetable proteins (20%) (Reynolds *et al*., 2019). It blends well with other foods such as millet, sorghum, cassava and sweet potatoes (Ndung’u *et al*., 2016) and is associated with reduced risks to various diseases (Aune *et al*., 2016; Reynolds *et al*., 2019; Shewry & Hey, 2015). It is consumed as whole grains, bread, *chapati*, cakes, pancakes, biscuits, *macaroni*, porridge, pastries, noodles, crackers, rolls, doughnuts and other confectioneries (Pingali, 2012). Wheat provides energy (327 kc/100g; 60-80% carbohydrate), proteins (6-21%), fat (1.5-2.0%), vitamins (B complex and E), dietary fibre, phenolic acids (ferulic and vanillic acids), carotenoids, minerals (Fe, Zn, Ca, P and Se; 1.5-2.0%) and high levels of gluten (Balk *et al*., 2019; Ndung’u *et al*., 2016; Reynolds *et al*., 2019). Dietary fibre reduces the risk of cardiovascular diseases, diabetes (type 2) and cancer

(colorectal and breast) (Reynolds *et al*., 2019). Raw wheat is ground into flour or germinated and dried to make malt (Faltermaier *et al*., 2014). Wheat gluten is used in food processing industries and wheat straw, together with the husk (bran) (>150 million t pa), is either fed to animals as hay or utilised in the production of bioethanol (Novy *et al*., 2015; Shewry, 2019).

2.7 Wheat stem rust

2.7.1 Overview of the disease

Initial details on stem rust were independently reported in 1767 by Fontana (1932) and Tozzetti (Tozzetti & Alimurgia, 1952) and its causal agent named *Puccinia graminis* in 1797 by Persoon. After the rediscovery of Mendelian papers, Biffen (1905) demonstrated that inheritance of resistance to rusts follows the laws of genetics and is governed by a recessive gene that segregates in F_2 of a monohybrid ratio of three susceptible to one resistant. Stakman and Piemeisel (1917) reported a number of physiologic races of stem rust which varied in virulence to wheat lines having different resistance genes. This discovery impacted breeding because a study of biologic forms precedes breeding for resistance.

Stem rust fungus is a ubiquitous obligate biotroph in the phylum basidiomycota, class urediniomycetes, order pucciniales, family pucciniaceae and genus puccinia (Agrios, 2005a; Duplessis *et al*., 2011; Helfer, 2014). Order pucciniales is divided into 14 families in which more than 160 genera and 7800 species have been described (Ciccarelli *et al*., 2006; Duplessis *et al*., 2011; Toome & Aime, 2015). Studies of rDNA sequence data indicate that the pathogen is composed of various variants which are morphologically identical but genetically specialized to attack different hosts (Abbasi *et al*., 2005; Ciccarelli *et al*., 2006; Cuomo *et al*., 2017). The disease is characterized by raised orange-red to black pustules on stems, leaves, leaf sheaths, spikes, glumes, awns and seeds of susceptible wheat cultivars (Marsallas & Goldberg, 2016). Conspicuously erumpent pustules have shredded epidermal tissues at the margin and may erupt through both leaf surfaces, being larger on the lower surface (Wiese, 1987). The pustules are either oval, elongated or spindle-shaped and measure up to 3×10 mm (Roelfs *et al*., 1992).

Wheat stem rust causes high yield reductions under epidemic conditions leading to famine, economic and political crises (Olivera *et al.*, 2015). A healthy crop is annihilated or reduced to black tangles of broken stems and shrivelled grains when a susceptible genotype is severely infected 2-3 weeks before physiological maturity (Singh *et al.*, 2015). Stem rust poses a high biosecurity threat because spores travel over large areas, build up rapidly and

attack economically important cereal grasses including wheat, barley (*Hordeum vulgare*), oats (*Avena sativa*), triticale (*X. Triticosecale*) and rye (Orton *et al*., 2019; Steffenson *et al*., 2017; Visser *et al*., 2019).

2.7.2 Biology of the fungus

The stem rust fungus has a macrocyclic lifecycle comprising of five distinct spore stages on the primary host with the sexual phase occurring on an alternate host (Barnes *et al*., 2020; Olivera *et al*., 2019; Zhao *et al*., 2016). Masses of single-celled dikaryotic (*n*+*n*) urediniospores are produced during the asexual phase to cause new infection cycles every two weeks. These urediniospores germinate when exposed to moisture (Garnica *et al*., 2014; Leonard & Szabo, 2005). The germ tube locates a stomatal aperture in which to establish an appresorium. This appresorium stays latent till light triggers the formation of a penetration peg (Milus *et al*., 2010). Subsequently, the sub-stomatal vesicle becomes the primary infection hyphae (Milus *et al*., 2010) and a specialised feeding structure called haustorium emerges (Garnica *et al*., 2014). Eventually, secondary infection hyphae and additional haustoria are formed. The haustoria proliferate plant cells to create an extrahaustorial matrix (Voegele & Mendgen, 2011). Other than nutrient acquisition, these structures apparently transport effector proteins into host cells to accelerate infection and to compromise host metabolism and defense responses (Garnica *et al*., 2014; Voegele & Mendgen, 2011). These phenomena occur in initial stages of infection though macroscopic symptoms of the disease appear in 8-10 days when urediniospore pustules erupt (Leonard & Szabo, 2005).

As the host plant senesce at the end of the season, the sexual cycle is initiated through formation of diploid (*2n*) teliospores. These spores are quiescent before completing meiosis to generate haploid (*n*) basidiospores which infect alternate hosts (Barnes *et al*., 2020; Jin *et al*., 2014; Zhao *et al*., 2016). This produces haploid pycniospores which fertilize receptive hyphae to restore the dikaryotic mycelia that results in aeciospores which re-infect cereal hosts to re-initiate the asexual cycle (Garnica *et al*., 2014). Therefore, the sexual cycle yields phenotypes with new virulence patterns because of segregation and re-assortment of variance at the genomic level (Olivera *et al*., 2019).

2.7.3 Life cycle

Stem rust are spores of a macrocyclic fungus of a heteroecious nature and exist in a continuum of uredinial generations while alternating between its primary, ancillary and alternate hosts (Barnes *et al*., 2020; Duplessis *et al*., 2011). Infection commences with

deposition of urediniospores on the surface of a susceptible plant in warm and moist conditions of 18-30ºC, where, in about three hours, it penetrates leaf and stem cuticles through germ tubes until it encounters a stomate (Marsallas & Goldberg, 2016).

Once a stomate is recognized by guard cells, an appressorium is formed over it and penetrates through stomatal openings using a peg which grows between guard cells, whereupon a primary infection hyphae emerges from a vesicle in the substomatal cavity. Intercellular biotrophic infection is initiated by a haustorial mother cell which penetrates the host cell wall to form an intracellular haustorium and a secondary infection hyphae for intercellular spread (Koeck *et al*., 2011). Haustoria are used in cytoplasmic nutrient acquisition and metabolism (Voegele & Mendgen, 2011) and in delivering virulence effectors which promote infection by altering host defense responses (Kemen *et al*., 2005). A single spore reproduces tens of thousands of urediniospores which either infect other plants or are transported by wind to other regions (Meyer *et al*., 2017; Visser *et al*., 2019).

In about 14 days, from infection to sporulation, most parts of the plant will be covered in pustules therefore inhibiting photosynthesis (Leonard & Szabo, 2005). As the growing season terminates and the plant starts to set seed, senesce and die, instead of producing urediniospores, black over-withering binucleate teliospores are produced to form telia on leaf sheaths and culm and may rupture epidermal cells to release spores (Marsallas & Goldberg, 2016). Unlike the more sensitive urediniospores, teliospores survive in a dormant state on wheat stubbles despite their inability to infect wheat.

Cold temperatures break dormancy of diploid teliospore cells to produce single haploid basidiospores via meiosis (Schumann & Leonard, 2000). Basidiospores are transported by wind to barberry (*Berberis vulgaris* L.) plants, where they infect, germinate and form blisters on upper leaf surfaces which produce reproductive cells that fertilize receptive cells inside compatible neighbouring spermagonia (Marsallas & Goldberg, 2016; Meyer *et al*., 2017; Visser *et al*., 2019). In this step of fertilization, two haploid nuclei fuse to create entirely new genetic characteristics and possibly new forms of virulence (Cuomo *et al*., 2017; Zhao *et al*., 2016). Once fertilized, hyphae grow downwards through barberry leaves and eventually produce reddish pustules on the underside of the leaf marked by numerous pitch or orange to black structures called aecia. When exposed to moisture, aecia forcefully eject the next spore forms of stem rust called aeciospores which vary in their genetic material due to the cycle of sexual recombination on the alternate host barberry (Barnes *et al*., 2020; Olivera *et al*., 2019; Zhao *et al*., 2016). The microscopic aeciospores once again directly

infect wheat to produce reddish brown urediniospores that are the hallmark of this disease (Agrios, 2005a).

2.7.4 Epidemiology

The pathogen-host-environment interaction of stem rust is complex and the nature of the pathogen changes depending on the genetics of the host plant (Wiese, 1987). There is variation among stem rust races in terms of virulence, aggressiveness and fitness to survive in different environments (Cuomo *et al*., 2017; Newcomb *et al*., 2016; Olivera *et al*., 2017). Generally, however, the disease thrives in a wide range of environments ranging from warm to humid conditions within temperatures of 15-30 ºC (Milus *et al*., 2010). It is favoured by heavy dews, warm temperatures and high relative humidity in the cropping season (Wiese, 1987).

Minimum, optimum and maximum temperatures for germination and sporulation of stem rust are 2, 15-24 and 30 °C, and 5, 30 and 40 °C, respectively (Chaves *et al*., 2008). Adequate humidity for 6-8 hours and 1-3 hours of low light intensity are required to initiate the infection process (Chaves *et al*., 2008). Maximum infection is attained within 8-12 hours of dew at 18 °C followed by ≥10,000 cd sr m^{-2} of light at 30 °C (Milus *et al*., 2010) and penetration pegs develop after ≥3 hours as the plant dries slowly after dew (Milus *et al*., 2010). Infection is high in crops which are planted late in the season or in lower altitudes (Singh *et al.,* 2015). A single uredinium produces up to 10,000 spores daily, 10% of which potentially germinate and it takes 14-17 days for initial infections to become severe (Agrios, 2005b).

2.7.5 Genetic diversity in stem rust of wheat

Genetic diversity is the heritable variation within or between populations of organisms (Jovovic *et al*., 2020; Olivera *et al*., 2019). Evolution of stem rust parallels the domestication of wheat (Helfer, 2014; Ravensdale *et al*., 2011) and results in genomic variability of the pathogen (Cuomo *et al*., 2017). This is attributed to evolution of predominant races or incursion of exotic ones (Cuomo *et al*., 2017; Visser *et al*., 2019). Abebe *et al*. (2013) reported genetic diversity in physiologic races of stem rust in Ethiopia based on infection types exhibited on standard differential sets. The increase in variability of virulence in stem rust pathogens makes elite germplasm vulnerable to emerging races (Abebe *et al*., 2013; Newcomb *et al*., 2016). Therefore, diversity in races informs the evaluation of

breeding materials for durable resistance to known races (Cuomo *et al*., 2017; Newcomb *et al*., 2016).

Pathogens recombine alleles for virulence on the alternate host to overcome existing resistance (Barnes *et al*., 2020; Cuomo *et al*., 2017; Zhao *et al*., 2016). Variation in virulence is also attributed to nucleus migration and somatic hybridization, where dikaryotic hyphae from different isolates exchange nuclei or chromosomes (Cuomo *et al*., 2017; Li *et al*., 2019). Moreover, mutation within Avr genes alters their DNA sequence to result in alleles which are virulent to deployed resistance genes (Park, 2016; Saunders *et al*., 2019). The use of a few varieties by farmers causes genetic drift (loss of alleles) (Leonova *et al*., 2002). When virulent races move across fields, they increase in number through gene flow. Ultimately, selection for resistance to rust, especially under monoculture, results in evolution of virulence therefore rendering the existing resistance ineffective (Bhattacharya, 2017; Lewis *et al*., 2018).

2.7.6 Host range

Different *formae speciales* (f. sp.) of the stem rust pathogen colonize more than 365 cereal and grass species in 54 genera (Abbasi *et al*., 2005; Ciccarelli *et al*., 2006; Gultyaeva *et al*., 2021; Marsallas & Goldberg, 2016). For instance, *Puccinia graminis* f. sp. *tritici*, the pathogen for stem rust of wheat, has been identified on 112 species of cultivated and wild grasses in Israel (Gerechter-Amitai & Wahl, 1966; Gultyaeva *et al*., 2021; Kislev, 1982), with wheat being its primary asexual host and barley, triticale, rye and oat among others as its ancillary hosts (Orton *et al*., 2019; Park, 2016; Steffenson *et al*., 2017). In nature, Berberis spp. (*B. vulgaris*, *B. canadensis* Mill. and *B. fendleri*), Mahonia spp. and their hybrid (*X. Mahoberberis*) are the alternate hosts on which it completes its sexual cycle (Jin *et al*., 2014; Olivera *et al*., 2019; Zhao *et al*., 2016). Rusts co-evolve with their hosts in a given environment (Helfer, 2014; Ravensdale *et al*., 2011) with alternate hosts serving as major sources of new genetic characteristics and aggressiveness through sexual recombination and reassortment of avirulence genes (Barnes *et al*., 2020; Olivera *et al*., 2019; Zhao *et al*., 2016).

2.7.7 Effects of stem rust on wheat

Nearly 50 million ha of wheat accounting for about 25% of the world's wheat area and 19% of global production (145 million t) lie in the migration path of stem rust (Evanega *et al*., 2014; Reynolds & Borlaug, 2006). The disease causes yield reductions of 50 to 100% in susceptible cultivars when conditions are conducive to disease infection and development

(Singh *et al*., 2015). Spores are spread by wind and water over extensive geographical areas (Meyer *et al*., 2017; Visser *et al*., 2019). These new isolates have the ability to attack previously resistant cultivars (Park, 2016; Soko *et al*., 2018). Crops that appear healthy 2-3 weeks before harvest are devastated by an explosive build-up of the disease (Leonard & Szabo, 2005).

P. graminis f. sp. *tritici* impairs conduction of water and translocation of nutrients especially during the grain-filling period by feeding on host cells hence reduces the quantity and quality of yield (Park, 2016; Soko *et al*., 2018). A high disease infection suppresses the plant's photosynthetic capability and increases the rate of water loss (Leonard & Szabo, 2005; Soko *et al*., 2018). It significantly reduces the size of kernels thereby compromising grain yield (Aleri *et al*., 2019; Soko *et al*., 2018). Besides, infected stems are weakened therefore predisposed to lodging and contributing to further yield losses, deteriorated forage quality and hampering of mechanization (Berry & Spink, 2012; Leonard & Szabo, 2005). Furthermore, it leads to poor germination, stunted growth and reduced flowering (Park, 2016). However, the scale of loss to stem rust depends on the crop's developmental stage, level of host plant resistance, virulence of the pathogen, prevailing environmental conditions and point of disease onset in the growing season (McIntosh, 2009; Soko *et al*., 2018; Visser *et al*., 2019).

2.7.8 Stem rust (*Puccinia graminis*) races

Stem rust races are diverse in terms of prevalence and adaptability to different climatic conditions (Nirmala *et al*., 2016). Microscopy, phenotypic and genotypic analyses are used to differentiate races (Berlin, 2012). A number of stem rust race nomenclature systems exist including the North American system by Roelfs and Martens (1988). Globally, 16 stem rust races have been isolated with a majority of new races detected in Africa (Nirmala *et al*., 2016; Olivera *et al*., 2017; Patpour *et al*., 2016). Races (and race variants) *TTKSK* (2001; *Sr31*), *TTKST* (2006; *Sr31, +Sr24*), *TTTSK* (2007; *Sr31, +Sr36*), *PTKST* (2008; *Sr31, +Sr24, -Sr21*), *PTKSK* (2009; *+Sr31, -Sr21*), *TTKTK* (2014; *Sr31, +SrTmp*), *TTKTT* (2014; *Sr31, +Sr24, +SrTmp*), *TTHSK* (2014; low infection types (ITs) on *Sr24, Sr30, Sr36 and SrTmp*), *PTKTK* (2014; low ITs on *Sr21, Sr24 and Sr36*), *TTHST* (2014), *TKTTF* (2015; Digalu race), *TTTTF* (2017; low ITs on *Sr24* and *Sr36*) and *TTKTT+Sr8155-B1* (2019; +6AS QTL) have been discovered in Kenya (Bhavani *et al*., 2019; Singh *et al*., 2015; Patpour *et al*., 2016). Races *TTKSK, TTKST, TTTSK, TTKTK, TTKTT* and *TTKTT+Sr8155-B1* belong

to the *Ug99* lineage with *TTKSK* and *TTKTT* having defeated available resistance in varieties Cacuke and Kenya Robin, respectively (Newcomb *et al*., 2016; Olivera *et al*., 2015; Pretorius *et al*., 2012).

2.7.9 Stem rust epidemics in wheat

Rust fungi are the most damaging plant pathogens and cause large-scale crop failure and famine (Hodson, 2011; Park *et al*., 2011). This is due to the narrow genetic base for resistance resulting in epidemics that cause high economic losses (Braun, 2011; Cavanagh *et al*., 2013; Soko *et al*., 2018). For instance, the breakdown of gene *Sr36* in cultivar Enkoy in Ethiopia during 1993-1994 caused a 42% loss in yield (Dubin & Brennan, 2009). Stem rust epidemics have been reported in Central India; 1946-1947 (2,000,000 t), North America; 1904 and 1954 (1,300,000 t -3,700,000 t), Australia; 1947-1948 (270,000 t), North America (1904-1962) and parts of Africa (Hodson, 2011; Pretorius *et al*., 2007).

Exotic incursions of urediniospores establish new race lineages that are adapted to the new environment (Park, 2016). Furthermore, rapid selection of rare but virulent alleles is associated with the short life cycle and large populations of the pathogen leading to epidemics (Bhattacharya, 2017; Lewis *et al*., 2018; Saunders *et al*., 2019). Therefore, elucidation of the pathogen virulence structure and diversity of races facilitate appropriate management of the disease through breeding for durable resistance (Brown, 2015). A number of strategies to breed for resistance to stem rust, yield potential and adaptability to target environments continue to be undertaken (Bailey *et al*., 2019; Macharia, 2015). This is achieved through monitoring migration of races and testing germplasm for genetic resistance and enhancing the capacity of national and international breeding programmes (Hodson, 2011; Park, 2016; Park *et al*., 2011).

2.8 Control strategies

2.8.1 Chemical control of stem rust

Fungicides control stem rust if seeds are treated before planting or when applied before economic injury levels (Wallwork & Garrard, 2020). In wheat, heterocyclic compounds like triazole ($C_2H_3N_3$) and triazole-strobilurin mixtures are applied at tillering and flowering growth stages (Amaro *et al*., 2020; Wanyera & Wanga, 2016). Strobilurin-containing fungicides such as azoxystrobin (Quadris®), pyraclostrobine (Headline®) and trifloxystrobin (Stratego®) are systemic fungicides with different modes of action (Amaro *et al*., 2020). Once inside the fungus, fungicides damage cell membranes, inactivate critical

proteins and affect the functionality of key metabolic processes within fungal cells (Price *et al*., 2015). Whereas triazoles prevent infections by inhibiting spore germination, strobilurins kill mycelium and other fruiting bodies within the leaf to 'arrest' the disease (Amaro *et al*., 2020). Triazoles are broad spectrum xylem-systemic fungicides that penetrate young leaf and stem tissues to inhibit cell membrane sterol biosynthesis leading to abnormal fungal growth (Price *et al*., 2015). Strobilurins are broad spectrum systemic fungicides which are applied before infection or in early stages of disease development and interfer with respiratory chain enzymes of the fungus to inhibit spore germination (Amaro *et al*., 2020). They confer 'the greening effect' on wheat that maintains the green leaf area longer and its effects last for 22 to 30 days even at lower rates (Amaro *et al*., 2020; Wallwork & Garrard, 2020).

To date, chemical products with one or a combination of different active ingredients have been tested, approved and released under commercial names to control wheat stem rust in Kenya (Wanyera & Wanga, 2016). However, the cost of fungicides is prohibitive for routine use by a majority of farmers who are resource-poor and there are harmful effects to human health and the environment that are associated with their use (Alemu & Mideksa, 2016; Varshney *et al*., 2012). On average, it takes two years for stem rust to resist fungicides (Jørgensen *et al*., 2018; Oliver, 2014). In addition, farmers ought to be skilled in all aspects of their use and application (Wanyera *et al*., 2009). For instance, fungicides should be applied at the right stage of the crop and in a non-wet and windy environment (Tadesse *et al*., 2010). Furthermore, the efficacy of fungicides vary with virulence patterns of the pathogen and is particularly low towards the end of the growing season (Loughman *et al*., 2005). Therefore, chemical control of stem rust is a short-term management strategy (Wanyera *et al*., 2009).

2.8.2 Cultural control of stem rust

Cultural practices of controlling stem rust enhance the existing resistance to reduce the disease pressure by delaying the onset of infection and initial severities (Roelfs *et al*., 1992). Early planting or planting early maturing cultivars, removing volunteer crops and clearing alternate host species significantly reduce pathogen variability and survivability (Barnes *et al*., 2020; Leonard & Szabo, 2005). In addition, the timing, frequency and amount of water and fertilizer applied should be regulated (Wegulo, 2012). Furthermore, early maturing cultivars should be planted downwind while late maturing cultivars are planted upwind. Other strategies involve minimizing widespread over-season survival of inoculum both on primary and accessory host species through cultivation or grazing (McIntosh, 2009).

However, cultural practices are not fully effective and losses to infection remain high because the disease continue to be disseminated via wind and water (Meyer *et al*., 2017; Visser *et al*., 2019).

2.8.3 Genetic resistance to stem rust

Plants use various mechanisms such as resistance, tolerance and avoidance to protect themselves from pathogens (Cesari, 2018). Resistance is the capacity to stop (complete) or restrict (partial) the ability of a pathogen to colonise a plant (Agrios, 2005a). Therefore, to infect and cause disease, pathogens overcome natural defenses of plant cells including pre-formed barriers such as plasma membranes and innate immune systems (Jones & Dangl, 2006). Upon detection of pathogen-associated molecular patterns (PAMPs) like chitin and flagellin (Dodds & Rathjen, 2010) delivered into host cells by biotrophic fungi (Giraldo & Valent, 2013), resistance proteins are triggered to signal downstream factors that induce defense responses (Jones & Dangl, 2006). However, pathogens evolve to evade or suppress plant defense responses by secreting virulence proteins (effectors) which target plant molecules to facilitate pathogen fitness (Dodds & Rathjen, 2010). Most of these effectors are products of resistance genes as demonstrated in flax rust pathosystems by Flor (1956).

Host plant resistance is the most effective strategy to control stem rust in wheat (Ellis *et al*., 2014; Nelson *et al*., 2018) because it is not only environment-friendly but also generates the highest return on investment in research (Evanega *et al*., 2014; Reynolds & Borlaug, 2006). In Australia, a reduction in yield losses to stem rust of $A438 million in wheat is attributed to genetic resistance compared to a paltry $A32 million and $A33 million to cultural and chemical control, respectively (Murray & Brennan, 2009). It is possible to control stem rust when it is detected while in isolated areas and the spore load is light. Therefore, on suspicion of its occurrence, disease incidence and severity is monitored and determined and new races detected because any delay facilitates further spread (Fetch *et al*., 2016).

Genetic resistance is classified as vertical (qualitative) or horizontal (quantitative) (Lowe *et al*., 2011). Vertical resistance follows the gene-for-gene hypothesis in which each avirulence (Avr) effector gene in the pathogen has a corresponding host resistance (R) protein (Flor, 1971; Petit & Fudal, 2017). The host responds to the pathogen when both gene products are compatible otherwise there is no host plant reaction. Considering that avirulent genes mutate to become virulent, therefore failling to be recognized by genes for resistance,

imply a race specific type of resistance (Boyd *et al*., 2013). The haustorium controls the interaction of rust fungus and the host (Catanzariti *et al*., 2010). Vertical resistance genes are effective at all growth stages, however, the host is resistant to particular races of a pathogen and its inheritance is qualitative (Lagudah, 2011). One or a few major genes govern elicitation of hypersensitive responses and lignification of cells when Avr gene products are recognised (Leonard & Szabo, 2005). Verticle resistance is short-lived because of rapid evolution of virulence, therefore, it is characterized by 'boom and bust' cycles (Pretorius *et al*., 2012) which highlight the need for continuous incorporation of effective genes for resistance (Evanega *et al*., 2014). Notwithstanding race specificity, verticle resistance genes are pyramided for broad and long-lasting resistance (Bhavani *et al*., 2019; Randhawa *et al*., 2018; Zhang *et al*., 2019).

In contrast, horizontal resistance is marked by a decrease in infection that results from resistance which varies quantitatively and that is buttressed by confounding effects of minor genes at multiple loci (Huerta-Espino *et al*., 2020). It provides adult (post seedling) plant resistance (APR) which is detected as field resistance (slow rusting) resulting from diverse gene combinations (Bhavani *et al*., 2019; Randhawa *et al*., 2018; van der Plank, 2012). Although APR genes are influenced by the environment, they offer durable resistance, prolong the latent period and reduce the duration of sporulation, number and size of uredinia to lower the severity of infection (Figueroa *et al*., 2020; Lowe *et al*., 2011; Priyamvada *et al*., 2011). APR genes confer partial resistance to different races and each gene contributes small to intermediate effects to the phenotype (Huerta-Espino *et al*., 2020).

2.9 Race specific and race non-specific resistance

Race specific (R) genes are essential for resistance to stem rust and conform to the "gene-for-gene" concept by conferring resistance against races which carry corresponding Avr genes (Flor, 1971). Most reported R genes encode immune receptor proteins associated with nucleotide-binding leucine-rich-repeats (NB-LRR) (Dodds & Rathjen, 2010) that recognize specific pathogen effector proteins (Avr) (Koeck *et al*., 2011) while others have receptor-like-proteins (Jones & Dangl, 2006). Flax (*Linum usitatissimum* L.) rust is the most elucidated with > 30 corresponding R and Avr genes characterized in the host and pathogen (*Melampsora lini* L.), respectively (Lawrence *et al*., 2007; Ravensdale *et al*., 2011). To date, more than 60 R genes have been catalogued in wheat and its relatives, all of which encode

NB-LRR immune receptors (McIntosh *et al*., 2017). However, R genes lead to evolution of virulence because of the strong selection pressure on the pathogen (Helfer, 2014).

On the contrary, race non-specific (APR) genes provide broad-spectrum resistance to multiple variants of the pathogen through physiological mechanisms which are independent from immune recognition (Krattinger *et al*., 2009; Moore *et al*., 2015). For instance, *Sr57* (syn. *Lr34*/*Yr18*/*Pm38*) confers resistance to stem, leaf and stripe rust as well as powdery mildew and encodes an adenosine triphosphate-binding cassette (ABC) transporter protein (Krattinger *et al*., 2009; Luo *et al*., 2021). Similarly, *Sr55* (syn. *Lr67/Yr46/Pm46*) confers resistance to the three rust diseases and powdery mildew but encodes a non-ABC (hexose) transporter (Moore *et al*., 2015). Since race non-specific genes enhance effectiveness of race specific genes, the best strategy would to combine both gene classes (Ellis *et al*., 2014). Australian and North American wheat cultivars are cushioned from stem rust through deployment of multiple resistance genes (Ellis *et al*., 2014; Park, 2016).

2.10 Wheat genetic resources

Plant breeders use genetically diverse resources to broaden the spectrum of resistance to stem rust in wheat (Mujeeb-Kazi *et al*., 2013). These resources comprise of landraces, obsolete cultivars, wild relatives, modern cultivars and elite breeding lines (Jovovic *et al*., 2020; Wingen *et al*., 2017). Approximately 850,000 wheat accessions are stored in 229 independent collections globally (Mitrofanova, 2012). Some of the most important gene banks include the Svalbard Global Seed Vault (Norway), the National Centre for Genetic Resources (US), the USDA (US), Seeds of Discovery, CIMMYT (Mexico), Leibniz Institute of Plant Genetics and Crop Plant Research (IPK) Gatersleben (Germany), the John Innes Centre (UK), N. I. Vavilov Institute of Plant Genetic Resources (VIR) (Russia) and the Australian Winter Cereals Collection (Australia) which harbor a vast array of genetic diversity for introgression into adapted cultivars (Longin & Reif, 2014). These resources are promising for future production demands because they are a reservoir of important genes for resistance to diseases, yield, agronomic performance as well as adaptability (Qian *et al*., 2017; Riaz, 2018; Vikram *et al*., 2016). For instance, the semi-dwarfing gene *Rht8* and photoperiod insensitivity gene *Ppd*_D1 are derived from landraces (Kowalski *et al*., 2016).

Wheat gene pools are classified based on evolutionary and cytogenetic relationships (Chaudhary *et al*., 2016). A number of QTLs confer resistance to stem rust through reshuffling and recombining of genes (Yu *et al*., 2014). Species in the primary gene pool

have genomes which are homologous to bread wheat (BBAADD) (Feuillet *et al*., 2008) and include hexaploid spelt (*T. spelta*, BBAADD), tetraploid durum (*T. turgidum* subsp. *durum*, BBAA), diploid einkorn (*T. monococcum*, AA), tetraploid emmer (*T. dicoccum*, BBAA), diploid goat grass (*A. tauschii*, DD) and landraces of hexaploid and tetraploid wheat (Athiyannan, 2019; Avni *et al*., 2017; Luo *et al*., 2017). These species are genetically diverse but compatible (Feuillet *et al*., 2008). Landraces are diverse, adaptated to a given environment and are resistant to a range of insect pests and diseases (Wingen *et al*., 2017). This source is preferred due to the ease of crossing with adapted cultivars and resistance is transferred via direct hybridization, homologous recombination and backcrossing (Uauy, 2017; Wędzony *et al*., 2014).

Species in the secondary gene pool only have one genome in common with bread wheat (Feuillet *et al*., 2008; Huang *et al*., 2018) and consist of both *Triticum* and *Aegilops* species. *Triticum* species include tetraploid Timopheev's wheat (*T. timopheevii*, GGAA) and tetraploid Armenian wild emmer (*T. araraticum*, GGAA) while *Aegilops* species include *A. speltoides* and *A. longissima* (Feuillet *et al*., 2008; Huang *et al*., 2018). The transfer of genes from these species is comparatively complex and often associated with hybrid seed death, female sterility of F_1 hybrids and reduced chromosome pairing (Ogbonnaya *et al*., 2013). These resources are therefore utilised through direct crossing, backcrossing, chromosome recombination, embryo rescue and genome editing technologies (Kumlehn *et al*., 2018; Rodríguez-Leal *et al*., 2017; Uauy, 2017; Wędzony *et al*., 2014).

The tertiary gene pool is composed of diploid and polyploid species whose genomes are non-homologous to bread wheat, the so called "alien genes" (Xu *et al*., 2020). These wild and cultivated relatives of bread wheat possesses valuable genes for resistance to stem rust (Crespo-Herrera *et al*., 2017; Rodríguez-Leal *et al*., 2017). Chromosomes from these species are transferred into wheat (King *et al*., 2017). For instance, many genes that are useful in resistance to pests and diseases, yield and adaptation are located on the seven chromosomes of rye (Crespo-Herrera *et al*., 2017). This demonstrates the plasticity of the wheat genome and the importance of variation in wheat breeding (Wulff & Moscou, 2014). However, interspecific transfer is often trivial with minimal chances of chromosome pairing, therefore, techniques such as distant hybridization, irradiation and tissue culture-based embryo rescue are employed (Mujeeb-Kazi *et al*., 2013; Uauy, 2017; Wędzony *et al*., 2014). Moreover, the linkage drag effect associated with large alien chromosome segments may carry deleterious traits (Gill *et al*., 2011; Voss-Fels *et al*., 2017) while its linkage block may be inherited due to

homeology (Pumphrey *et al.,* 2012). Nevertheless, the tertiary gene pool is heavily relied upon for resistance to stem rust in bread wheat (Molnár-Láng, 2015).

2.11 Genes conferring resistance to stem rust

To identify potential sources of resistance, genotypes are conventionally screened under a standard disease pressure or subjected to molecular markers in order to identify parents and transfer desirable genes (Babu *et al*., 2020; Marsalis & Goldberg, 2016). The decision to apply classical or molecular technique(s) depends on the objective(s) of the breeder and availability of necessary resources (Babu *et al*., 2020; Bakkeren & Szabo, 2020). To guarantee the efficacy of resistance genes in target environments, the Borlaug Global Rust Initiative (BGRI) conducts shuttle breeding in collaboration with CIMMYT (Leonardo *et al*., 2017; Yu *et al*., 2014).

A number of seedling (R) and adult plant resistance (APR) genes have been catalogued in wheat and its wild relatives (Wellings *et al*., 2012). To date, 65 genes and alleles have been characterized in 55 loci (McIntosh *et al*., 2017; Randhawa *et al*., 2018), three of which consist of allelic series *Sr7* (*Sr7a* and *Sr7b*), *Sr8* (*Sr8a* and *Sr8b*) and *Sr9* (*Sr9a*, *Sr9b*, *Sr9d*, *Sr9e*, *Sr9f*, *Sr9g* and *Sr9h*) (Park, 2016). At locus *Sr9*, alleles *Sr9a*, *Sr9b* and *Sr9f* are derived from *T. aestivum* while the rest are from *T. turgidum* (McIntosh *et al*., 1995). Of the remaining 51 loci, 21 are derived from *T. aestivum* while 30 are from wild relatives (‘alien’ genes) (McIntosh *et al*., 1995). Genes and alleles from fifty of the catalogued loci are R based while the rest confer APR (Szabo *et al*., 2014). Quantitative trait loci (QTLs) 1BS, 2AL, 2BS, 2DL, 5AL, 5BL, 6AL and 7BL confer APR (Yu *et al*., 2014). APR genes *Sr2* [Syn.=*Yr30/Lr27*] (3BS), *Sr55* [Syn.=*Lr67/Yr46/Pm46/Ltn3*] (4DL), *Sr57* [Syn.=*Lr34/Yr18/Pm38/Sb1/Bdv1/Fhb/ltn1*] (7DS) and *Sr58* [Syn.=*Lr46/Yr29/Ts/Pm39/Ltn2*] (1BL) are pleiotropic and are associated with morphological traits: *Sr2*; pseudo-black chaff (Kota *et al*., 2006), *Sr55*; leaf tip necrosis (ltn) (Juliana *et al*., 2015), *Sr57*; ltn (Rahmatov *et al*., 2019) and *Sr58*; ltn (Juliana *et al*., 2015). *Sr2* was discovered in a tetraploid wheat ‘Yaroslav’ and introgressed into hexaploid wheat ‘Hope’ and ‘H44-24’ by McFadden (1939). However, it confers inadequate levels of resistance when deployed in isolation (Kota *et al*., 2006). A combination of *Sr2* with unknown additive genes of similar nature creates an *Sr2* complex which offer sufficient levels of APR to stem rust (Moore *et al*., 2015). Moreover, *Sr12* and *Sr57* work in concert to confer APR (McIntosh *et al*., 2017; Randhawa *et al*., 2018).

Compared to APR genes, R genes are easier to identify and deploy in breeding programmes (Boyd *et al*., 2013). Nevertheless, their usefulness is limited due to incompatibility with the genetic background and the negative linkage drag that results from large alien chromatin segments deposited in the genome (Bhavani *et al*., 2019; Voss-Fels *et al*., 2017). Notwithstanding, *Sr9h*, *Sr13a*, *Sr13b*, *Sr15*, *Sr21*, *Sr22*, *Sr23*, *Sr24*, *Sr25*, *Sr26*, *Sr27*, *Sr28*, *Sr32*, *Sr33*, *Sr35*, *Sr36*, *Sr37*, *Sr38*, *Sr39*, *Sr40*, *Sr42*, *Sr44*, *Sr45*, *Sr46*, *Sr47*, *Sr50*, *Sr51*, *Sr52*, *Sr53*, *Sr59*, *SrHuw234*, *SrND643*, *SrCad*, *SrTA10171*, *SrTA10187*, *SrTA1662*, *SrTmp* and *Sr1RS^{Amigo}* are used alongside suitable APR genes in gene pyramiding (Bhavani *et al*., 2019; Patpour *et al*., 2016; Zhang *et al*., 2019). Near immunity to 5% level of disease is achieved when 4-5 R genes are combined in suitable genetic backgrounds (Bhavani *et al*., 2019; Luo *et al*., 2021; Randhawa *et al*., 2018).

Based on phenotypic data, at least 28 genes, namely, *Sr2* (syn. *Yr30*), *Sr13*, *Sr21*, *Sr22*, *Sr24*, *Sr25*, *Sr26*, *Sr27*, *Sr28*, *Sr32*, *Sr33*, *Sr35*, *Sr36*, *Sr37*, *Sr39*, *Sr40*, *Sr42*, *Sr44*, *Sr45*, *Sr46*, *Sr47*, *Sr51*, *Sr52*, *Sr53*, *Sr55*, *Sr56*, *Sr57* and *Sr58* are effective or partially effective to variants of *Ug99* (Newcomb *et al*., 2016). In addition, *SrArs7t*, *SrCad*, *SrND643*, *SrTA10171*, *SrTA10187*, *SrTA1662*, *SrTmp*, *SrWeb* and *Sr1RS^{Amigo}* possess varying levels of resistance to *Ug99* although their relationship with designated genes is yet to be established (Klindworth *et al*., 2012; Olson *et al*., 2013). Interestingly, although gene *Sr8155B1* is ineffective against race *TTKSK*, it confers adequate levels of resistance to recent variants in the *Ug99* race group (Nirmala *et al*., 2017). Cloning genes *Sr13a*, *Sr21*, *Sr22*, *Sr33*, *Sr35*, *Sr45*, *Sr50*, *Sr55* and *Sr57* has provided knowledge on their mechanisms of resistance and has assisted in developing their diagnostic gene-based markers (Chen *et al*., 2018; Steuernagel *et al*., 2016; Zhang *et al*., 2017). However, the emergence of more virulent races of stem rust results in the break down of deployed resistance genes (Patpour *et al*., 2016). For instance, an incursion of race *TKTTF* in 2013-14 ‘defeated’ the widely deployed gene *SrTmp* in varieties Kenya Robin and Digalu in Kenya and Ethiopia, respectively (Olivera *et al*., 2015; Patpour *et al*., 2016). Most stem rust races have identical fingerprints confirming their common ancestry (Pretorius *et al*., 2010). Nevertheless, the use of multiple APR genes or combining 4-5 R genes in suitable genetic backgrounds of APR provides adequate levels of proctection from stem rust (Ellis *et al*., 2014; Figueroa *et al*., 2020; Singh *et al*., 2015).

2.12 Wheat breeding in Kenya

In 1900s, Kenyan wheat comprised largely of introductions from Australia which was subsequently replaced by Canadian, Italian and Egyptian founder lines (Dixon, 1960; Hurd *et al*., 1969; Thorpe, 1959). Prior to 1950, Kenyan breeding populations were crosses within these lines albeit limited additions from international programmes (Evans *et al*., 1969; Macharia, 2015; Thorpe, 1959). Throughout the history of wheat breeding in Kenya, the overarching objective has been resistance to rust diseases (Dixon, 1960; Hurd *et al*., 1969; Pinto & Hurd, 1970; Wanyera & Wanga, 2016). However, during 1980-1990, substantial resources were devoted to breeding for tolerance to drought (Kinyua *et al*., 2000) and resistance to insect pests, particularly the Russian wheat aphid (RWA) (Malinga *et al*., 2007).

Currently, the Kenyan wheat breeding programme is tasked with developing high yielding cultivars with desirable end use qualities, wide adaptability and tolerance/resistance to biotic and abiotic stresses, particularly, rust diseases and the RWA (Macharia & Ngina, 2017). Together with improved management practices, these efforts have increased yield from an average of one to three t ha^{-1} during 1920s and 2010s, respectively (Macharia, 2015). Over the same period, however, the demand for wheat has risen from 0.02 to 1 million t-a 50-fold surge. Generally, breeding for yield, yield stability and resistance to insect pests and diseases have taken a priority (Bailey *et al*., 2019; Tester & Langridge, 2010; van Eeuwijk *et al*., 2016).

2.13 Genetic gain in wheat breeding

Breeding concepts aimed at enhancing genetic gain in the short term addresses the current and future growth in the demand for wheat (Araus *et al*., 2018; Tadesse *et al*., 2019a). Genetic gain is constituted by responses to selection which are dependent on inheritance of genetic variation (Falconer & Mackay, 1996). Genetic improvement through selection and recombination results in enhanced yield potential and resistance to insect pests, diseases and abiotic stresses (Bailey *et al*., 2019; Posadas *et al*., 2014; Savary *et al*., 2019). Reported gains in yield of up to 0.53% (Dube *et al*., 2019; Leonardo *et al*., 2017) are partly due to enhanced survival of floret primordial despite a constant number of potential florets $spike^{-1}$ (Bailey *et al*., 2019; Guo *et al*., 2017; Sakuma & Schnurbusch, 2020). They are also attributed to pleiotropic effects on spike fertility by gibberellic acid (GA)-insensitive dwarfing genes *Rht-B1b* and *Rht-D1b* (Alonso *et al*., 2018; Guo *et al*., 2017) and enhanced photosynthesis post anthesis (González *et al*., 2014; Taylor & Long, 2017). Since the harvest index (HI) of most

cultivars is close to maximum (0.6), further genetic gain is dependent on increase in biomass which requires improved resource use efficiency (Reynolds *et al*., 2017). The photosynthetic capacity is harnessed through the multi-ovary characteristic which enables florets to set up to four kernels instead of one (Bailey *et al*., 2019; Bustos *et al*., 2013; Guo *et al*., 2017).

Traits of interest are apportioned weights relative to their economic importance, heritability (H^2) and genetic correlations. Thereafter, selection changes the genetic frequencies of alleles at segregating loci which are responsible for either an increase or a decrease in variation and mean phenotypic value of traits by a given margin per generation to create populations of new genotypic values (Bernardo, 2010). Estimates of H^2 and expected genetic gain predict the effect of selection (Sattar *et al*., 2003). Genetic gain (response to selection) is the difference in mean phenotypic value between offsprings of selected parents and the parental generation before selection (Heffner *et al*., 2010). Unrelated parents complement each other to maximise genetic variation (Bernardo, 2010). In addition, the choice of parents and cross depends on the proportion of genes that each parent is expected to contribute to the progeny. Therefore, the genotypic frequency of progenies depend on the parents, the number of segregating loci (genes), inheritance of the trait and the interaction of genes (governing the trait) among themselves and with the environment (Bernardo, 2010; Heffner *et al*., 2010; van Eeuwijk *et al*., 2016).

2.14 Genotypic stability

Evaluation of genotypes across environments or in different seasons introduces genotype-by-environment interactions (GEI) (Ceccarelli & Grando, 2007; van Eeuwijk *et al*., 2016). Disease pressure fluctuates with varying seasonal conditions thus affecting genotypic responses. Existence of cross over type of interaction complicates the breeder's selection due to reversal in performance of genotypes across sites (Ceccarelli & Grando, 2007; van Eeuwijk *et al*., 2016). Management of GEI thus requires selection for specific adaptability by idenfiying genotypes for given environments or broad adaptability across environments. The differences in genotypic performance across environments has led to an increased emphasis on stability of genotypes which is critical for identification of well-buffered cultivars (Lin *et al*., 1986; van Eeuwijk *et al*., 2016).

A number of stability statistics have been used in the past to identify superior genotypes and these include variance (S^2), coefficient of variability (CV %), the Wricke's ecovalence (W^2) and cultivar superiority (Francis & Kannenberg, 1978; Lin *et al*., 1986; Liu

et al., 2017; Wricke, 1962). Among these approaches, cultivar superiority has been successfully employed to identify genotypes based on their general superiority across environments (Lin & Binns, 1985; Lin & Binns, 1988). It is defined as the distance mean square between the genotypes's response and the minimum response averaged across environments. This method measures superiority based on one parameter thus simplifying the screening process. Furthermore, the difference between the mean of the best genotype and the mean of each genotype averaged across environments achieves optimum productivity for the entire region. The specific adaptability of a genotype is identified by plotting minimum and test genotype responses on location means (Lin & Binns, 1985; Lin & Binns, 1988).

CHAPTER THREE
ADULT PLANT RESISTANCE TO STEM RUST AND AGRONOMIC PERFORMANCE OF BREAD WHEAT GENOTYPES IN KENYA

Abstract

Wheat (*Triticum aestivum* L.) production in Kenya is below its potential due to stem rust (*Puccinia graminis* f. sp. *tritici*) disease. However, adult plant resistance (APR) genes from diverse sources are effective in managing the disease. A field study was therefore conducted to determine APR to stem rust among introduced Australian bread wheat genotypes alongside grain yield (GY) and agronomic performance. Sixty-four genotypes including two controls, Kenya Robin and Cacuke, were evaluated over two seasons in a partially balanced *lattice*-square design with three replicates. Genotypes Sunguard, Lancer and Gauntlet were resistant (R) to moderately resistant (MR) to stem rust. Mean GY, 1000-kernel weight (TKW) and test weight (TW) ranged from 0.26-3.37 t ha^{-1}, 8.9-28.3 g and 41.4-74.5 kg hL^{-1}, respectively. Effects due to genotype, season and genotype-by-season interaction were significant ($p \leq 0.05$) for area under disease progress curve (AUDPC), coefficient of infection (CI), final disease severity (FDS), GY, TKW and TW. Regression analyses revealed a significant reduction in GY, TKW and TW with an increase in FDS. Significant ($p < 0.01$) positive correlations were revealed in AUDPC, CI and FDS. AUDPC, CI and FDS were negatively correlated with GY, TKW, TW and harvest index (HI). Heritability (H^2) for AUDPC, GY and TKW was 73.3%, 44.3% and 61.8%, respectively. Genotypes Sunguard, Lancer and Gauntlet were identified as stable in resistance to stem rust and high yielding. These genotypes are recommended as sources of genes for introgression into adapted Kenyan cultivars and candidates for future deployment as stem rust resistant varieties.

3.1 Introduction

Wheat is a major crop globally as a source of food, nutrition and livelihood (Balk *et al*., 2019; Shiferaw *et al*., 2013). In sub-Saharan Africa (SSA), its demand has been rising steadily at ~ 4.2% per annum due to growth in population, urbanization and household income (Mason *et al*., 2022; Shiferaw *et al*., 2013; Tadesse *et al*., 2019b). However, wheat production in SSA only meets ~28% of regional requirements (USDA-FAS, 2022). In 2020, for instance, of the 760.9 million tonnes (t) produced worldwide, SSA contributed a paltry 9.3 million t yet consumption was nearly 33.8 million t (USDA-FAS, 2022). To offset this deficit, a 30% growth in yield ought to be executed through annual increases of at least 2%

(Ray *et al*., 2013; Valluru *et al*., 2014). However, current levels of genetic gain for grain yield (GY) are approaching a plateau and, therefore, are insufficient to meet the rising demand (Araus *et al*., 2018; Reynolds *et al*., 2017; Tadesse *et al*., 2019a).

Wheat production is adversely affected by both biotic and abiotic factors that reduce the quantity and quality of yield (Leonard & Szabo, 2005; Park, 2016; Savary *et al*., 2019; Soko *et al*., 2018). Among major biotic stresses of wheat are the three foliar diseases of economic significance namely stem (syn. black) rust (*Puccinia graminis* Pers. f. sp. *tritici* Eriks. and E. Henn.), stripe (syn. yellow) rust (*Puccinia striiformis* Westend. f. sp. *tritici* Eriks.) and leaf (syn. brown) rust (*Puccinia triticina* Eriks. f. sp. *tritici*) (Chen, 2020; Lewis *et al*., 2018; Saunders *et al*., 2019). Of these, stem rust is the most devastating because it hinders sustainable production of wheat and other cereal grains (Dean *et al*., 2012; Szabo *et al*., 2014). Stem rust reduces the number of kernels spike^{-1} and causes shrivelling of kernels (Brinton & Uauy, 2019; Soko *et al*., 2018). Currently, > 90% of cultivars grown globally are susceptible to this disease (Braun, 2011) and its aggressiveness is attributed to its specificity and the ability to evolve rapidly thereby generating many variants with different virulences (Cuomo *et al*., 2017; Olivera *et al*., 2019; Terefe *et al*., 2016). This has compelled the international wheat research community to continuously improve the genetics of wheat against emerging stem rust races (Park *et al*., 2011). The existing genetic variation is relied upon for crop improvement (Jovovic *et al*., 2020; McDonald, 2014; Mujeeb-Kazi *et al*., 2013). It is useful in breeding for adaptability to different wheat production environments and accumulation of grain yield (Glazmann *et al*., 2010; Valluru *et al*., 2014).

Several studies identified quantitative trait loci (QTLs) in diverse germplasm for resistance to stem rust (Rahmatov *et al*., 2019; Randhawa *et al*., 2018; Wessels *et al*., 2019). QTLs for resistance to stem rust and GY-related traits are discovered via marker trait associations using mapping populations derived from bi-parental crosses and diversity panels using genome-wide association studies (Lopes *et al*., 2015; Mengistu *et al*., 2012). These QTLs are introgressed into adapted genetic backgrounds through artificial hybridization from which wheat is bred for adaptation to target environments which has increased production from one to three t ha^{-1} (Fedoroff, 2015). To date, more than 65 stem rust (Sr), 70 yellow rust (Yr) and 79 leaf rust (Lr) resistance genes have been catalogued (McIntosh *et al*., 2017). However, most of them are race specific and are often overcome by new races with corresponding virulence (Singh *et al*., 2015). Singly deployed race specific genes are broken down when new forms of virulence emerge (Pretorius *et al*., 2012). The deployment of race

non-specific genes is however considered more effective in managing these rusts. Durable resistance is attained when race specific genes are combined with race non-specific genes (Ellis *et al*., 2014; Figueroa *et al*., 2020). Nonetheless, the challenge is to identify optimum genes and their combinations for the least possibility of break down by new virulent races (Ellis *et al*., 2014).

Despite the efforts made in identifying genes for resistance to stem rust and developing resistant genotypes, the resurgence of new races remains a challenge (Lewis *et al*., 2018; Saunders *et al*., 2019). For instance, stem rust isolates that were collected during an epidemic in Germany revealed new races (Olivera *et al*., 2017). Flath *et al*. (2018) reported 43% of the previously resistant genotypes becoming susceptible. In Kenya, the wheat breeding programme based at the Kenya Agricultural and Livestock Research Organization (KALRO) in Njoro in collaboration with the International Maize and Wheat Improvement Centre (CIMMYT) deploys resistant wheat varieties to farmers (Bhavani *et al*., 2019; Macharia & Ngina, 2017; Njau *et al*., 2013). However, deployed resistance often become vulnerable to races which continuously emerge (Lewis *et al*., 2018; Saunders *et al*., 2019). The evolution of virulence, therefore, underscores the need for continuous research for new sources of resistance. Consequently, identification of novel sources of resistance is a sustainable strategy that potentially confers durable resistance through strategic introgression of resistance genes into adapted cultivars. The objective of this study was therefore to identify stable stem rust resistant genotypes with acceptable GY and desirable agronomic traits from introductions.

3.2 Materials and methods

3.2.1 Experimental site description

The experiment was conducted at the International Stem Rust Phenotyping Platform established at the Kenya Agricultural and Livestock Research Organization (KALRO), Food Crops Research Institute (FCRI), Njoro (35° 55′ 60″ E, 0° 19′ 60″ S) over two seasons. The research centre is situated in Nakuru County in the Central Rift Valley highlands of Kenya and is elevated at approximately 2185 masl and lies within the Lower Highland III (LH_3) Agro-Ecological Zone (AEZ) (Jaetzold *et al*., 2010). The soils are predominantly well drained volcanic *mollic andosols* which are dark brown to greyish with a thick humic top soil and an average *pH* of 7.0 (Jaetzold & Schmidt, 1983). The research centre receives approximately 980 mm of precipitation annually with average minimum and maximum

temperatures of 9.7 and 25 °C, respectively. These climatic conditions are suitable for cultivation of wheat in the off-season (January to May) and main-season (June to October) and favour the occurrence of stem rust.

3.2.2 Genotypes

Sixty-two Australian bread wheat introductions alongside two controls, Cacuke and Kenya Robin, were used in this study. The introductions are crosses of CIMMYT genotypes derived from diverse parents following different selection histories, and assembled based on their responsiveness to stem rust and agronomic performance in different environments. Genotype Cacuke (Canadian/Cunningham/Kennedy) is highly susceptible to several races of stem rust while genotype Kenya Robin (Babax/*Lr42*//Babax*2/3/Tukuru; *Sr2* and *SrTmp*) is high yielding but susceptible to races *TTKTK* and *TTKTT*. The pedigree information for the sixty-two Australian bread wheat genotypes is shown in Appendix 2.

3.2.3 Experimental procedure

A portion of land previously not under any crop of the grass family in the past two seasons was cleared in preparation for cultivation. Soil samples were taken and analysed at the soil science laboratory for plant nutrient status, soil *pH* and soil moisture. From these analyses, appropriate interventions were undertaken including liming using calcium carbonate ($CaCO_3$). In a span of one week, the land was disc ploughed and harrowed to pulverize the soil, mix crop residues and remove weeds. A week later, it was harrowed again to break the soil clods further and provide a good tilth suitable for a seed bed. A rotavator was used to turn the soil until the seed bed was fine and levelled.

The experiment was set up in a partially balanced *lattice* square design (Gomez & Gomez, 1984) where genotypes were randomly assigned to eight blocks each having eight experimental units and replicated three times. Thus, there were 64 experimental units of 70×50 cm per replicate. Blocks and replicates were separated by paths measuring 30 and 50 cm, respectively, while a 50 cm alleyway was maintained around the whole experiment. Each experimental unit had a 70 cm (length) double row furrow of 10 cm (width) by 5 cm (depth) and the two rows in the double row were 20 cm apart while the distance from one double row to the next was 30 cm. A mixture of susceptible cultivars was planted as a spreader row around the experiment 2 weeks before planting genotypes. Additional spreader rows were planted within the experiment to separate replicates and after every 2 blocks.

As furrows were made, diammonium phosphate fertilizer (DAP) (18:46:0) was concomitantly mixed with soil along the furrows at the recommended rate of 150 kg ha^{-1} to supply an equivalent amount of 27 kg N ha^{-1} and 69 kg P ha^{-1}. Five grams of seeds were sown for each entry at an equivalent seed rate of 125 kg ha^{-1} with a seeding depth of 2-3 cm and a 5 cm intra-seed spacing along the row. Furrows were then covered lightly with sufficient amount of fine soil. After sowing, a pre-emergence herbicide, Stomp® 455 CS (*pendimethalin*), was sprayed at the rate of 3.0 L ha^{-1} (150 mls/20 L knap sack sprayer) to control annual grasses and broad-leaved weeds. At 1-3 leaf stage (GS 12) (Zadoks *et al*., 1974), a selective post-emergence broad spectrum herbicide, Buctril® MC (*bromoxynil octanoate* 225 g ha^{-1} and *MCPA Ethyl Hexyl Ester* 225 g ha^{-1}), was applied at the rate of 1.5 L ha^{-1} to control broad-leaved weeds. At tillering stage (GS 20-29), urea [$CO(NH_2)_2$] (46:0:0) fertilizer was applied at the rate of 100 kg ha^{-1} to supply an equivalent amount of 46 kg N ha^{-1} for higher amounts of N and enhanced availability of NH^{4+} (Ghaly & Ramakrishnan, 2013). To control sucking and chewing insect pests, a systemic foliar insecticide, Thunder® OD 145 (*imidacloprid* 100 g/l + *beta-cyfluthrin* 45 g/l) was applied at the rate of 300 ml ha^{-1} as soon as infestations were noticed at tillering (GS 20-29) and ear-emergence (GS 50-59).

Tagging was done at the edge of every plot to indicate the replication, block, plot number and name of the genotype. For artificial inoculation, stem rust inoculum was obtained from cultivars Cacuke, Kenya Robin, Duma, Kwale, Digalu, Eagle 10, KS Mwamba, Kenya Kingbird and Kasuko in the disease nursery. Young leaves and stems with disease were cut using a pair of scissors which were sterilized in alcohol-soaked (70%) wipes after every sample and discarded safely. Samples were placed in brown envelopes and labelled to indicate the name of the cultivar, date of collecting the sample and the name of the person who collected the sample. They were then transferred to the crop pathology laboratory and chopped into small pieces and soaked in distilled water over night. The spores were washed off and the mixture filtered on a sieve. The stem rust spore suspension was prepared by adding 2 drops of a light mineral oil Soltrol® 130 Isoparaffin (Chevron Phillips Chemical, TX) to a litre of the mixture as an emulsifying agent for stable oil-in-water emulsions. The inoculum was adjusted to a concentration of 4×10^6 spores ml^{-1}. At booting stage (GS40-49), ten plants were randomly selected after every five metres on spreader rows for inoculation. These plants were needle-injected with ~1 mL of fresh stem rust inoculum in the tissues using a hypodermic syringe. Foliar inoculation was also carried out using an ultra-ionic

atomizer hand sprayer as described by Njau *et al*. (2013). Inoculation was repeated after 7 days until the disease had fully developed on spreader rows.

3.2.4 Data collection

The first stem rust scores were taken when spreader rows and controls displayed a severity of ~ 50% as per the modified Cobb scale (Peterson *et al*., 1948). Three more scores were taken at an interval of seven days. Host plant reactions (HPRs) and severity were visually evaluated. HPRs were assessed as immune (I), resistant (R), resistant to moderately resistant (RMR), moderately resistant (MR), moderately resistant to moderately susceptible (MRMS), moderately susceptible (MS), moderately susceptible to susceptible (MSS) and susceptible (S) (Appendix 3a) (Roelfs *et al*., 1992). Severity was the percentage of pustules covering stems, leaves and spikes and was estimated on a scale ranging from 1-100%, where 1% = very low severity and 100% = complete susceptibility (Appendix 3b) (Peterson *et al.*, 1948).

Area under disease progress curve (AUDPC) was calculated for multiple scores and AUDPC values of 0-150, 151-300, 301-500 and > 500 represented high, moderate, low and very low levels of resistance, respectively (Jeger & Viljanen-Rollinson, 2001). AUDPC was estimated following Wilcoxson *et al*. (1975) as;

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

……..……..……(1)

where, y_i is the % disease severity on the i^{th} scoring; t_i is the number of days from sowing to i^{th} scoring; n is the total number of scores.

Coefficient of infection (CI) was the product of final disease severity (FDS) and constants for HRs (I=0.0, R=0.1, RMR=0.2, MR=0.3, MRMS=0.5, MS=0.7, MSS=0.9 and S=1.0) (Knott, 2012). CI values of 0-20, 21-40, 41-60 and > 60 represented high, moderate, low and very low levels of resistance, respectively (Knott, 2012). FDS was the average disease severity during the final score. FDS values of ≤ 30 and > 30 represented high and low levels of resistance, respectively.

Data were also collected on GY, days to heading (DH), plant height (PH), spike length (SL), kernels per spike (K S^{-1}), biomass (BM), TKW and TW. The DH was the difference between the date of sowing and the date at which 50% of plant heads in each plot were fully extended from the flag leaf sheaths. At physiological maturity, heights of five tillers each from a randomly selected plant in a plot were measured using a metre scale from

the soil surface to the top of the spikes excluding awns and the average PH obtained. To get the SL, the length of five spikes each from a randomly selected plant in a plot was measured using a 30 cm ruler from the top of the peduncle to the top of the spike excluding awns and the average SL for each plot obtained. K S^{-1} was determined by obtaining five spikes each from a tiller of a randomly selected plant in a plot and threshing them separately. Thereafter, kernels from each spike were counted and the average number of K S^{-1} obtained for each plot.

At physiological maturity, all plants in each plot were harvested by cutting at the base using a sickle and tied together. The plants were weighed using a Mettler PC 4400 DeltaRange® digital balance to get the BM for each plot. On the other hand, GY per plot was determined by weighing kernels obtained from all plants in each plot using a digital balance after standardization of the moisture content to 12%. Plants from each plot were threshed separately using an electronic threshing machine (ALMACO® Model LPTD, S/No.T09235) and kernels separated from bran using an electronic winnower (S/No. R78443). Kernels were then weighed on a digital balance to obtain the GY per plot.

An electronic grain counter (CONTADOR®, S/No. 14176107) was used to randomly count 1000 cleaned kernels from each plot. These kernels were subsequently weighed on a digital balance to obtain the TKW per plot. TW was kernel weight per volumetric bushel and was dependent on genotype, moisture content and degree of kernel damage (USDA-FGIS, 2013). Wheat reaches physiological maturity when the moisture content is 18-20% (Herbek & Lee, 2009) and TW reduces with an increase in the moisture. TW was determined by weighing cleaned kernels from each plot in a container of a standard volume. It indicated the quality of grains for that particular plot. HI was determined as the ratio of GY to BM for each plot as;

$$\text{Harvest index (HI)} = \frac{\text{Grain yield (GY)}}{\text{Biomass (BM)}} \quad \text{.....(2)}$$

3.2.5 Data analyses

Before analyses, AUDPC was square root transformed to obtain a normal frequency distribution. Data were then subjected to a restricted maximum likelihood (REML) estimation in GenStat version 16 (Patterson & Thompson, 1971) using the linear mixed model (LMM) below, with effect due to replicates, genotypes and seasons as fixed and effect due to blocks as random.

$$y_{ijkl} = \mu + r_i + G_j + S_k + GS_{jk} + \beta_{l(i)} + \varepsilon_{m(ijkl)}$$

where, y_{ijkl} is the response, μ is the overall mean, r_i is the effect due to the i^{th} replicate, G_j is the effect due to the j^{th} genotype, S_k is the effect due to the k^{th} season, GS_{jk} is the effect due to the interaction between the j^{th} genotype and the k^{th} season, $\beta_{l(i)}$ is the effect due to the l^{th} block nested within the i^{th} replicate and $\varepsilon_{m(ijkl)}$ is the random error component.

Correlation analyses were carried out in GenStat to measure relationships in AUDPC, CI, FDS, grain yield (GY), harvest index (HI), days to heading (DH), test weight (TW) and 1000-kernel weight (TKW). The coefficient of determination (R^2) values from regression analyses were estimates of the variation in FDS that is explained by variation in GY, TKW and TW whereas the slope (b-values) indicated the magnitude of the change in GY, TKW and TW that is occasioned by a unit change in FDS. Genetic correlation estimates were determined by coefficient of variation (CV %) and mean across seasons. Variance component estimates for genotype (σ_g^2), genotype-by-season interaction (GSI) (σ_{gs}^2) and residual (σ_e^2) were obtained by fitting the LMM using REML in GenStat with effect due to replicates and seasons as fixed and effect due to genotypes and blocks as random. Phenotypic coefficient of variation (PCV) and genotypic coefficient of variation (GCV) were computed according to Ogunniyan and Olokayo (2014) as;

$$\text{PCV} = \frac{\sqrt{\sigma_{ph}^2}}{\bar{x}} \times 100\% \quad \text{.....(3)}$$

$$\text{GCV} = \frac{\sqrt{\sigma_g^2}}{\bar{x}} \times 100\% \quad \text{.....(4)}$$

where, σ_{ph}^2 and σ_g^2 are variances due to phenotype and genotype, respectively, and $\bar{x}$ is the mean.

Broad-sense heritability (H^2) (%) was estimated according to equation 5. H^2 values > 60%, 30-60% and 0-30% were described as high, moderate and low, respectively (Johnson *et al*., 1955).

$$H^2 = \frac{\sigma_g^2}{\sigma_g^2 + \left(\frac{\sigma_{gs}^2}{s} + \frac{\sigma_e^2}{r}\right)} \quad \text{.....(5)}$$

where, σ_g^2 is variance due to genotype, σ_{gs}^2 is variance due to genotype-by-season interaction (GSI), s is the number of seasons, σ_e^2 is variance due to error (residual) and r is the number of replications.

Genotypic stability based on AUDPC was assessed using cultivar superiority as described by Lin and Binns (1985). The superiority of a genotype's performance was the distance mean square (MS) from the minimum response in each season and was determined as;

$$P_i = \left[n(\bar{X}_{i.} - \bar{M})^2 + \sum_{j=1}^{n}\left(X_{ij} - \bar{X}_{i.} - M_j + \bar{M}\right)^2\right]/(2n) \quad \ldots\ldots(6)$$

where, P_i is the superiority measure of the i^{th} genotype, n is the number of seasons, X_{ij} is performance of the i^{th} genotype in the j^{th} season and M_j is the minimum seasonal response.

Superiority of genotypes was based on P_i values which represented MS of the effect due to genotype $[n(\bar{X}_{i.} - \bar{M})^2]$, genotype-by-season interaction (GSI) $\left[\sum_{j=1}^{n}\left(X_{ij} - \bar{X}_{i.} - M_j + \bar{M}\right)^2\right]$ and genotype's general adaptability (Lin & Binns, 1985; Lin & Binns, 1988). Pairwise GSI MS between minimum and test genotype were used to avoid discarding genotypes with specific adaptability. Critical values for significance of P_i and GSI were the product of pooled residual MS from REML analyses and tabulated F-values for corresponding degrees of freedom (df), where the df for P_i and GSI were n and n-1, respectively (Lin & Binns, 1988). The Finlay and Wilkinson (1963) regression coefficients (b_i) on seasonal mean indicated the general response pattern among genotypes and were used to protect against discarding narrowly adapted genotypes. Genotypes with a slope of < 1, 1 and > 1 had low, average and high adaptability, respectively. b_i values < 0.7 and > 1.3 indicated adaptability to poor and better season(s), respectively (Lin & Binns, 1988).

$$b_i = \frac{\overline{Y_{i.}} - \overline{Y_{J.}}}{\overline{X_{i.}} - \overline{X_{J.}}} \quad \ldots\ldots(7)$$

where, $\overline{Y_{i.}}$ is mean across seasons, Y_{ij} is performance of the i^{th} genotype in the j^{th} season and X_j is the corresponding seasonal mean.

3.3 Results

3.3.1 Variance components

The main effects due to genotype and season were significant ($p \leq 0.001$) for all traits except the effect due to season on kernels per spike (K S^{-1}) (Appendix 4). The genotype-by-season interaction (GSI) was significant ($p \leq 0.001$) for area under disease progress curve (AUDPC), coefficient of infection (CI), final disease severity (FDS), grain yield (GY), 1000-kernel weight (TKW), test weight (TW) and spike length (SL). However, GSI was not

significant for days to heading (DH), harvest index (HI), biomass (BM), plant height (PH) and kernels per spike (K S^{-1}).

3.3.2 Genotypic performance for adult plant resistance, grain yield and agronomic traits

Area under disease progress curve (AUDPC), coefficient of infection (CI) and final disease severity (FDS) ranged from 13-1573, 0.1-100 and 2.3-100 in off-season and 0-1536, 0.2-99.1 and 0.1-99.9 in main-season, respectively (Table 3.1). The trend showed a higher prevalence of stem rust in off-season compared to main-season. The means for AUDPC, CI and FDS were 711 and 382, 50.8 and 25.9, and 58.7 and 32.6 in off-season and main-season, respectively (Table 3.1). Genotypes Sunguard, Lancer, Gauntlet, Shield, Magenta, Bolac and EGA Bounty were identified for low levels of < 300 for AUDPC, ≤ 20 for CI and ≤ 30 for FDS in both seasons (Table 3.2). AUDPC, CI and FDS for resistant genotypes ranged from 13-297, 0.1-14.6 and 2.3-26.1 in off-season and 0-155, 0.2-10.0 and 0.1-15.0 in main-season, respectively. On the basis of host plant reactions (HPRs), eight genotypes in the off-season and three genotypes in the main-season had HPRs of resistant (R) to moderately resistant (MR) with genotypes Lancer, Sunguard and Gauntlet having the lowest HPRs (Appendix 5).

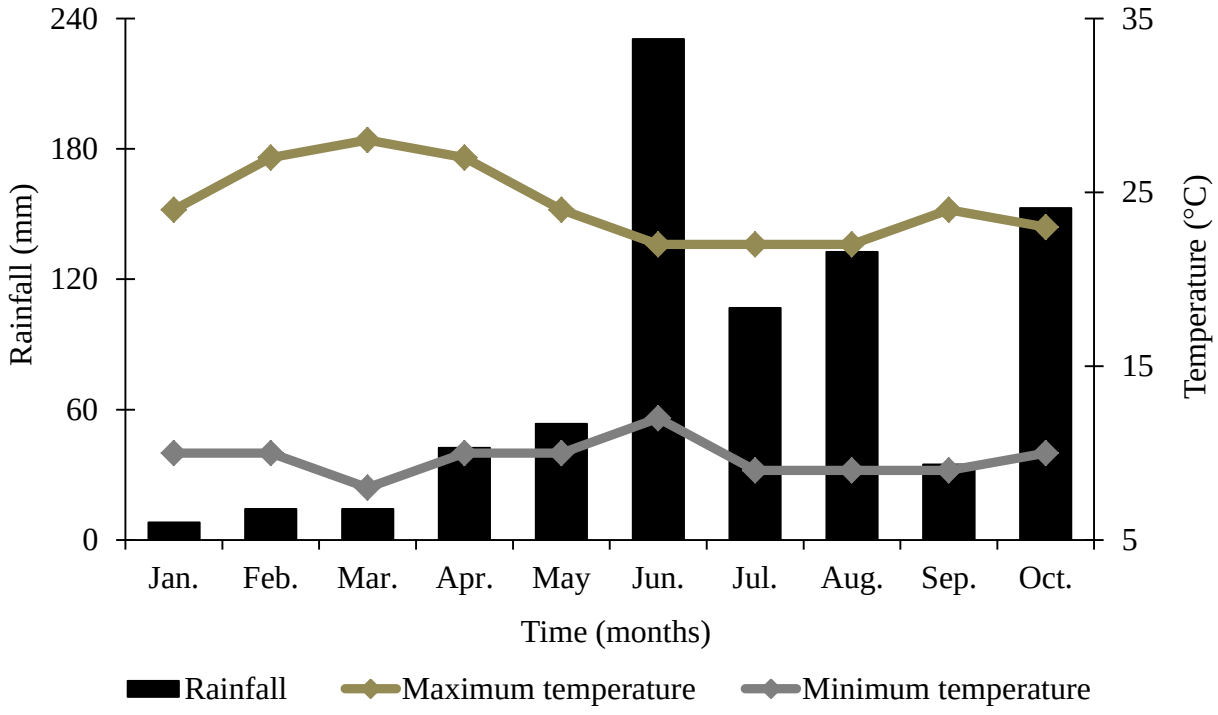


Figure 3.1 Total monthly rainfall (mm) and temperatures (˚C) during 2019 off-season (January to May) and 2019 main-season (June to October) at KALRO, Njoro.

Source: KALRO Njoro Meteorological Station No. 903502 (1), 2020.

Table 3.1 Range and mean values for disease variables, grain yield and agronomic performance of 64 bread wheat genotypes evaluated for resistance to stem rust over two cropping seasons in 2019 at KALRO, Njoro.

	Area under disease progress curve		Coefficient of infection		Final disease severity		Grain yield (t ha^{-1})	
Season	Range	Mean ± SE	Range	Mean ± SE	Range	Mean ± SE	Range	Mean ± SE
2019 off-season	13-1573	711 ± 53	0.1-100.0	50.8 ± 3.8	2.3-100.0	58.7 ± 3.3	0.14-4.93	2.01 ± 0.15
2019 main-season	0-1536	382 ± 42	0.2-99.1	25.9 ± 3.0	0.1-99.9	32.6 ± 3.2	0.30-2.44	0.91 ± 0.06
Mean[a]	3-1562	528 ± 46	0.2-98.3	38.4 ± 3.0	0.5-98.9	45.7 ± 3.2	0.26-3.37	1.46 ± 0.10

	Test weight (kg hL^{-1})		Days to heading		Plant height (cm)		1000-kernel weight (g)	
Season	Range	Mean ± SE	Range	Mean ± SE	Range	Mean ± SE	Range	Mean ± SE
2019 off-season	40.7-77.4	64.4 ± 1.1	50-82	69 ± 1	62.6-95.6	76.2 ± 0.8	10.7-32.9	20.8 ± 0.7
2019 main-season	37.6-76.1	56.5 ± 1.1	54-84	72 ± 1	50.1-91.1	73.2 ± 1.0	6.6-24.1	13.7 ± 0.5
Mean[a]	41.4-74.5	60.4 ± 1.1	51-84	71 ± 1	57.9-90.2	74.7 ± 0.8	8.9-28.3	17.2 ± 0.6

	Spike length (cm)		Harvest index		Kernels per spike		Biomass (t ha^{-1})	
Season	Range	Mean ± SE	Range	Mean ± SE	Range	Mean ± SE	Range	Mean ± SE
2019 off-season	7.0-12.3	9.3 ± 0.2	0.01-0.74	0.16 ± 0.01	23-53	38 ± 1	6.4-31.2	14.6 ± 0.6
2019 main-season	6.7-11.2	8.9 ± 0.1	0.04-0.28	0.12 ± 0.01	22-53	38 ± 1	2.8-14.1	7.8 ± 0.3
Mean[a]	7.2-11.8	9.1 ± 0.1	0.05-0.47	0.14 ± 0.01	22-53	38 ± 1	5.7-21.5	11.2 ± 0.4

[a]Mean values are a combination for 2019 off-season and 2019 main-season.

SE Standard error.

Table 3.2 Means of resistant genotypes (AUDPC ≤ 300, CI ≤ 20 and FDS ≤ 30) and controls evaluated for resistance to stem rust over two cropping seasons in 2019 at KALRO, Njoro.

	AUDPC		CI		FDS		GY		DH		HI		K S^{-1}		TW		TKW	
Genotypes	O	M	O	M	O	M	O	M	O	M	O	M	O	M	O	M	O	M
Sunguard	16	0	2.7	0.2	4.9	0.1	3.6	1.6	77	82	0.19	0.15	43	41	73.1	76.1	23.3	24.1
Lancer	13	0	1.1	1.1	2.3	0.3	3.9	2.4	76	78	0.74	0.20	38	38	77.4	70.9	25.4	20.8
Gauntlet	13	10	0.1	0.4	3.6	1.9	2.8	1.3	77	75	0.20	0.16	43	43	74.5	68.7	23.2	17.3
Shield	94	101	4.7	5.5	11.4	9.9	3.1	1.1	74	78	0.19	0.15	45	43	71.2	56.4	26.3	15.2
Magenta	194	59	7.7	3.4	19.6	6.6	4.9	1.8	68	77	0.31	0.20	42	41	76.4	69.6	31.2	19.2
Bolac	175	76	5.6	4.6	19.5	9.9	2.2	1.1	72	76	0.10	0.15	37	36	65.5	65.6	19.0	15.7
EGA Bounty	297	155	14.6	10.0	26.1	15.0	3.1	1.5	65	66	0.27	0.17	38	38	74.8	59.7	27.7	15.7
Controls																		
Cacuke[a]	1496	1201	97.0	95.3	96.9	96.5	2.4	0.7	59	60	0.22	0.13	41`	40	64.6	55.4	32.5	14.7
Kenya Robin[b]	1573	1329	97.8	96.8	97.1	96.7	1.3	0.6	69	72	0.10	0.10	48	47	56.2	45.2	20.1	10.9
Mean[c]	711	382	50.8	25.9	58.7	32.6	2.0	0.9	69	73	0.16	0.12	38	38	64.4	56.5	20.8	13.7
$LSD_{0.05}$	21.4	17.6	26.6	20.1	19.1	17.6	1.2	0.5	8.0	7.6	0.68	0.07	9.3	9.8	5.12	8.0	3.4	3.4
CV (%)	4.0	3.2	17.4	13.9	12.8	15.1	4.6	4.7	2.3	1.1	8.76	1.20	4.1	2.6	2.50	1.6	3.2	2.4

AUDPC area under disease progress curve, CI coefficient of infection, FDS final disease severity, GY grain yield (t ha^{-1}), DH days to heading, HI harvest index, K S^{-1} kernels per spike, TW test weight (kg hL^{-1}) and TKW 1000-kernel weight (g).
O 2019 off-season and M 2019 main-season.
[a]Control for stem rust.
[b]Control for grain yield and agronomic performance.
[c]Means stated are for all the 64 genotypes evaluated.

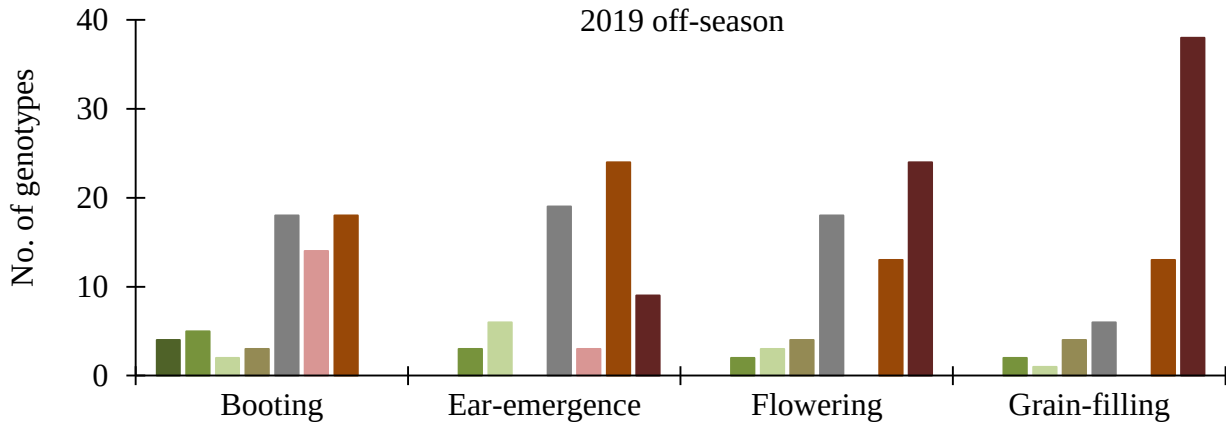
Mean grain yield (GY), 1000-kernel weight (TKW), test weight (TW), harvest index (HI) and biomass (BM) was higher in off-season than main-season (Table 3.1). The mean GY of 2.01 t ha^{-1} recorded during the off-season was higher than 0.91 t ha^{-1} which was recorded during the main-season. Despite the high disease pressure of stem rust as shown by high values of AUDPC, CI and FDS during the off-season, there was also high mean GY, TKW, TW and HI during the same period. Resistant genotypes Magenta with 4.9 and 1.8 t ha^{-1}, Lancer with 3.9 and 2.4 t ha^{-1}, Sunguard with 3.6 and 1.6 t ha^{-1}, EGA Bounty with 3.1 and 1.5 t ha^{-1}, Shield with 3.1 and 1.2 t ha^{-1}, Gauntlet with 2.8 and 1.3 t ha^{-1}, and Bolac with 2.2 and 1.1 t ha^{-1} significantly yielded higher than the best control Kenya Robin which had 1.3 t ha^{-1} and 0.9 t ha^{-1} in off-season and main-season, respectively (Table 3.2). The mean TKW and TW for these genotypes were 20.8 and 13.7 g, and 64.4 and 56.5 kg hL^{-1} in off-season and main-season, respectively (Table 3.1). However, the ranges for TKW and TW were 19.0-31.2 g and 15.2-24.1 g, and 65.5-77.4 kg hL^{-1} and 59.7-76.1 kg hL^{-1} in off-season and main-season, respectively (Table 3.2). Genotypes Magenta and Sunguard emerged with the highest TKW at 31.2 and 24.1 g while genotypes Lancer and Sunguard recorded the highest TW at 77.4 and 76.1 kg hL^{-1} in off-season and main-season, respectively.

The number of days to heading (DH), plant height (PH), spike length (SL) and kernels per spike (K S^{-1}) were not significantly affected by seasons (Table 3.1). Mean DH, PH and SL was 69, 76.2 cm and 9.3 cm in off-season and 72, 73.2 cm and 8.9 cm in main-season, respectively, while K S^{-1} ranged from 23-53 in both seasons (Table 3.1). In the resistant genotypes, DH, HI, and K S^{-1} ranged from 65-77, 0.10-0.74 and 37-45 in off-season and 66-82, 0.15-0.20 and 36-41 in main-season, respectively (Table 3.2). In terms of days to heading, genotype EGA Bounty was the earliest with 65 days in off-season and 66 days in main-season. Genotype Lancer had the highest HI of 0.74 in off-season, however, genotypes Lancer and Magenta were the best performing in main-season with HI of 0.20. On the other hand, in terms of K S^{-1}, genotype Shield emerged the best in off-season with 45 while genotypes Gauntlet and Shield was the best in main-season with 43 (Table 3.2).

Generally, shorter genotypes with PH of 57.9-74.6 cm that headed early (69 days) were more resistant to stem rust (mean AUDPC of 514) than taller genotypes with PH of 74.6-90.2 cm that headed late (73 days) which had a mean AUDPC of 605 (Appendices 5 and 6). GY, HI, TW and TKW values for off-season exceeded main-season values by 111%, 33%, 14% and 52%, respectively (Table 3.1 and Appendix 6). Kernels of susceptible genotypes were more shrivelled compared to those of resistant genotypes. For instance,

resistant genotype Sunguard had GY of 3.6 and 1.6 t ha^{-1}, HI of 0.19 and 0.15, K S^{-1} of 43 and 41, TW of 73.1 and 76.1 kg hL^{-1} and TKW of 23.3 and 24.1 g in off-season and main-season, respectively (Table 3.2). On the other hand, the susceptible control Cacuke recorded GY of 2.4 and 0.7 t ha^{-1}, HI of 0.22 and 0.13, K S^{-1} of 41 and 40, TW of 64.6 and 55.4 kg hL^{-1} and TKW of 32.5 and 14.7 g in off-season and main-season, respectively.

Genotypes varied in distribution of HRs to stem rust with growth stages (GS) in off-season and main-season (Figure 3.2). However, the trend showed a reduction in the number of genotypes which were immune (I), resistant (R) and resistant to moderately resistant (RMR) from booting (GS 40-49) to grain-filling (GS 70-79) with the highest number recorded at booting stage. On the other hand, the number of susceptible genotypes increased with GS with the highest number of genotypes which were moderately susceptible (MS) and susceptible (S) recorded at booting stage in main-season and grain-filling stage in off-season, respectively.



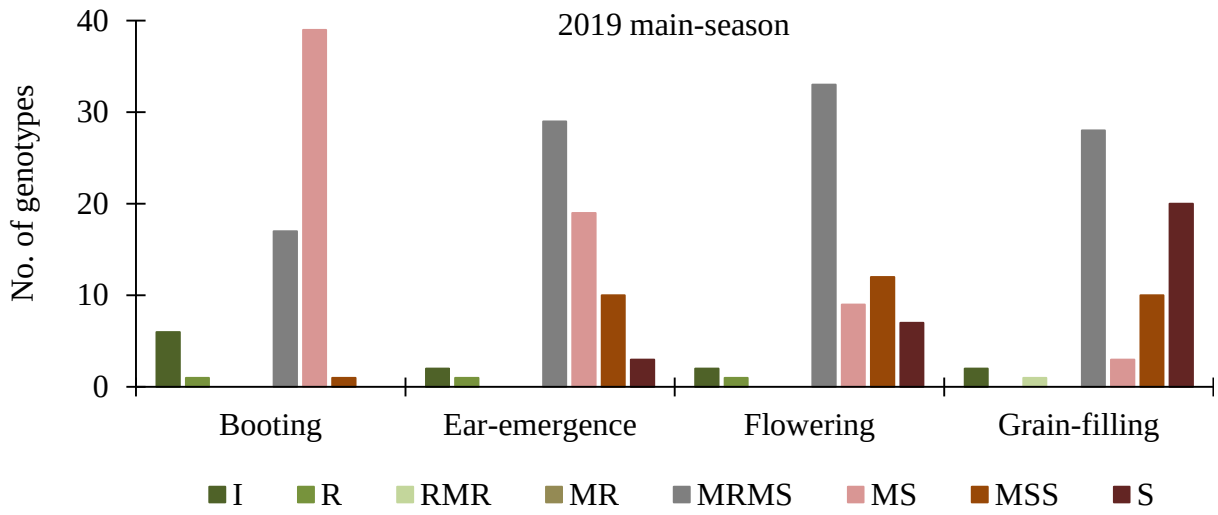


Figure 3.2 Histograms of distribution for host plant reactions of 64 bread wheat genotypes evaluated for resistance to stem rust during two cropping seasons in 2019 at KALRO, Njoro.

3.3.3 Regression and correlation analyses

Regression analyses revealed a decrease in GY (b = -0.0205), TKW (b = -0.0877) and TW (b = -0.2400) with a unit increase in FDS (Figures 3.3a, b and c). Coefficients of determination (R^2) between FDS and GY, TKW, and TW were 0.3868, 0.2150 and 0.4346, respectively, therefore, variation in FDS explained an estimated 39%, 22% and 43% of the variation in GY, TKW and TW, respectively. AUDPC, CI and FDS were highly correlated (Table 3.3). AUDPC was negatively correlated with GY (-0.6192***), HI (-0.5239***), DH (-0.0861), TW (-0.6518***) and TKW (-0.4543***). CI was negatively correlated with GY (-0.5816***), HI (-0.4702***), DH (-0.0499), TW (-0.6263***) and TKW (-0.4261***). FDS was negatively correlated with GY (-0.6219), HI (-0.5280***), DH (-0376), TW (-0.6592***) and TKW (-0.4637***). GY was positively correlated with TKW (0.8980***), TW (0.8760***) and HI (0.8241***) but was negatively correlated with DH (-0.2703*). DH were negatively correlated with HI (-0.4205***), TW (-0.3522**) and TKW (-0.5151***).

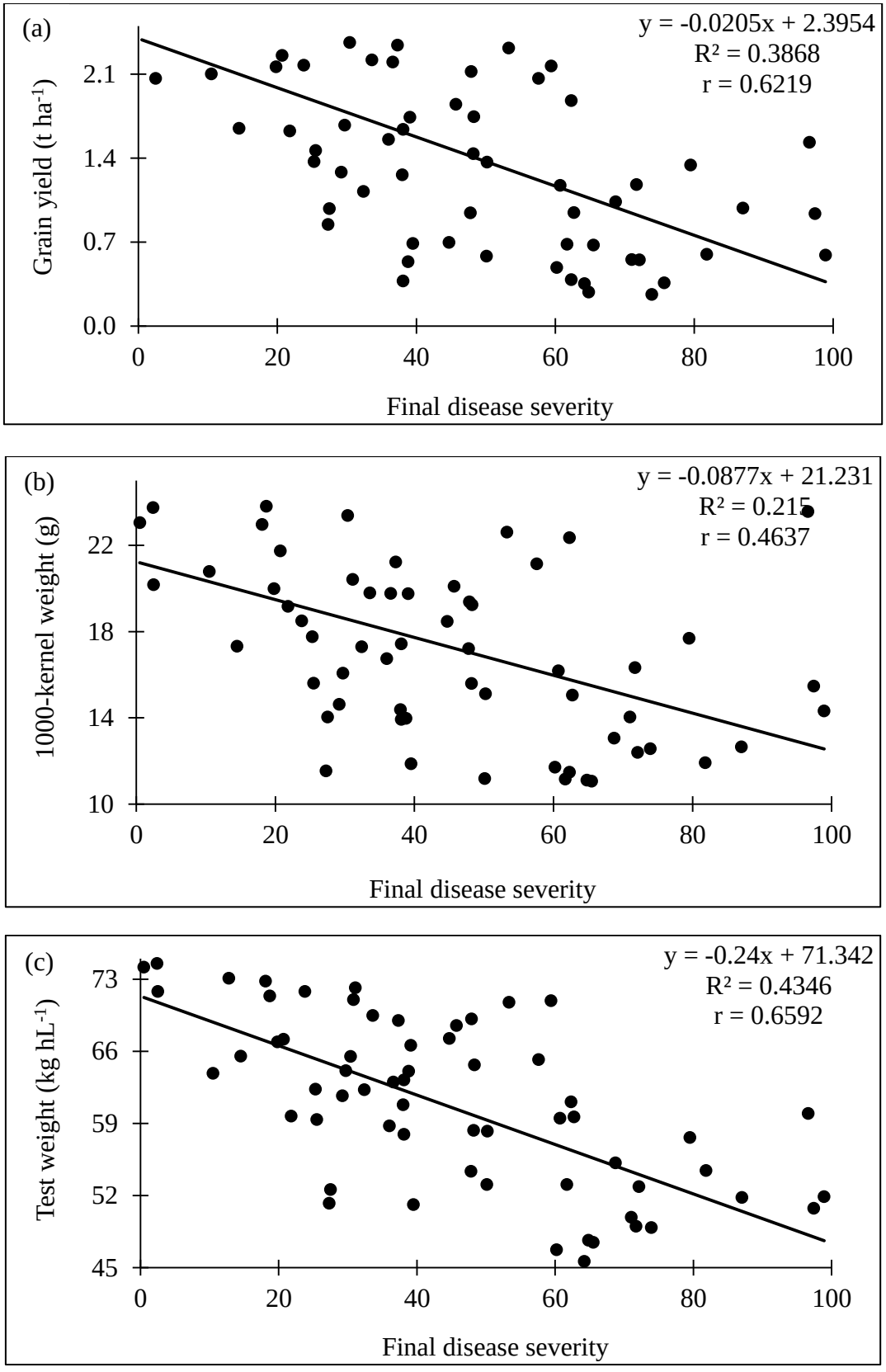


Figure 3.3 Regression for final disease severity against (a) grain yield, (b) 1000-kernel weight and (c) test weight of 64 bread wheat genotypes evaluated for resistance to stem rust over two cropping seasons in 2019 at KALRO, Njoro.

Table 3.3 Correlation coefficients for selected traits of 64 bread wheat genotypes evaluated for resistance to stem rust over two cropping seasons in 2019 at KALRO, Njoro.

	AUDPC	CI	FDS	Grain yield	Harvest index	Days to heading	Test weight	TKW
AUDPC	-							
CI	0.9481***	-						
FDS	0.9879***	0.9686***	-					
Grain yield	-0.6192***	-0.5816***	-0.6219	-				
Harvest index	-0.5239***	-0.4702***	-0.5280***	0.8241***	-			
Days to heading	-0.0861	-0.0499	-0.0376	-0.2703*	-0.4205***	-		
Test weight	-0.6518***	-0.6263***	-0.6592***	0.8760***	0.7638***	-0.3522**	-	
TKW	-0.4543***	-0.4261***	-0.4637***	0.8980***	0.7841***	-0.5151***	0.8547***	-

AUDPC area under disease progress curve, CI coefficient of infection, FDS final disease severity and TKW 1000-kernel weight.

*, ** and *** = significance at $p \leq 0.05$, $p \leq 0.01$ and $p \leq 0.001$, respectively.

3.3.4 Heritability and stability analyses

Variance due to genotype exceeded variance due to genotype-by-season interaction for all parameters measured (Table 3.4). On the other hand, variance due to genotype exceeded variance due to error for area under disease progress curve (AUDPC), coefficient of infection (CI), final disease severity (FDS), grain yield (GY), 1000-kernel weight (TKW), test weight (TW), days to heading (DH), kernels per spike (K S^{-1}) and spike length (SL) but variance due to error was more than variance due to genotype for harvest index (HI), biomass (BM) and plant height (PH). Low to high estimates for broad-sense heritability (H^2) were recorded (Table 3.4). Lowest and highest H^2 estimates of 20.6% and 73.3% were recorded for HI and AUDPC, respectively. CI, FDS, TW, TKW and GY were among the highly heritable traits with H^2 values of 70.7%, 67.3%, 69.9%, 61.8%, and 44.3%, respectively. Phenotypic coefficient of variation (PCV) and genotypic coefficients of variation (GCV) values were high (> 50%) for CI but low (< 50%) for AUDPC, DH, PH, SL, BM, K S^{-1}, TKW and TW. PCV and GCV values for FDS, GY and HI were 59.5% and 48.8%, 67.5% and 44.9%, and 84.5% and 39.1%, respectively (Table 3.4).

Based on AUDPC, genotypes Lancer, Gauntlet, Sunguard and Shield were identified as superior for performance and stability across the two cropping seasons since their P_i and MS(GSI) values were not significant (Table 3.5). However, values for genotype Shield were significantly higher compared to those of genotypes Lancer, Gauntlet and Sunguard. Genotypes Bolac, Magenta and EGA Bounty were stable but their performance was low when compared to minimum responses in each season. The trend showed that resistance to stem rust was higher in the main-season than in the off-season. Detailed results for performance and stability of all genotypes are shown in Appendix 7.

Genotype Lancer was not only the most resistant to stem rust but also the most stable in off-season and main-season with AUDPC values of 13 and 0, respectively (Tables 3.2 and 3.5) and recorded the minimum response in both seasons. This is despite genotype Sunguard being the best performing across seasons with a mean AUDPC of 3 (Table 3.5 and Appendix 5). Genotype Gauntlet was more adapted for resistance to stem rust in the off-season than genotype Sunguard with AUDPC values of 13 and 16, respectively (Table 3.2 and Figures 3.5b and c). In the main-season, genotype Sunguard was more adapted for resistance to stem rust than genotype Gauntlet with AUDPC values of 0 and 10, respectively (Table 3.2 and Figures 3.5b and c).

Table 3.4 Estimates of variation and heritability for selected parameters of 64 bread wheat genotypes evaluated over two cropping seasons in 2019 at KALRO, Njoro.

Parameter	σ^2_{ph}	σ^2_g	σ^2_{gs}	σ^2_e	H^2 (%)	PCV (%)	GCV (%)
AUDPC	86.63	63.50	4.81	18.32	73.3	1.7	1.4
Coefficient of infection	926.90	564.60	126.10	236.20	70.7	79.3	61.9
Final disease severity	738.40	497.30	81.20	159.90	67.3	59.5	48.8
Days to heading	89.98	63.59	5.65	20.74	50.6	13.4	11.2
Plant height (cm)	84.25	34.06	1.01	49.18	40.4	12.3	7.8
Spike length (cm)	1.39	0.99	0.11	0.29	71.2	13.0	10.9
Biomass (t ha^{-1})	23.99	4.93	1.26	17.80	20.6	43.8	19.8
Grain yield (t ha^{-1})	0.97	0.43	0.28	0.26	44.3	67.5	44.9
Harvest index	0.014	0.003	0.001	0.01	21.4	84.5	39.1
Kernels $spike^{-1}$	96.92	43.41	24.72	28.78	44.8	25.9	17.3
1000-kernel weight (g)	27.02	16.69	5.86	4.47	61.8	30.2	23.8
Test Weight (kg hL^{-1})	93.25	65.20	7.41	20.64	69.9	16.0	13.4

σ^2_{ph} phenotypic variance, σ^2_g genotypic variance, σ^2_{gs} variance due to genotype-by-season interaction, σ^2_e variance due to error, H^2 heritability in broad-sense, PCV phenotypic coefficient of variation, GCV genotypic coefficient of variation, and AUDPC area under disease progress curve.

Table 3.5 Superiority measure (P_i), mean squares (MS) of genotype-by-season interaction (GSI) and $\boldsymbol{b_i}$ values of the area under disease progress curve for the best performing genotypes.

Rank[a]	Genotype	Mean	P_i (10^1)	MS(GSI) (10^1)	b_i
	Minimum response	3	0.00	0.00	0.01
1	Lancer	4	0.01	0.01	1.00
2	Sunguard	3	0.02	0.02	0.81
3	Gauntlet	12	0.17	0.17	4.33
4	Shield	101	27.94	0.67	-1.86
5	Bolac	123	53.37*	12.33	0.13
6	Magenta	116	60.40*	24.81	0.10
7	EGA Bounty	222	174.47*	27.74	0.09

[a]Ranking of genotypes was based on P_i.

The comparison between genotypes Gauntlet and Sunguard reveals that although the performance of genotype Gauntlet in the off-season was as good as that of genotype Lancer, the performance of genotype Sunguard across seasons was the closest to that of genotype Lancer (Figures 3.5a, b and c). Nevertheless, the three genotypes were consistently well ranked across seasons.

3.4 Discussion

The area under disease progress curve (AUDPC), coefficient of infection (CI) and final disease severity (FDS) are reliable measures of APR (Ellis *et al*., 2014; Figueroa *et al*., 2020; Huerta-Espino *et al*., 2020). Significant effect due to genotype-by-season interaction (GSI) highlighted the effect of environment on genotypic variation for resistance to stem rust. Differences in genotypes for AUDPC, CI and FDS seemed to depend on seasonal variation. In this study, AUDPC, CI and FDS were lower in main-season than in off-season. Genotypes Sunguard, Lancer, Gauntlet, Shield, Magenta, Bolac and EGA Bounty were identified as possessing APR due to their low levels of AUDPC, CI and FDS in both seasons. In general, susceptibility increased from booting to grain-filling stage with the disease being higher in off-season than in main-season. Genotypes possessing APR characteristically displayed low host plant reactions ranging from R (resistant) to MRMS (moderately resistant to moderately susceptible) when compared to those lacking APR thus highlighting the importance of APR genes in reducing stem rust. The high level of stem rust in off-season compared to main-season was possibly due to seasonal variation in environmental conditions and disease pressure in which the off-season received less and poorly distributed rainfall with higher temperatures while the main-season received more and well distributed rainfall and lower temperatures. The minimum and maximum temperature of 10-26 °C in off-season and 8-23 °C in main-season, respectively were more favourable to infection and development of stem rust in the former than in the latter (Figure 3.1).

Agronomic performance was also related to environmental conditions and disease pressure. However, the trend showed a general reduction in GY and agronomic performance with a reduction in stem rust. For instance, GY, 1000-kernel weight (TKW) and test weight (TW) reduced from 2.01 t ha^{-1} to 0.91 t ha^{-1}, 20.8 g to 13.7 g and 64.4 kg hL^{-1} to 56.5 kg hL^{-1} with a reduction in AUDPC, CI and FDS from 711 to 382, 50.8 to 25.9 and 58.7 to 32.6 in off-season and main-season, respectively. In addition, seasonal variation significantly affected harvest index (HI), biomass (BM), plant height (PH) and spike length (SL). In a

study by Brinton and Uauy (2019) and Leonardo *et al*. (2017), variation in environmental conditions significantly influenced GY and agronomic performance of wheat. This is because yield is genetically complex and is highly influenced by the environment (Brinton & Uauy, 2019; Golan *et al*., 2015; González *et al*., 2014). In a separate study by Park (2016) and Singh *et al*. (2015), the scale of yield loss from stem rust was highly dependent on the timing of infection.

The plant canopy intercepts sunlight which is essential for photosynthesis (Kowalski *et al*., 2016; Reynolds *et al*., 2017; Taylor & Long, 2017). Photosynthetically active radiation (PAR) controls stomatal conductance which regulates the rate of photosynthesis (Asseng *et al*., 2019; Taylor & Long, 2017). Therefore, the high canopy during off-season compared to main-season was more efficient in intercepting PAR thus resulting in high yield during the former compared to the latter. These results are consistent with Asseng *et al*. (2019) who reported an increase in yield with an increase in plant canopy. Plant growth is a function of hormones whose regulation is temperature-dependent (Rahman *et al*., 2017). Therefore, high temperatures enhance the rate of photosynthesis for high yield whereas low temperatures reduce the rate of photosynthesis resulting in low yield. In previous studies by Bayeh (2010) and Kamran *et al*. (2013), earliness was found to increase yield by accelerating plant growth. In this study, early heading in off-season resulted in an increase in GY, 1000-kernel weight (TKW), test weight (TW) and kernels per spike (K S^{-1}).

Regression of FDS on GY, TKW and TW indicated a linear negative response with a reduction in GY, TKW and TW resulting from an increase in FDS. The disease impairs photosynthesis and mobilization of water and essential nutrients especially during the grain-filling period thus reducing yield (Park, 2016; Soko *et al*., 2018). These findings concur with Aleri *et al*. (2019) and Odemba (2018) who reported a significant decrease in the quantity and quality of kernels with an increase in stem rust. TW is an estimate of the quality of kernels and the amount of extractable flour (Manley *et al*., 2009; Maphosa *et al*., 2014). Further, correlation analyses revealed a significant negative relationship between disease resistance traits, AUDPC, CI and FDS and agronomic traits, GY, TKW, TW and HI. However, early maturing genotypes yielded highly despite having high levels of stem rust. The high yield in early maturing plants which were susceptible to stem rust is attributed to disease escape. Shorter plants which headed early produced more yield than taller plants which headed late. Previous studies showed that short plants which are early maturing plants produces more tillers and spikelets compared to tall plants which are late maturing (Bayeh, 2010; Kamran *et*

al., 2013; Singh *et al*., 2015). However, Kirby (2002) reported high yield in tall plants which was attributed to the competitive advantage in tall plants for sunlight. Genes for earliness and height are also responsible for photoperiodism (Alvarez *et al*., 2016; Kamran *et al*., 2013; Kowalski *et al*., 2016). Therefore, the high yield in short and early maturing plants is due to efficiency in the use of assimilates and a reduction in losses to lodging (Berry & Spink, 2012). Early maturing plants utilise more assimilates for grain-filling and undergo senenscence after physiological maturity (Distelfeld *et al*., 2014; González *et al*., 2014). Since correlation predicts the performance of one trait based on another, selecting for positively correlated traits is carried out synchronously (Lozada *et al*., 2020).

The high variance due to genotype indicate that phenotypic variance is largely attributed to genotypic variance (Lozada & Carter, 2019; Tadesse *et al*., 2019a). Since phenotypic variance is due to variance in genotype, season and GSI, seasons cause a positive or negative change in genotypic performance (Acquaah, 2012; Falconer & Mackay, 1996). Broad-sense heritability (H^2) indicate the magnitude of variation attributed to genotype (Acquaah, 2012; Khan *et al*., 2015; Toker, 2004). H^2 values showed that variance due to genotype was high on AUDPC, CI, SL, TW and TKW, moderate on DH, K S^{-1}, GY and PH, and low on HI and BM. Therefore, using phenotypic performance to select for resistance to stem rust and yield is worthwhile. Contrary to previous findings by Yadav *et al*. (2011), H^2 for GY, DH, BM and HI was moderate to low. This is because they are complex quantitative traits under a polygenic system (Brinton & Uauy, 2019; Golan *et al*., 2015; Riaz, 2018). Phenotypic coefficient of variation (PCV) and genotypic coefficient of variation (GCV) indicate variation in phenotype and genotype, respectively (Ogunniyan & Olakojo, 2014). PCV and GCV for most traits was below 50 % implying uniformity in genotypes. However, high PCV and GCV values for CI, GY and HI showed high phenotypic and genotypic variability for these traits.

Based on AUDPC, genotypes Lancer, Sunguard and Gauntlet exhibited superior performance for resistance to stem rust and were stable across the two seasons. Therefore, the three genotypes are well-buffered for resistance to stem rust since they were consistently well ranked in the two seasons. Genotype Lancer displayed broad adaptability in resistance to stem rust as indicated by the minimum response in both seasons. Therefore, genotype Lancer was suitable across seasons. On the other hand, in the off-season, the performance of genotypes Lancer and Gauntlet was comparable therefore the two genotypes had similar adaptability during this season. During main-season, however, the performance of genotype

Lancer was comparable to genotype Sunguard suggesting similar adaptability of these genotypes during this season. Genotype Lancer was therefore adapted for resistance to stem rust in both seasons while genotypes Gauntlet and Sunguard were adapted for resistance to stem rust in off-season and main-season, respectively. Genotypes specifically adapted to a given season(s) were reported by Szareski *et al*. (2017). Specific adaptability of genotypes implies deployment of such genotypes to mega-environments or environments with similar characteristics.

Appearance of more virulent races of stem rust as a result of sexual recombination and invasion of exotic spores limits the deployment of resistant genotypes (Olivera *et al*., 2019; Saunders *et al*., 2019). Selection for grain yield (GY) and agronomic performance also reduces the available genetic diversity for resistance to stem rust (Cavanagh *et al*., 2013; Jovovic *et al*., 2020; Vikram *et al*., 2016). Therefore, resistance genes are introgressed into adapted cultivars to protect them from the disease (Riaz, 2018). Abundance of adult plant resistance (APR) genes from diverse sources provide a durable and broad-spectrum resistance to a multitude of races of *P. graminis* resulting in a significant reduction in the rate of infection and development of stem rust (Huerta-Espino *et al*., 2020; Krattinger *et al*., 2009; Moore *et al*., 2015).

3.5 Conclusion

Genetic variation existed for resistance to stem rust, grain yield and agronomic performance. However, performance of genotypes was significantly affected by season and genotype-by-season interaction. Genotypes Sunguard, Lancer, Gauntlet, Shield, Bolac, Magenta and EGA Bounty were identified for adult plant resistance to stem rust. In addition, these genotypes were among the best performing in terms of grain yield and agronomic performance. Genotypes identified as resistant or moderately resistant could be used as breeding lines and deployed as a component of the integrated stem rust management programme and as parental stock in the wheat breeding programme in Kenya. Genotypes Lancer, Sunguard and Gauntlet were not only highly ranked for resistance to stem rust but also displayed stable performance with genotype Lancer displaying broad adaptability across seasons and genotypes Gauntlet and Sunguard having specific adaptability for off-season and main-season, respectively.

CHAPTER FOUR
SEEDLING RESISTANCE TO STEM RUST IN INTRODUCED BREAD WHEAT GENOTYPES IN KENYA

Abstract

Stem rust (*Puccinia graminis* f. sp. *tritici*) is a major constraint to wheat (*Triticum aestivum* L.) production in Kenya. The emergence of virulent races necessitates concurrent search for genetic resistance. A study was therefore carried out to determine seedling resistance in introduced Australian wheat genotypes to stem rust isolates *TTKSK* and *TTKTT*. Sixty four genotypes including two controls, Cacuke and Kenya Robin, were planted in two sets of plastic pots for each isolate in the greenhouse at the Kenya Agricultural and Livestock Research Organization (KALRO), Njoro. At two-leaf stage, seedlings were inoculated separately with fresh urediniospores first by brushing followed by spraying. Scoring of infection types (ITs) for stem rust was done fourteen days after inoculation. Isolate *TTKSK* was avirulent to seventeen genotypes while isolate *TTKTT* was avirulent to fourteen genotypes. Genotypes Lancer, Sunguard, Gauntlet, Scepter, Merlin, Magenta, Spitfire, Coolah, Dart, Preston and Janz were found to possess resistance (ITs ≤ 2+) to both isolates with a rating of between immune to very resistant (IT 0;) and moderately resistant (IT 2+). Genotypes Lancer and Sunguard recorded IT 0; for both isolates while genotype Gauntlet had IT 1 to isolate *TTKSK* and IT 2- to isolate *TTKTT*. Six genotypes were resistant to isolate *TTKSK* but susceptible to *TTKTT*. On the other hand, two genotypes were resistant to isolate *TTKTT* but susceptible to isolate *TTKSK*. Genotypes identified as resistant possess seedling resistance to the stem rust isolate(s) hence useful sources of resistance in breeding programmes for improvement of germplasm against stem rust.

4.1 Introduction

Wheat (*Triticum aestivum* L.) is a major source of food and nutrition to many people around the world (Shiferaw *et al*., 2013). However, despite the projected annual increases in demand of ~1.7% up to 2050 (Braun, 2011), wheat productivity is either increasing at ≤ 1.1% per annum or stagnating (McKenzie & Williams, 2015; Ray *et al*., 2013). Stem (syn. black) rust (*Puccinia graminis* Pers. f. sp. *tritici* Eriks. and E. Henn.) is a major limitation to wheat production causing between 80 and 100% yield loss in susceptible genotypes (Park, 2016; Soko *et al*., 2018). Resistance genes from diverse sources are known to protect wheat genotypes against the pathogen and help to mitigate these losses (Bansal *et al*., 2013; Mago *et*

al., 2015). However, deployed resistance genes become ineffective when more virulent races (and race variants) emerge (Bhattacharya *et al*., 2017; Lewis *et al*., 2018; Saunders *et al*., 2019). For instance, at least sixteen (16) variants of stem rust race *TTKSK* have so far been catalogued in bread wheat (Bhavani *et al*., 2019).

Seedling and adult plant resistance (APR) genes are the two major classes of genes considered when breeding for resistance to stem rust with the latter being more preferred for their durability (Ellis *et al*., 2014; Figueroa *et al*., 2020). Nonetheless, combining both gene classes reduces the possibility of new virulent races emerging to defeat conferred resistance (Ellis *et al*., 2014; Randhawa *et al*., 2018). For instance, Australian and North American cultivars are cushioned from a multitude of stem rust races by both seedling and APR genes (Ellis *et al*., 2014; Park, 2016). The accuracy of field evaluations for APR is compromised by effects of the environment, disease pressure, sequential infection, differences in plant growth and other diseases that influence expression of stem rust (Riaz & Hickey, 2017). Conversely, greenhouse tests for resistance genes are more efficient in terms of space, time and resources (Riaz, 2018; Riaz & Hickey, 2017). Besides, produced infection types (ITs) are more uniform (Prins *et al*., 2016; Riaz & Hickey, 2017).

To date, more than 60 seedling resistance genes have been discovered for resistance to stem rust in wheat with 34 being effective to at least one variant of the pathogen (McIntosh *et al*., 2017; Rahmatov *et al*., 2019; Spanic *et al*., 2015). Out of the > 60 genes, only a few are utilised in breeding because of varying levels of protection and the undesirable linkage drag (Bhavani *et al*., 2019; Voss-Fels *et al*., 2017). Seedling resistance genes are effective at all stages of the plant, are inherited qualitatively and are characterized by hypersensitive responses (Lagudah, 2011). They are typified by 'boom and bust' cycles because their effectiveness is short-lived (Pretorius *et al*., 2012). The evolution of virulence against a large proportion of deployed seedling resistance genes necessitates continuous incorporation for new sources of resistance (Evanega *et al*., 2014; Singh *et al*., 2015). However, singly deployed seedling resistance genes create a strong selection pressure on virulent mutants which usually occur at a low frequencies within the pathogen population thus rendering resistance of these genes ineffective (Burdon *et al*., 2014; Niks *et al*., 2015). Therefore, pyramiding these genes confers broad and long-lasting resistance (Randhawa *et al*., 2018; Zhang *et al*., 2019).

Previous studies revealed the existence of seedling resistance genes in breeding lines which are effective against a number of stem rust races differring in virulence (Rahmatov *et*

al., 2019; Singh *et al*., 2014). Seedling resistance is based on the gene-for-gene concept (Flor, 1971) where an IT produced by a pathogen on the host is compared to an IT produced by the same isolate on a host that carries a known seedling resistance gene (Flath *et al*., 2018; Jin *et al*., 2007; Riaz & Hickey, 2017). Therefore, depending on its interaction with a cognate avirulence (Avr) gene, the host is resistant (ITs: 1-2) but if the pathogen bypasses this recognition, the host is susceptible (ITs: 3-4) (Leonard & Szabo, 2005; Zambino *et al*., 2000). Low ITs (1-2) indicate the presence of the gene(s) conditioning resistance in the host against the tested isolate while high ITs (3-4) indicate the presence of the gene(s) conditioning susceptibility in the host against the tested isolate (Flath *et al*., 2018). The objective of this study was, therefore, to determine seedling responses to two stem rust races *TTKSK* and *TTKTT* in introduced Australian bread wheat genotypes in the absence of possible confounding effects of the environment.

4.2 Materials and methods

4.2.1 Collection of stem rust samples

Genotypes Cacuke and Kenya Robin which are susceptible to prevalent stem rust races in Kenya were planted in the greenhouse at KALRO, Njoro. Five seeds of each genotype were separately sown in plastic pots representing experimental units. The pots, measuring 6 cm (length) × 6 cm (width) × 6 cm (height), were filled with 130 cm^3 of vermiculite mixed with 3 granules of diammonium phosphate (DAP) fertilizer (18:46:0) to supply an equivalent amount of 27 kg NO_3^- ha^{-1} and 69 kg P_2O_5 ha^{-1} and seeds planted to a depth of 2 cm. Pots were labelled with the name of the genotype and date of planting and placed on raised plastic trays in the growth chamber at room temperatures and watered adequately over trays.

Seedlings were inoculated at the two-leaf stage. Inoculation was done late in the afternoon with fresh urediniospores collected from corresponding genotypes in the disease nursery following standard procedures (Figure 4.1a). Spores were suspended in 250 ml of distilled water and two drops of a light mineral oil Soltrol® 130 Isoparaffin (Chevron Phillips Chemical, TX) added and shaken gently before sieving to drain the inoculum in a dispenser (Jin *et al*., 2007). The inoculum was adjusted to a concentration of 4×10^6 spores ml^{-1}. Seedlings were inoculated first by brushing the inoculum on leaves and stems followed by spraying as fine mist from a distance of ~30 cm. Inoculated seedlings were then air-dried for 10-20 minutes and placed in polythene hoods inside a dew cabinet (Percival model I-36,

Perry, IA) for incubation at temperatures and relative humidity of 18-20 ºC and ~100%, respectively, in the dark for 48 hours (Figure 4.1b). These conditions were maintained during the day using a humidifier and misting the dew cabinet 3-4 times a day with distilled water using a hand sprayer. After the dew process, fluorescent lights were turned on to provide light to complete the infection process and temperatures raised gradually to 25 ºC for 3 hours. Thereafter, seedlings were transferred to a temperature and water-controlled growth and sporulation chamber at 18-25 ºC under natural light with additional light provided by fluorescent tubes placed at ~1 m above the seedlings and closelv monitored for symptoms of disease development. (a) (b

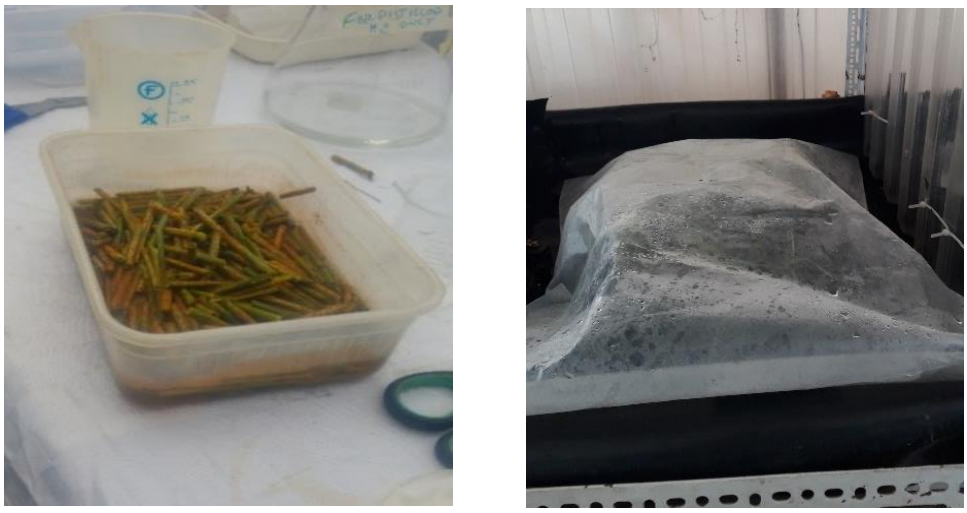


Figure 4.1 Seedling evaluation in the greenhouse at KALRO, Njoro: (a) preparation of stem rust inoculum and (b) seedlings incubated in the dew chamber.

4.2.2 Purification and bulking of isolates

Fourteen days after inoculation, one fresh and distinct stem rust pustule (large/unique) was collected from an infected stem or leaf from each pot. A sharp razor blade was used to cut out tissues around the pustule. Pustules were carefully placed in a pre-labelled gelatin capsules and sealed. Alcohol-soaked (70%) wipes were used to sterilize the razor blade between collections. The single pustules were washed off in distilled water to prepare inoculum of pure isolates. To bulk the pure isolates, five sets of the two genotypes were planted, inoculated and incubated as described in section 4.3.1 and bulk inoculum of pure isolates collected separately from each genotype following the procedure described in section 3.3.3.

4.2.3 Evaluation of genotypes

Sixty-two Australian bread wheat introductions and two controls were evaluated against stem rust isolates *TTKSK* (detected in Kenya in 2001 and virulent on *Sr31*) [purified on Cacuke] and *TTKTT* (detected in Kenya in 2014 and virulent on *SrTmp*) [purified on Kenya Robin] to characterize ITs and virulence patterns. Two sets of each genotype were planted in the greenhouse as described in section 4.3.1 for each isolate. At the two-leaf stage, each set was inoculated and incubated separately and monitored for symptoms of disease development. Tests were repeated to clarify ambiguous results.

4.3 Data collection

ITs were scored according to Stakman *et al*. (1962) as 0 (immune), ; (very resistant), 1 (resistant), 2 (moderately resistant), X (mesothetic or heterogenous), 3 (moderately susceptible) and 4 (susceptible) (Table 4.1 and Figure 4.2). All ITs observed on stems and leaves were recorded in the order of their prevalence with the most frequent IT recorded first. A comma (,) was used to segregate more than one IT. A forward slash (/) differentiated symptoms on the first and second stem or leaf with letters "n" and "c" indicating more than usual necrosis and chlorosis, respectively. In addition, plus (+) and minus (-) signs described pustules which were relatively larger or smaller, respectively, than is normal. IT 0; was between immune and very resistant. IT 1 was differentiated further into 1-, 1, 1+ while IT 2 was differentiated further into 2-, 2 and 2+ as shown in Figure 4.2.

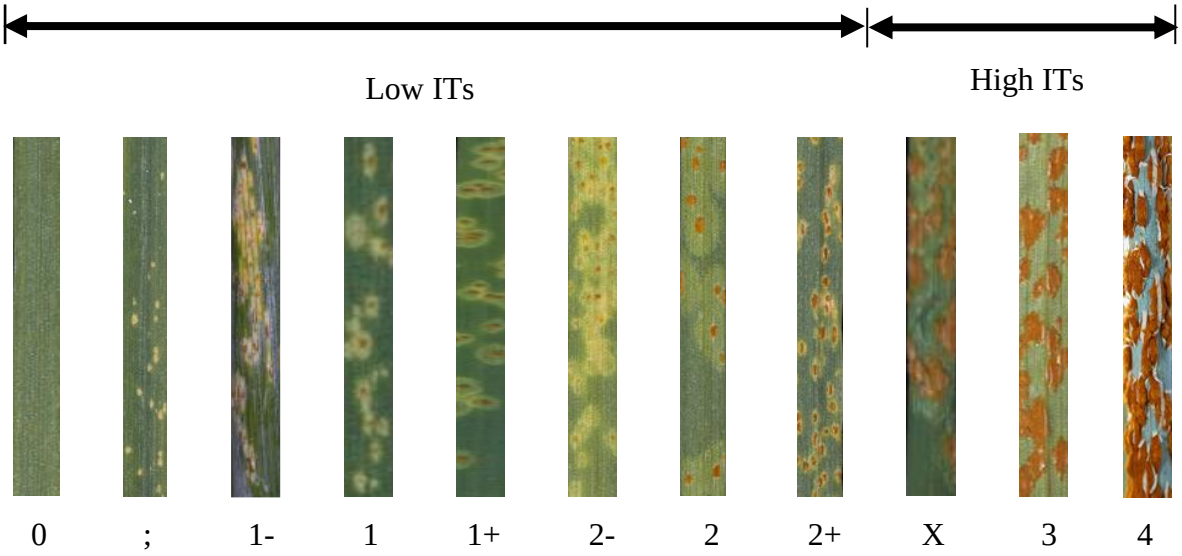


Figure 4.2 Infection types fourteen days after inoculation with *Puccinia graminis* f. sp. *tritici*.

Source: Stakman *et al*. (1962).

Data were visually taken with the assumption that ITs in the greenhouse and field are highly correlated. ITs 0, ;, 1-, 1, 1+, 2-, 2 and 2+ were rated low (incompatible) therefore the tested isolate was avirulent (Avr) to the resistant (R) host while ITs X, 3 and 4 were rated high (compatible) therefore the tested isolate was virulent (Vr) to the susceptible (S) host.

Table 4.1 Seedling infection types and description of symptoms.

Host plant reaction	Infection type	Description of symptoms
Immune	0	No sign of infection to the naked eye but minute flecks may be visible under low magnification.
Very resistant	;	No uredinia but distinct flecks of varying sizes. Usually a chlorotic yellow but occasionally necrotic.
Resistant	1	Small uredinia surrounded by yellow chlorotic or necrotic areas.
Moderately resistant	2	Small to medium-sized uredinia typically in a dark green island surrounded by a chlorotic area.
Mesothetic/ heterogenous	X	A range of infection types from resistant to susceptible scattered randomly on a single leaf, caused by a single isolate but not a mixture.
Moderately susceptible	3	Medium-sized uredinia with infrequent coalescence and development of disease is somewhat sub-normal. True hypersensitiveness is absent, however, chlorotic areas may be present.
Susceptible	4	There are large, numerous and confluent uredinia, however, hypersensitiveness is entirely absent.

Source: Stackman *et al*. (1962).

4.4 Results

4.4.1 Responses of genotypes to isolates *TTKSK* and *TTKTT*

Genotypes produced different infection types (ITs) in the greenhouse for isolates with all ITs observed except X and 4 (Table 4.2). Genotype Cacuke scored 3+ for both isolates while genotype Kenya Robin scored 3+ for isolate *TTKTT* and 2+ for isolate *TTKSK*. Seventeen (17) genotypes (28.3%) were resistant (ITs ≤ 2+) to isolate *TTKSK* while forty-three (43) genotypes were susceptible (ITs > 2+). Two genotypes, Wyalkatchem and Yitpi, did not germinate. On the other hand, fourteen (14) genotypes (22.6%) were resistant to

isolate *TTKTT* while forty-eight (48) genotypes were susceptible. Therefore, isolate *TTKTT* was 5.7% more virulent compared to isolate *TTKSK*. Genotypes Lancer, Sunguard, Gauntlet, Scepter, Merlin, Magenta, Spitfire, Coolah, Dart, Janz and Preston exhibited resistance to both isolates.

Table 4.2 Infection types of 64 bread wheat genotypes evaluated in the greenhouse against stem rust isolates *TTKSK* and *TTKTT* at KALRO, Njoro.

	TTKSK		*TTKTT*			*TTKSK*		*TTKTT*	
Genotype	Set 1	Set 2	Set 1	Set 2	Genotype	Set 1	Set 2	Set 1	Set 2
Cacuke	3+	2+ , 3	3+	3 , 2+ / 3+	Espada	2- / 3	; , 2-	3+	3+
Kenya Robin	2	2 , 2+	3+	3+	Estoc	3	3+ , 3	3+	3 , 3+
Coolah	; , 2-	;	1 , 2-	0;	Forrest	3+ , 3	3+ , 3	3+	3+
DS Faraday	2 , 2+	3 , 2	3+	2	Gauntlet	; , 1	; , 1	; , 1 , 1+	; , 1 , 2-
Chara	3+	3+ , 3	3+	3+ / 2-	Gazelle	2-	2-	3+	3+
LRPB Flanker	2 , 2+	3 , 2+	2+ , 3	2+ , 3	Janz	2-	; , 2-	2 , 2-	2 , 2-
LRPB Reliant	2+ , 3-	3+	3	3	Kiora	2+ , 3	NG	3 , 3-	3 , 2
Ninja	3+	3+	3+	3+ , 3	Lancer	0;	0;	0;	0;
Sunmax	3+	2- , 3+	3+	3+ , 3	Livingston	3+ , 3	3+ , 3	3	3+ , 3
Tenfour	3+	3+ , 3	NG	3+ , 3	Mace	3 , 2+	2+ , 3	2+ , 3	3+
Tungsten	3+	3 , 2+	3+	3+	Magenta	2-	1 , 2-	2-	2
Axe	2 / 3+	2	2	3+ / 2-	Merlin	2 , 2+	2	2	2
B53	2+ , 3-	NG	3+	2+ , 3	Mitch	3+	3 , 3+	3+ , 3	3+
Beckom	3+	3+	3+	3+	Orion	2-	2-	2+ , 3	2+
Bremer	2- / 3+	2-	3+	2+	Gladius	3+	3+	3+	3+
Buchanan	3+ , 3	3+ , 3	3+	3	Preston	2+	2	NG	2 , 2+

Calingiri	3+	3 , 2+	3+	3+	Scepter	2-	NG	2 , 2-	2-

Table 4.2 Continued

	TTKSK		*TTKTT*			*TTKSK*		*TTKTT*	
Genotype	Set 1	Set 2	Set 1	Set 2	Genotype	Set 1	Set 2	Set 1	Set 2
Cobalt	3+	3 , 3+	3+	3+	Scout	3+	3+	3+	3+
Cobra	3+	3+	3+	3+ , 3	Shield	0; , 1	0;	3 , 2+	2 , 2+
Condo	3+ , 3	3+	3+	3 , 3+	Spitfire	2	2 , 2-	2	2
Corack	2+ , 3	2+ , 3	3 , 2+	3	Steel	3+	3+ , 3	3+	3+
Correll	2 , 3	3 , 2+	3 , 2+	3 , 3+	Sunguard	0;	NG	0;	0;
Cosmick	3+	3+ , 3	3 , 3+	3 , 3+ / 3, 2+	Bolac	3- , 2	2 , 2-	2-	2-
Cutlass	; , 2	; , 2-	3 , 3+	3 , 2+	Suntop	3+ , 3	3+	3+	3+
Dart	2 , 2-	2 , 2+	2-	2	Supreme	2-	2-	3 , 2+	3 , 2+
Derrimut	3 , 3+	3+ , 3	3 , 3+	3+ , 3	Trojan	3	3+ , 3	3+ , 3	3+
DS Darwin	3 , 3-	3	3 , 3-	3	Viking	3+ , 3	3+	3	3 , 3+
DS Pascal	3+	NG	3+	3+	Wallup	NG	3+ , 3	3+ , 3 / 2	3
EGA Bounty	3+ , 3	3+	3 , 3+	3+ , 3c	Westonia	2-	1 , 2-	2+ , 3-	2
EGA Gregory	2+ , 3	3 , 2+	3 , 3-	3 , 3-	Wyalkatchem	NG	NG	3+	3+ , 3
Baxter	3+ / ;2	; / 1	3+ / 2-	2- / 3+ , 3	Yitpi	NG	NG	; , 1	; , 1
Emu Rock	2 , 2- / 3 , 3+	2 , 2+	2 , 2-	2	Zen	NG	3+ , 3	3+ , 3	3+ , 3

NG Did not germinate.

Genotypes Lancer and Sunguard were immune to very resistant (IT 0;) to isolates *TTKSK* and *TTKTT* while genotype Gauntlet was resistant (IT 1) to isolate *TTKSK* and moderately resistant (IT 2-) to isolate *TTKTT*. Genotypes Shield, Westonia, Gazelle, Orion, Supreme and Cutlass were resistant to isolate *TTKSK* with ITs 1, 2-, 2-, 2-, 2- and 2 but susceptible to isolate *TTKTT* with ITs 3, 3-, 3+, 3, 3 and 3+, respectively (Table 4.2). Genotypes Bolac and Emu Rock were resistant to isolate *TTKTT* with ITs 2- and 2 but susceptible to isolate *TTKSK* with ITs 3- and 3+, respectively. Genotype Yitpi was resistant to isolate *TTKTT* with IT 1 while genotype Wyalkatchem was susceptible with IT 3+. Generally, however, the pattern of distribution of genotypes for ITs was comparable between isolates (Figures 4.3a and b) with a few exhibiting resistance (ITs ≤ 2+) while a majority of exhibiting susceptibility (ITs > 2+).

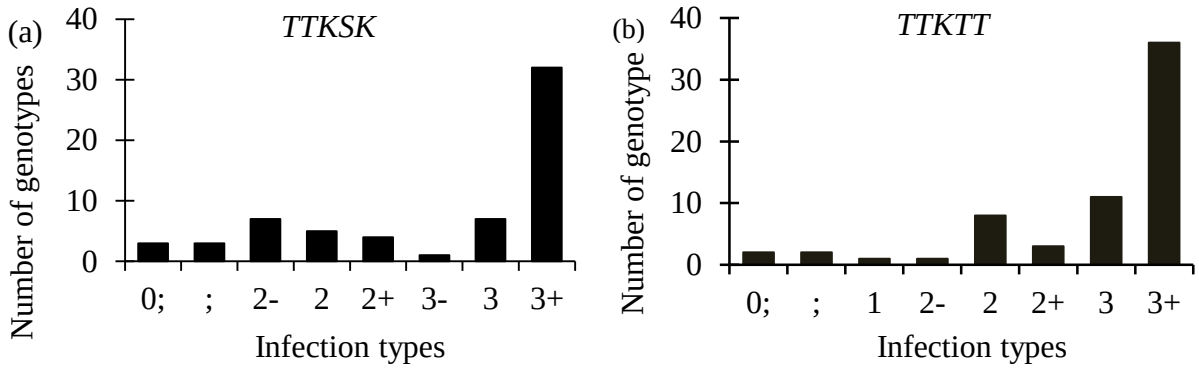


Figure 4.3 Frequencies of infection types for 62 Australian bread wheat (*Triticum aestivum* L.) introductions and two controls evaluated in the greenhouse against stem rust isolates (a) *TTKSK* and (b) *TTKTT*.

4.5 Discussion

A low infection type (ITs) implied the presence of the resistance gene(s) to which the tested isolate was avirulent (Jin *et al*., 2008). Low ITs to both isolates, therefore, suggested that the genotytpe(s) possessed effective resistance against both isolates. In this study, a number of genotypes which were identified for resistance at adult plant stage displayed seedling susceptibility and *vice versa*. Seedling susceptibility of genotypes which were resistant at adult plant stage confirms adult plant resistance (APR) (Figueroa *et al*., 2020; Lagudah *et al*., 2009; Rahmatov *et al*., 2019). Genotypes Sunguard, Lancer, Gauntlet and Magenta which were identified for APR were resistant to both isolates *TTKSK* and *TTKTT* while genotype Shield which was identified for APR was resistant to isolate *TTKSK* but susceptible to isolate *TTKTT*. On the other hand, genotypes Bolac and EGA Bounty were

susceptible to both isolates despite possessing APR. Given that none of the APR genotypes was immune at seedling stage suggests that observed APR was conferred by minor genes. These findings are similar to those of Mago *et al*. (2015) and Zhang *et al*. (2016) who reported seedling susceptibility in genotypes possessing APR. Conversely, genotypes Scepter, Spitfire, Merlin, Coolah, Janz, Dart and Preston were resistant to both isolates despite lacking APR thus suggesting the presence of major genes in these genotypes. This pattern of resistance was similar to that of Aleri *et al.* (2019) and Odemba (2018) who reported resistance and susceptibility during seedling and adult plant stages.

Resistance at seedling stage is usually associated with hypersensitive responses which are attributed to major genes. A hypersensitive response occurs when the pathogen attack signals defense mechanisms in the host that results in death of cells at or around the point of infection to restrict the spread of infection (Singh *et al*., 2014). However, evolution of virulence creates races which are virulent to these genes (Lewis *et al*., 2018; Niks *et al*., 2015; Saunders *et al*., 2019). Postulation uncovers genes for resistance and indicate variation in the resistance spectrum and other aspects of host-pathogen interaction (Flath *et al*., 2018; Singh *et al*., 2014). Besides, it helps in formulating research strategies on resistance to stem rust (Park *et al*., 2011). However, postulation of resistance was beyond the scope of this study.

Genetic resistance results in a positive economic outcome and reduces the negative ecologic impact of chemical control. Unfortunately, biological resistance is short-lived due to concurrent evolution of virulence against deployed resistance genes. It is therefore imperative to continuously search for diverse sources of resistance. Durable resistance to multiple races of stem rust is achieved when both major and minor genes are combined. Therefore, deployment of both gene classes could be an effective strategy against the disease.

4.6 Conclusion

This study established the existence of seedling resistance in the Australian bread wheat introductions. 17 genotypes (28.3%) were resistant to isolate *TTKSK* while 14 genotypes (22.6%) were resistant to isolate *TTKTT*. Genotypes Shield, Westonia, Gazelle, Orion, Supreme and Cutlass were resistant to isolate *TTKSK* but susceptible to isolate *TTKTT*. On the other hand, genotypes Bolac and Emu Rock were resistant to isolate *TTKTT* but susceptible to isolate *TTKSK*. Genotypes Lancer, Sunguard, Gauntlet, Scepter, Merlin, Magenta, Spitfire, Coolah, Dart, Janz and Preston exhibited resistance to both isolates.

CHAPTER FIVE

GENERAL DISCUSSION, CONCLUSIONS AND RECOMMENDATIONS

5.1 General discussion

Wheat is an important source of food and nutritional security in sub-Saharan Africa (CIMMYT & ICARDA, 2020; Ndung'u *et al*., 2016). The demand for wheat continues to increase owing to population growth, urbanization and change in eating habits (Fedoroff, 2015; McKenzie & Williams, 2015; Shiferaw *et al*., 2013). However, stem rust disease is a major biotic factor limiting its production in eastern Africa in general and Kenya in particular. The frequent occurrence of stem rust epidemics in the region underscores the importance of the disease (Prins *et al*., 2016; Soko *et al*., 2018; Wanyera & Wanga, 2016). In Kenya, yield losses of up to 100% attributed to stem rust have been reported in farmers' fields (Wanyera & Wanga, 2016). The disease causes a significant reduction in the quantity and quality of harvested kernels (Aleri *et al*., 2019; Odemba, 2018; Soko *et al*., 2018). Therefore, it is imperative that adapted cultivars are continuously bred for resistance to stem rust, grain yield and stability of performance in different environments.

The cultivation of resistant varieties has been effective against stem rust. However, mutation and somatic hybridization of the pathogen has led to evolution of more virulent races resulting in the rapid breakdown of existing resistance genes (Li *et al*., 2019; Park, 2016). In addition, such races emerge from sexual recombination, incursion of exotic spores and movement of spores within and between epidemiological zones (Olivera *et al*., 2019; Saunders *et al*., 2019; Soko *et al*., 2018). Furthermore, intensive selection for yield and selfing has substantially reduced the available genetic diversity hence narrowing the genetic base for resistance to stem rust (Cavanagh *et al*., 2013; Voss-Fels *et al*., 2015). Currently, over 90 % of the released varieties are susceptible to the disease. Therefore, constant efforts are needed to search for novel sources of resistance genes from diverse germplasm so that to mitigate the impact of the disease. This broadens the spectrum of genetic resistance and ensures that breeders are always a step ahead of the pathogen (Anderson *et al*., 2010; Mackay *et al*., 2016; Singh & Janeja, 2021). Genetic improvement for resistance to stem rust, grain yield and stability of performance across environments defines the success of a variety in terms of adoption by farmers and popularity with processors and consumers (Ceccarelli & Grando, 2007; Ndung'u *et al*., 2016; Tester & Langridge, 2010). Therefore, breeding aims at combining these qualities by exploiting genetic variation (Pretorius *et al*., 2017).

Deployment of diverse sources of resistance limits the evolution of the pathogen and reduces the severity of stem rust (Park, 2016). When adequate levels of adult plant resistance (APR) genes are accumulated or when 4-5 seedling resistance genes (R) are pyramided in suitable genetic backgrounds, other prophylactic measures of managing stem rust become obsolete (Bhavani *et al*., 2019; Luo *et al*., 2021; Zhang *et al*., 2019). The International Centre for Maize and Wheat Improvement (CIMMYT) facilitates breeding for resistance to stem rust, grain yield and stability of performance in target environments through the Borlaug Global Rust Initiative (BGRI) using shuttle breeding (Gupta *et al*., 2017; Tomar *et al*., 2014).

Since 2005, the search for resistance to current and anticipated stem rust races continue to be undertaken at the International Stem Rust Phenotyping Platform domiciled at the Kenya Agricultural and Livestock Research Organization (KALRO), Njoro. However, APR requires several rounds of evaluation due to the low level of expression and quantitative inheritance (Niks *et al*., 2015; Riaz & Hickey, 2017; Velu & Singh, 2013). In addition, although considerably effective, conventional phenotyping is weather dependent, time inefficient and often compromised by untargeted diseases and rare-variant associations (Riaz & Hickey, 2017; Singh & Janeja, 2021; Voss-Fels *et al*., 2019). On the other hand, greenhouse evaluation for seedling resistance to stem rust minimises variation in response and is more time and resource efficient (Prins *et al*., 2016; Riaz, 2018; Riaz & Hickey, 2017). However, the use of single R genes favour selection for more virulent mutants, which are usually present at low frequencies in the natural population, therefore rendering conferred resistance ineffective (Burdon *et al*., 2014; Niks *et al*., 2015). Therefore, the next-generation genotyping and sequencing technologies improves detection of rare alleles to better explain the observed variation (Varshney *et al*., 2014; Voss-Fels *et al*., 2019).

Notwithstanding, field and greenhouse evaluations are instrumental in facilitating the identification of genes for subsequent introgression into adapted cultivars to culminate into resistance to stem rust and eventual increase in grain yield, and stability of performance (Daetwyler *et al*., 2014; Mengistu *et al*., 2012; Voss-Fels *et al*., 2019). Breeding for resistance to stem rust, grain yield and stability of performance results in an optimum breeding benefit (Bernardo, 2010; Ceccarelli & Grando, 2007; Njau *et al*., 2013). This results from enhanced genotypic frequency for these traits (Leonardo *et al*., 2017; Sharma *et al*., 2012). However, the effectiveness of identified genes is dependent on the genetic diversity of donor sources in terms of mean and genotypic variance (Qian *et al*., 2017; Riaz, 2018; Vikram *et al*., 2016). Genotypes identified as possessing APR to stem rust could be used to

enhance the performance of popular Kenyan varieties like Kenya Wren, Kenya Korongo, Kenya Hawk12 and Njoro BWII which have low levels of resistance to stem rust (Macharia *et al*., 2016). Synchronously, utilising Kenyan variety Kenya Kingbird which possesses APR to stem rust (Macharia *et al*., 2016) as a parental line to breed genotypes identified as having high grain yield and agronomic performance for adaptability to the Kenyan environment could be worthwhile.

5.2 Conclusions

The following conclusions were drawn:

i. Genotypes Sunguard, Lancer, Gauntlet, Shield, Magenta, Bolac and EGA Bounty were identified as possessing adult plant resistance (APR) to stem rust.
ii. Similarly, genotypes Sunguard, Lancer, Gauntlet, Magenta, Merlin, Scepter, Spitfire, Coolah, Janz, Shield, Dart and Preston were uncovered for bearing seedling resistance, particularly to stem rust isolates *TTKSK* and *TTKTT*.
iii. Genotypes Sunguard, Lancer and Gauntlet were further identified as high yielding and possessing superior agronomic performance and yield stability in addition to possessing APR and seedling resistance to stem rust isolates *TTKSK* and *TTKTT*.

5.3 Recommendations

i. Further studies are suggested to understand the genetic basis of resistance to stem rust in genotypes identified in this study.
ii. Field trials at six to 10 locations in other major wheat growing regions would further confirm genotypic stability for resistance to stem rust and yield-related traits.
iii. Effectiveness of identified resistance ought to be investigated further using other stem rust races (or variants) such as the *Ug99* race variant *TTKTT+Sr8155-B1* [*TTKTT+*] which was recently detected in Kenya.
iv. Resistant genotypes with superior grain yield and stable in performance ought to be submitted to the national performance trials (NPT) for testing and possible release to farmers or incorporated in Kenyan breeding programmes as sources of genes for resistance to stem rust, grain yield and agronomic performance.
v. Considering that only a few resistance genes are effective against current and anticipated stem rust races, their deployment could be staggerred to minimise the possibility of being rendered ineffective.

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APPENDICES

GS00-09: Germination	GS50-59: Ear emergence
GS03 Completion of imbibition	GS51 First spikelet of ear visible above flag leaf
GS07 Coleoptile emerged	GS55 Ear 50% emerged on main stem
GS10-19: Seedling growth	GS59 Ear emergence is complete
GS10 First leaf through coleoptile	GS60-69: Flowering
GS11 First unfolded leaf	GS61 Start of flowering
GS13 3 unfolded leaves	GS65 Pollen sacs visible on outside of glumes
GS15 5 unfolded leaves	GS69 Flowering complete
GS19 ≥ 9 unfolded leaves	GS70-79: Grain filling (Milk development)
GS20-29: Tillering	GS71 Start of grain-filling (grain watery ripe)
GS20 Main shoot only	GS73 Early milk
GS21 Main shoot and 1 tiller	GS75 Medium milk
GS23 Main shoot and 3 tillers	GS77 Late milk
GS25 Main shoot and 5 tillers	GS80-89: Dough development
GS29 Main shoot and 9 ≥ tillers	GS83 Early dough stage
GS30-39: Stem elongation	GS85 Soft dough stage
GS30 Start of stem elongation	GS87 Hard dough stage
GS31 First node detectable	GS90-99: Ripening
GS32 Second node detectable	GS91 Grain hard (difficult to divide)
GS33 Third node detectable	GS92 Grain hard (not dented by thumbnail)
GS39 Flag leaf emergence	GS93 Grain loosening in daytime
GS40-49: Booting	GS94 Overripe
GS41 Flag leaf sheath extending	GS95 Seed dormant
GS43 Start of booting phase	GS96 Viable seed has 50% germination
GS45 Flag leaf sheath swollen	GS97 Seed not dormant
GS47 Flag leaf sheath opening	GS98 Secondary dormancy
GS49 Leaf sheath splitting open	GS99 Secondary dormancy lost

Appendix 1. Growth stages of wheat.

Source: Zadoks *et al*. (1974).

Appendix 2. Pedigree for introduced Australian bread wheat genotypes.

Genotype	Pedigree
Coolah	EGA Gregory/VQ2791//EGA Gregory
DS Faraday	Gregory/UQ01484//3*Gregory
Chara	BD225/CD87
LRPB Flanker	EGA Gregory//EGA Gregory/Lang
LRPB Reliant	LRPB Crusader/EGA Gregory
Ninja	Calingiri/Wyalkatchem derivative
Sunmax	CRW142.16/2*SunzellA
Tenfour	N/A
Tungsten	Axe with a European winter wheat background
Axe	-0AUS/DT29361//RAC820/Excalibur/3/-0AUS/DT29361//RAC820/Excalibur
B53	N/A
Beckom	N/A
Bremer	DM02-25-SB02-167/Correll// Mace
Buchanan	Frederick/Sprague
Calingiri	Chino/Kulin//Reeves
Cobalt	N/A
Cobra	Westonia/W29
Condo	WW-80/2*WW-15
Corack	Wyalkatchem/Silverstar A// Wyalkatchem
Correll	CHA/Mengavi-8156//CNO67/GLL//Bezostaya 2/4/N10/BVR14 //5*Burt/3/3*Raven/5/Sr21/4*Lance//4*Bayonet/6/C 8 MM/C 8 HMM/4/M-8-DAG-3-B19-H9-9/Dagger/3/Sabre/MEC 3//Insignia
Cosmick	N/A
Cutlass	RAC1316/2*Fang
Dart	Sunbrook/Janz//Kukri
Derrimut	N/A
DS Darwin	Maris-Huntsman/Boxer//Monopol
DS Pascal	FAWWON105/CFR00-687-55
EGA Bounty	Batavia/2*Leichhardt
EGA Gregory	Pelsart/2*Batavia DH
Baxter	QT2327/Cook//QT2804
Emu Rock	96W657-37/Kukri
Espada	CO5583*B117/NH5441*F03//RAC875-2/-0AUS/3/-0AUS/DT29361//RAC820/EXCALIBUR
Estoc	Trident/Molineux/4/VPM 1/5*COOK//3*Spear/3/Sabre/MEC 3//Insignia /5/VM931/RAC935
Forrest	96 WFHB 5568/2*Kohika
Gauntlet	Kukri/Sunvale
Gazelle	24K1056/VPM/3*Vasco

Janz	3-AG-3/4*Condor//Cook
Kiora	N/A
Lancer	VII84/Chara//Chara/3/Lang
Livingston	SUN129A/Sunvale
Mace	Wyalkatchem/Stylet//Wyalkatchem
Magenta	Carnamah/Tammin-18
Merlin	Calidad//Yecora F 70/Ciano F 67/3/76ECN44/4/Hartog*3/Quarrion
Mitch	QT10422/GILES
Orion	TATIARA/QAL2000
Gladius	CO5583*B117/NH5441*F03//RAC875-2/-0AUS/3/-0AUS/DT29361//RAC820/Excalibur
Preston	N/A
Scepter	RAC1480/2*Mace
Scout	Sunstate/QH71-6//Yitpi
Shield	AGT-Scythe/CO-7138(CO-7412)//(CO-7413)RAC-1105/CO-7165
Spitfire	Drysdale/Kukri
Steel	Composite cross of unknown germplasm
Sunguard	SUN289E/*Sr2*Janz
Bolac	Nesser/2*VI252
Suntop	Sunco/2*Pastor//SUN436E
Supreme	LoPh-Nyabing.3*Calingiri/4*VPM Arrino
Trojan	LPB 00LR000041/Sentinel3R
Viking	(S) Early-Baart[113];
Wallup	Chara/Wyalkatchem
Westonia	Spica/Timgalen//Tosca/3/Cranbrook//Bob-White*2/Jacup
Wyalkatchem	Machete/W84-129*504
Yitpi	C8MMC8HMM/Frame
Zen	Calingiri/Wyalkatchem

N/A Not available.

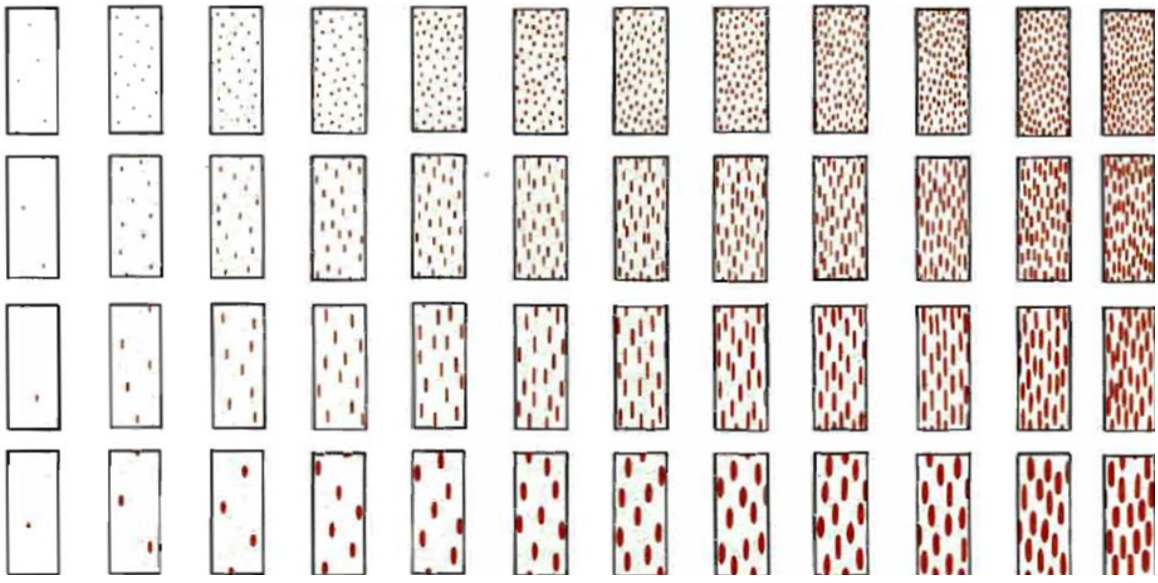
Appendix 3. (a) Adult plant resistance host plant reactions (HPRs) and disease severity (DS) for *Puccinia graminis* f. sp. *tritici* and (b) the modified Cobb scale.

(a)

Host plant reactions	DS (%)	Symptoms
Immune	0	No uredinia or other macroscopic signs of infection.
Resistant	1-5	Small uredinia surrounded by necrosis.
Resistant to moderately resistant	10-20	-
Moderately resistant	20-30	Small to medium uredinia surrounded by chlorosis or necrosis.
Moderately resistant to moderately susceptible	30-40	-
Moderately susceptible	40-50	Medium-sized uredinia that may be associated with chlorosis.
Moderately susceptible to susceptible	50-70	Medium to large uredinia with very few or no chlorosis.
Susceptible	70-100	Large uredinia without chlorosis or necrosis

Source: Roelfs *et al.* (1992).

(b)



A:	0.37	1.85	3.7	7.4	11.1	14.8	18.5	22.2	25.9	29.6	33.3	37.0
B:	1	5	10	20	30	40	50	60	70	80	90	100

A: Actual percentage of urediniospores.

B: Disease severity on the modified Cobb scale.

Source: Peterson *et al*. (1948).

Appendix 4. Combined REML variance component analyses for selected parameters of 64 bread wheat (*Triticum aestivum* L.) genotypes evaluated for resistance to stem rust over two cropping seasons in 2019 at KALRO, Njoro.

(i) Response variate: Area under disease progress curve

Fixed model: constant + replicate + genotype + season + genotype.season
Random model: replicate.block
Number of units: 384

Estimated variance components

Random term	component	s.e.
replicate.block	-0.11	0.53

Residual variance model

Term	Model (order)	Parameter	Estimate	s.e
Residual	Identity	Sigma2	18.53	1.72

Deviance: -2*Log-Likelihood

Deviance	d.f.
1143.28	252

Tests for fixed effects

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	1.46	2	0.73	8.6	0.510
genotype	1444.63	63	22.81	186.7	<0.001
season	295.16	1	295.16	233.0	<0.001
genotype.season	111.34	63	1.77	233.0	0.001

Dropping individual terms from full fixed model

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	1.46	2	0.73	8.6	0.510
genotype.season	111.34	63	1.77	233.0	0.001

Standard errors of differences

	Replicate	Season	Genotype	Genotype.Season
Average	0.5109	0.4393	2.4700	3.5040
Maximum			2.4740	3.5140
Minimum			2.4690	3.5030

(ii) Response variate: Coefficient of infection

Fixed model: constant + replicate + genotype + season + genotype.season
Random model: replicate.block
Number of units: 384

Estimated variance components

Random term	component	s.e.
replicate.block	11.9	10.8

Residual variance model

Term	Model (order)	Parameter	Estimate	s.e
Residual	Identity	Sigma2	238.8	22.1

Deviance: -2*Log-Likelihood

Deviance	d.f.
1803.04	252

Tests for fixed effects

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	4.20	2	2.10	12.8	0.163
genotype	990.53	63	15.71	232.3	<0.001
season	249.33	1	249.33	233.0	<0.001
genotype.season	162.11	63	2.57	233.0	<0.001

Dropping individual terms from full fixed model

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	4.20	2	2.10	12.8	0.163
genotype.season	162.11	63	2.57	233.0	<0.001

Standard errors of differences

	Replicate	Season	Genotype	Genotype.Season
Average	1.3510	0.9436	5.4250	7.6100
Maximum			5.4350	7.6180
Minimum			5.4030	7.5490
Average variance of differences			29.430	

(iii) Response variate: Final disease severity

Fixed model: constant + replicate + genotype + season + genotype.season
Random model: replicate.block
Number of units: 384

Estimated variance components

Random term	component	s.e.
replicate.block	6.00	6.70

Residual variance model

Term	Model (order)	Parameter	Estimate	s.e
Residual	Identity	Sigma2	161.70	15.00

Deviance: -2*Log-Likelihood

Deviance	d.f.
1701.99	252

Tests for fixed effects

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	1.74	2	0.87	12.10	0.444
genotype	1248.02	63	19.78	228.10	<0.001
season	403.94	1	403.94	233.00	<0.001
genotype.season	157.23	63	2.50	233.00	<0.001

Dropping individual terms from full fixed model

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	1.74	2	0.87	12.10	0.444
genotype.season	157.23	63	2.50	233.00	<0.001

Standard errors of differences

	Replicate	Season	Genotype	Genotype.Season
Average	2.0070	1.2980	7.5130	10.5000
Maximum			7.5340	10.5200
Minimum			7.4700	10.3800
Average variance of differences			56.4400	110.3000

(iv) Response variate: Grain yield (t ha^{-1})

Fixed model: Constant + replicate + genotype + season + genotype.season
Random model: replicate.block
Number of units: 383 (1 unit excluded due to missing value)

Estimated variance components

Random term	component	s.e.
replicate.block	0.0102	0.0110

Residual variance model

Term	Model (order)	Parameter	Estimate	s.e
Residual	Identity	Sigma2	0.262	0.0243

Deviance: -2*Log-Likelihood

Deviance	d.f.
69.85	251

Tests for fixed effects

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	1.26	2	0.63	12.2	0.550
genotype	842.61	63	13.36	227.8	<0.001
season	446.03	1	446.03	232.1	<0.001
genotype.season	266.95	63	4.24	232.1	<0.001

Dropping individual terms from full fixed model

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	1.26	2	0.63	12.2	0.548
genotype.season	266.95	63	4.24	232.1	<0.001

Standard errors of differences

	Replicate	Season	Genotype	Genotype.Season
Average	0.08157	0.05231	0.30300	0.42350
Maximum	0.08162		0.32140	0.47360
Minimum	0.08147		0.30070	0.41760
Average variance of differences			0.09183	0.17940

(v) Response variate: Biomass (t ha^{-1})

Fixed model: constant + replicate + genotype + season + genotype.season
Random model: replicate.block
Number of units: 384

Estimated variance components

Random term	component	s.e.
replicate.block	0.02	0.55

Residual variance model

Term	Model (order)	Parameter	Estimate	s.e
Residual	Identity	Sigma2	17.96	1.66

Deviance: -2*Log-Likelihood

Deviance	d.f.
1137.07	252

Tests for fixed effects

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	21.61	2	10.80	9.4	0.004
genotype	182.26	63	2.88	199.8	<0.001
season	242.92	1	242.92	233.0	<0.001
genotype.season	75.73	63	1.20	233.0	0.166

Dropping individual terms from full fixed model					
Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	21.61	2	10.80	9.4	0.004
genotype.season	75.73	63	1.20	233.0	0.166

Standard errors of differences

	Replicate	Season	Genotype	Genotype.Season
Average	0.5354	0.4325	2.4490	3.4620
Maximum			2.4500	3.4620
Minimum			2.4490	3.4600

(vi) Response variate: Harvest index

Fixed model: constant + replicate + genotype + season + genotype.season
Random model: replicate.block
Number of units: 383 (1 unit excluded due to missing value)

Estimated variance components

Random term	component	s.e.
replicate.block	0.00013	0.00043

Residual variance model

Term	Model (order)	Parameter	Estimate	s.e
Residual	Identity	Sigma2	0.0129	0.00120

Deviance: -2*Log-Likelihood

Deviance	d.f.
-696.41	251

Tests for fixed effects

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	4.53	2	2.26	10.2	0.154
genotype	164.31	63	2.60	209.7	<0.001
season	12.72	1	12.72	232.1	<0.001
genotype.season	70.57	63	1.12	232.1	0.272
Dropping individual terms from full fixed model					
Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	4.60	2	2.30	10.2	0.150
genotype.season	70.57	63	1.12	232.1	0.272

Standard errors of differences

	Replicate	Season	Genotype	Genotype.Season
Average	0.01535	0.01162	0.06627	0.09334
Maximum	0.01536		0.07022	0.10430
Minimum	0.01532		0.06601	0.09279
Average variance of differences				0.008714

(vii) Response variate: Days to heading

Fixed model: Constant + replicate + genotype + season + genotype.season
Random model: replicate.block
Number of units: 384

Estimated variance components

Random term	component	s.e.
replicate.block	3.02	1.54

Residual variance model

Term	Model (order)	Parameter	Estimate	s.e
Residual	Identity	Sigma2	20.60	1.91

Deviance: -2*Log-Likelihood

Deviance	d.f.
1191.36	252

Tests for fixed effects

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	5.31	2	2.65	15.9	0.101
genotype	1053.40	63	16.72	241.5	<0.001
season	46.44	1	46.44	233.0	<0.001
genotype.season	11.57	63	0.18	233.0	1.000
Dropping individual terms from full fixed model					
Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	5.31	2	2.65	15.9	0.101
genotype.season	11.57	63	0.18	233.0	1.000

Standard errors of differences

	Replicate	Season	Genotype	Genotype.Season
Average	1.0380	0.4632	2.7500	3.7980
Maximum			2.7660	3.8100
Minimum			2.7180	3.7050
Average variance of differences			7.5630	14.4200

(viii) Response variate: Plant height (cm)

Fixed model: constant + replicate + genotype + season + genotype.season
Random model: replicate.block
Number of units: 384

Estimated variance components

Random term	component	s.e.
replicate.block	0.25	1.57

Residual variance model

Term	Model (order)	Parameter	Estimate	s.e
Residual	Identity	Sigma2	49.62	4.60

Deviance: -2*Log-Likelihood

Deviance	d.f.
1396.06	252

Tests for fixed effects

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	3.75	2	1.87	9.7	0.205
genotype	325.99	63	5.16	204.8	<0.001
season	17.70	1	17.70	233.0	<0.001
genotype.season	66.29	63	1.05	233.0	0.385
Dropping individual terms from full fixed model					
Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	3.75	2	1.87	9.7	0.205
genotype.season	66.29	63	1.05	233.0	0.385

Standard errors of differences

	Replicate	Season	Genotype	Genotype.Season
Average	0.9152	0.7190	4.0840	5.7640
Maximum			4.0860	5.7650
Minimum			4.0800	5.7520
Average variance of differences				

(ix) Response variate: Spike length (cm)

Fixed model: constant + replicate + genotype + season + genotype.season
Random model: replicate.block
Number of units: 384

Estimated variance components

Random term	component	s.e.
replicate.block	0.0149	0.0131

Residual variance model

Term	Model (order)	Parameter	Estimate	s.e
Residual	Identity	Sigma2	0.285	0.0264

Deviance: -2*Log-Likelihood

Deviance	d.f.
93.92	252

Tests for fixed effects

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	12.48	2	6.24	12.9	0.013
genotype	1353.72	63	21.47	232.9	<0.001
season	44.48	1	44.48	233.0	<0.001
genotype.season	133.65	63	2.12	233.0	<0.001

Dropping individual terms from full fixed model

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	12.48	2	6.24	12.9	0.013
genotype.season	133.65	63	2.12	233.0	<0.001

Standard errors of differences

	Replicate	Season	Genotype	Genotype.Season
Average	0.09049	0.05451	0.31740	0.44250
Maximum			0.31850	0.44330
Minimum			0.31520	0.43610
Average variance of differences				

(x) Response variate: Kernels spike^{-1}

Fixed model: constant + replicate + genotype + season + genotype.season
Random model: replicate.block
Number of units: 384

Estimated variance components

Random term	component	s.e.
replicate.block	-1.23	0.77

Residual variance model

Term	Model (order)	Parameter	Estimate	s.e
Residual	Identity	Sigma2	37.30	3.46

Deviance: -2*Log-Likelihood

Deviance	d.f.
1313.39	252

Tests for fixed effects

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	1.26	2	0.63	4.9	0.570
genotype	414.35	63	6.45	88.4	<0.001
season	0.65	1	0.65	233.0	0.420
genotype.season	6.22	63	0.10	233.0	1.000

Dropping individual terms from full fixed model

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	1.26	2	0.63	4.9	0.570
genotype.season	6.22	63	0.10	233.0	1.000

Standard errors of differences

	Replicate	Season	Genotype	Genotype.Season
Average	0.5253	0.6234	3.3630	4.8740
Maximum			3.4050	4.9870
Minimum			3.3430	4.8590
Average variance of differences			11.3100	23.7600

(xi) Response variate: Test weight (kg hL^{-1})

Fixed model: constant + replicate + genotype + season + genotype.season

Random model: replicate.block

Number of units: 350 (34 units excluded due to missing values)

Estimated variance components

Random term	component	s.e.
replicate.block	1.09	1.05

Residual variance model

Term	Model (order)	Parameter	Estimate	s.e
Residual	Identity	Sigma2	20.47	2.05

Deviance: -2*Log-Likelihood

Deviance	d.f.
1024.78	218

Tests for fixed effects

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	4.25	2	2.13	12.4	0.161
genotype	1057.53	63	16.77	197.7	<0.001
season	332.94	1	332.94	200.6	<0.001
genotype.season	125.46	63	1.99	201.2	<0.001

Dropping individual terms from full fixed model

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	3.15	2	1.57	12.4	0.246
genotype.season	125.46	63	1.99	201.2	<0.001

Standard errors of differences

	Replicate	Season	Genotype	Genotype.Season
Average	0.8018	0.5107	2.9380	4.0900
Maximum	0.8063		4.3370	6.5390
Minimum	0.7931		2.6710	3.6940
Average variance of differences	0.6430		8.7460	17.0700

(xii) Response variate: 1000-kernel weight (g)

Fixed model: constant + replicate + genotype + season + genotype.season
Random model: replicate.block
Number of units: 382 (2 units excluded due to missing values)

Estimated variance components

Random term	component	s.e.
replicate.block	0.114	0.169

Residual variance model

Term	Model (order)	Parameter	Estimate	s.e
Residual	Identity	Sigma2	4.446	0.414

Deviance: -2*Log-Likelihood

Deviance	d.f.
781.44	250

Tests for fixed effects

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	8.32	2	4.16	11.4	0.044
genotype	1643.71	63	26.04	220.8	<0.001
season	1103.8	1	1103.08	231.3	<0.001
genotype.season	311.15	63	4.94	231.3	<0.001

Dropping individual terms from full fixed model

Fixed term	Wald statistic	n.d.f.	F statistic	d.d.f.	F pr
replicate	9.04	2	4.52	11.4	0.036
genotype.season	311.15	63	4.94	231.3	<0.001

Standard errors of differences

	Replicate	Season	Genotype	Genotype.Season
Average	0.3140	0.2161	1.2440	1.7430
Maximum	0.3144		1.3870	2.1330
Minimum	0.3138		1.2340	1.7220
Average variance of differences			1.5470	3.0410

Appendix 5. Means of disease variables for 64 bread wheat genotypes evaluated for resistance to stem rust in 2019 at KALRO, Njoro.

	Area under disease progress curve			Coefficient of infection			Final disease severity			Host plant reaction	
Genotype	2019 OS	2019 MS	Mean	2019 OS	2019 MS	Mean	2019 OS	2019 MS	Mean	2019 OS	2019 MS
Cacuke	1496	1201	1376	97.0	95.3	96.0	96.9	96.5	96.6	S	S
Kenya Robin	1573	1329	1465	97.8	96.8	97.6	97.1	96.7	97.4	S	S
Coolah	750	620	703	55.5	47.9	51.4	68.7	53.2	60.7	S	S
DS Faraday	601	392	490	36.7	26.8	31.8	56.3	40.0	48.2	MSS	MSS
Chara	907	472	670	75.3	39.7	57.4	76.3	46.7	61.7	S	S
LRPB Flanker	861	341	566	59.4	16.1	37.7	70.0	30.1	50.2	S	MRMS
LRPB Reliant	497	148	293	46.3	10.5	28.1	50.2	16.7	33.6	MSS	MRMS
Ninja	1174	417	747	87.2	22.9	54.3	91.0	34.8	62.3	S	MSS
Sunmax	228	150	192	18.7	13.4	16.3	31.3	23.2	27.3	MSS	MRMS
Tenfour	1537	1536	1562	97.6	99.1	98.3	98.1	99.9	98.9	S	S
Tungsten	808	800	811	64.9	60.3	62.8	74.9	66.7	71.0	S	S
Axe	702	826	755	47.6	53.4	50.1	65.0	60.0	62.3	S	S
B53	1196	579	853	84.1	51.2	67.6	84.1	53.4	68.7	S	S
Beckom	343	237	283	19.8	13.2	16.3	34.5	25.0	29.7	MRMS	MRMS
Bremer	1240	792	990	84.3	57.7	71.0	84.4	60.0	72.1	S	MSS
Buchanan	1492	839	1158	95.9	62.6	79.1	96.2	63.2	79.5	S	S
Calingiri	1104	343	664	86.0	28.5	56.7	85.1	36.5	60.2	S	MSS
Cobalt	1089	944	1050	85.5	75.8	80.8	87.5	76.5	81.8	S	S
Cobra	374	81	198	25.4	6.5	16.3	39.5	10.5	25.5	MRMS	MRMS
Condo	1058	380	692	72.0	16.8	44.3	82.6	33.2	57.6	S	MRMS
Corack	440	135	265	19.7	5.5	12.6	40.8	10.0	25.3	MRMS	MRMS
Correll	1036	745	885	81.7	60.5	71.4	83.0	60.1	71.7	S	S
Cosmick	1403	385	807	95.1	31.6	63.3	94.8	35.0	64.8	S	S
Cutlass	632	187	386	46.6	14.9	30.8	55.6	23.3	39.5	S	MRMS
Dart	286	104	185	11.0	3.0	6.7	30.7	9.9	19.8	RMR	MRMS
Derrimut	760	355	542	53.5	26.8	40.5	59.4	31.7	45.7	S	S
DS Darwin	399	254	319	35.4	15.9	26.3	46.3	26.9	37.3	MSS	MRMS
DS Pascal	973	659	804	76.3	42.1	59.1	78.4	50.1	64.2	S	S
EGA Bounty	297	155	222	14.6	10.0	12.6	26.1	15.0	20.7	MRMS	MRMS
EGA Gregory	498	316	408	36.8	14.7	26.2	48.9	26.7	38.1	S	MRMS
Baxter	580	106	293	40.1	7.1	24.0	49.8	11.8	31.1	S	MRMS
Emu Rock	345	113	210	16.6	5.1	10.8	35.1	8.5	21.8	MRMS	MRMS
Espada	360	302	322	27.9	23.2	25.4	38.4	35.0	36.6	MSS	MS

Appendix 5 continued

	Area under disease progress curve			Coefficient of infection			Final disease severity			Host plant reaction	
Genotype	2019 OS	2019 MS	Mean	2019 OS	2019 MS	Mean	2019 OS	2019 MS	Mean	2019 OS	2019 MS
Estoc	393	303	354	36.1	15.4	25.8	42.4	29.9	36.0	MSS	MRMS
Forrest	450	94	238	31.6	6.8	19.4	42.9	11.7	27.5	MRMS	MRMS
Gauntlet	13	10	12	0.1	0.4	0.2	3.6	1.9	2.5	RMR	RMR
Gazelle	1028	877	968	86.6	63.0	75.0	88.6	63.2	75.7	S	S
Janz	311	109	190	17.2	7.6	12.5	34.1	13.4	23.8	MRMS	MRMS
Kiora	485	109	264	29.5	8.1	18.9	43.3	15.0	29.2	MSS	MRMS
Lancer	13	0	4	1.1	1.1	1.3	2.3	0.3	0.5	R	I
Livingston	1128	341	684	87.9	15.9	52.1	88.7	30.0	59.4	S	MRMS
Mace	806	258	491	71.0	13.6	42.9	72.1	23.5	48.3	S	MRMS
Magenta	194	59	116	7.7	3.4	5.3	19.6	6.6	12.8	MR	MRMS
Merlin	325	101	199	9.6	4.6	7.4	28.7	8.3	18.7	MR	MRMS
Mitch	1176	573	835	84.6	29.4	57.2	86.6	43.5	65.5	S	MSS
Orion	643	319	466	57.1	23.4	40.4	62.3	33.3	47.8	S	MSS
Gladius	385	172	265	31.4	10.9	21.1	40.6	20.1	30.4	MSS	MRMS
Preston	1438	706	1054	94.7	38.1	66.2	95.8	53.1	73.9	S	S
Scepter	479	133	279	39.3	4.9	22.0	49.9	11.7	30.8	S	MRMS
Scout	831	557	698	74.2	34.3	54.6	78.2	46.7	62.7	S	S
Shield	94	101	101	4.7	5.5	4.9	11.4	9.9	10.5	MRMS	MRMS
Spitfire	312	69	164	12.5	3.9	8.0	28.3	8.3	18.1	MR	MRMS
Steel	1560	969	1239	100.0	71.4	85.8	100.0	73.4	87.0	S	S
Sunguard	16	0	3	2.7	0.2	1.3	4.9	0.1	2.4	R	I
Bolac	175	76	123	5.6	4.6	5.0	19.5	9.9	14.5	MR	MRMS
Suntop	684	592	633	41.6	46.0	43.5	57.8	49.8	53.3	S	S
Supreme	553	121	293	44.6	5.4	24.7	54.8	10.1	32.4	MSS	MRMS
Trojan	447	293	353	40.2	17.2	28.9	44.6	30.3	38.0	MSS	MRMS
Viking	722	252	447	64.9	14.6	39.6	69.0	26.8	47.9	S	MRMS
Wallup	764	191	410	48.3	8.9	28.5	62.4	15.3	39.1	S	MRMS
Westonia	1082	184	533	69.4	6.8	38.3	76.7	11.9	44.7	S	MRMS
Wyalkatchem	680	199	397	52.5	8.4	30.5	60.9	15.1	38.1	S	MRMS
Yitpi	496	164	297	55.0	12.2	33.6	58.6	18.5	38.8	S	MRMS
Zen	766	295	498	59.4	18.4	38.5	71.0	29.9	50.1	S	MSS

I immune, R resistant, RMR resistant to moderately resistant, MR moderately resistant, MRMS moderately resistant to moderately susceptible, MS moderately susceptible, MSS moderately susceptible to susceptible and S susceptible, and 2019 OS 2019 off-season and 2019 MS 2019 main-season.

Appendix 6. Means of grain yield and agronomic performance for 64 bread wheat genotypes evaluated for resistance to stem rust in 2019 at KALRO, Njoro.

	Grain yield (t ha^{-1})			Biomass (t ha^{-1})			Harvest index			Kernels spike^{-1}		
Genotype	2019 O	2019 M	Mean	2019 O	2019 M	Mean	2019 O	2019 M	Mean	2019 O	2019 M	Mean
Cacuke	2.40	0.67	1.53	11.1	6.9	9.0	0.22	0.13	0.17	41	40	40
Kenya Robin	1.28	0.57	0.94	14.2	5.9	10.0	0.10	0.10	0.10	48	47	47
Coolah	1.57	0.78	1.17	16.4	8.5	12.6	0.10	0.09	0.10	37	36	37
DS Faraday	2.18	0.71	1.44	14.6	11.5	13.0	0.17	0.07	0.12	29	34	32
Chara	0.79	0.57	0.68	13.0	7.1	10.1	0.06	0.08	0.07	36	40	38
LRPB Flanker	1.94	0.81	1.37	16.9	11.3	14.3	0.16	0.08	0.11	42	40	41
LRPB Reliant	3.34	1.10	2.22	21.4	6.6	13.9	0.16	0.16	0.16	49	43	46
Ninja	0.32	0.45	0.39	10.1	6.8	8.6	0.03	0.07	0.05	31	33	32
Sunmax	0.58	1.10	0.85	31.2	11.7	21.5	0.02	0.09	0.06	51	51	51
Tenfour	0.60	0.59	0.59	11.8	5.1	8.5	0.10	0.13	0.11	42	39	40
Tungsten	0.71	0.41	0.55	12.3	7.5	9.8	0.06	0.06	0.06	39	39	39
Axe	2.83	0.94	1.88	11.0	6.8	8.9	0.26	0.14	0.20	39	38	38
B53	1.64	0.46	1.04	13.9	4.7	9.4	0.15	0.10	0.12	41	41	41
Beckom	2.38	0.96	1.67	11.7	5.3	8.6	0.20	0.19	0.19	44	41	43
Bremer	0.72	0.40	0.55	18.2	6.8	12.6	0.05	0.06	0.05	32	33	33
Buchanan	1.76	0.93	1.34	11.0	9.0	10.1	0.18	0.11	0.14	36	35	35
Calingiri	0.61	0.38	0.49	11.2	6.1	8.7	0.06	0.06	0.06	34	33	33
Cobalt	0.69	0.50	0.60	10.0	6.1	8.3	0.07	0.09	0.08	31	36	33
Cobra	2.13	0.78	1.46	20.5	6.2	13.3	0.10	0.12	0.11	32	34	33
Condo	3.01	1.14	2.06	11.4	5.9	8.6	0.27	0.20	0.24	50	48	49
Corack	1.73	1.02	1.37	13.9	4.6	9.2	0.14	0.28	0.21	36	34	35
Correll	1.95	0.40	1.18	15.7	9.1	12.5	0.13	0.04	0.09	29	29	29
Cosmick	0.23	0.34	0.28	9.4	4.3	7.0	0.02	0.08	0.05	32	32	32
Cutlass	0.91	0.45	0.69	16.5	9.3	12.8	0.07	0.04	0.05	28	28	28
Dart	3.32	1.00	2.16	8.5	6.3	7.5	0.48	0.14	0.32	43	42	43
Derrimut	2.51	1.18	1.85	14.7	10.6	12.5	0.18	0.12	0.15	44	44	44
DS Darwin	3.61	1.06	2.34	22.6	9.8	16.0	0.17	0.11	0.14	39	39	39
DS Pascal	0.40	0.32	0.35	11.5	6.2	8.7	0.04	0.05	0.05	28	27	27
EGA Bounty	3.07	1.46	2.25	12.9	9.1	10.9	0.27	0.17	0.22	38	38	38
EGA Gregory	2.39	0.87	1.64	15.5	10.8	13.3	0.15	0.08	0.11	35	34	34
Baxter	3.45	1.73	2.59	17.9	8.8	13.4	0.20	0.21	0.19	46	45	46
Emu Rock	2.28	0.95	1.62	13.5	5.7	9.5	0.18	0.17	0.18	30	30	30

Appendix 6 continued

	Grain yield (t ha^{-1})			Biomass (t ha^{-1})			Harvest index			Kernels spike^{-1}		
Genotype	2019 O	2019 M	Mean	2019 O	2019 M	Mean	2019 O	2019 M	Mean	2019 O	2019 M	Mean
Espada	3.16	1.24	2.20	17.2	10.5	13.9	0.18	0.11	0.15	44	43	44
Estoc	2.26	0.83	1.55	20.5	9.0	14.8	0.12	0.09	0.11	38	38	38
Forrest	1.41	0.50	0.96	16.0	10.2	13.1	0.09	0.05	0.07	32	33	32
Gauntlet	2.79	1.33	2.06	16.0	8.9	12.4	0.20	0.16	0.18	43	43	43
Gazelle	0.41	0.30	0.35	13.2	6.6	9.9	0.06	0.04	0.05	36	36	36
Janz	3.19	1.15	2.17	17.2	8.2	12.7	0.19	0.14	0.16	37	36	36
Kiora	1.79	0.76	1.28	21.3	9.8	15.5	0.08	0.08	0.08	43	43	43
Lancer	3.86	2.44	3.15	16.0	12.6	14.3	0.74	0.20	0.47	38	38	38
Livingston	2.65	1.69	2.17	13.0	7.3	10.2	0.22	0.23	0.22	44	44	44
Mace	2.79	0.68	1.74	13.6	5.2	9.4	0.22	0.14	0.18	37	37	37
Magenta	4.93	1.81	3.37	16.8	9.1	12.9	0.31	0.20	0.25	42	41	41
Merlin	4.02	1.23	2.63	14.9	7.6	11.3	0.39	0.17	0.28	38	37	38
Mitch	0.68	0.66	0.67	12.4	7.4	9.9	0.05	0.09	0.07	46	42	44
Orion	1.34	0.56	0.95	11.7	7.0	9.4	0.15	0.09	0.12	29	28	29
Gladius	3.28	1.44	2.36	19.7	10.6	15.2	0.18	0.13	0.16	40	39	40
Preston	0.14	0.37	0.26	7.0	3.9	5.5	0.01	0.09	0.05	35	34	35
Scepter	3.60	1.81	2.70	15.5	10.4	12.9	0.23	0.18	0.20	38	37	37
Scout	1.26	0.61	0.93	12.8	7.4	10.1	0.10	0.08	0.09	43	42	43
Shield	3.07	1.13	2.10	18.1	7.8	12.9	0.19	0.15	0.17	45	43	44
Spitfire	3.86	1.36	2.61	13.5	7.5	10.5	0.31	0.18	0.24	40	40	40
Steel	1.20	0.77	0.98	10.8	10.2	10.5	0.10	0.08	0.09	53	53	53
Sunguard	3.64	1.63	2.64	20.9	10.5	15.7	0.19	0.15	0.17	43	41	42
Bolac	2.25	1.05	1.65	24.8	7.4	16.1	0.10	0.15	0.12	37	36	36
Suntop	3.10	1.52	2.31	13.7	14.1	13.9	0.26	0.10	0.18	38	37	38
Supreme	1.61	0.62	1.12	6.8	5.9	6.4	0.28	0.11	0.19	37	37	37
Trojan	1.65	0.87	1.26	14.4	7.4	10.9	0.12	0.12	0.12	38	38	38
Viking	2.97	1.29	2.13	16.1	9.6	12.9	0.21	0.14	0.18	34	33	34
Wallup	2.28	1.22	1.75	13.0	8.2	10.6	0.19	0.15	0.17	38	38	38
Westonia	0.66	0.73	0.69	6.4	4.9	5.7	0.12	0.15	0.14	28	27	28
Wyalkatchem	0.35	0.41	0.38	8.1	2.8	5.5	0.07	0.16	0.11	24	22	23
Yitpi	0.26	0.81	0.54	16.3	7.5	11.9	0.02	0.11	0.06	33	33	33
Zen	0.81	0.39	0.60	10.4	5.0	7.7	0.08	0.09	0.08	33	32	33

Appendix 6 continued

	Days to heading			Plant height (cm)			Spike length (cm)			1000-kernel weight (g)			Test weight (kg hL^{-1})		
Genotype	2019 O	2019 M	Mean	2019 O	2019 M	Mean	2019 O	2019 M	Mean	2019 O	2019 M	Mean	2019 O	2019 M	Mean
Cacuke	59	60	60	78.0	75.4	76.7	11.1	10.7	10.9	32.5	14.7	23.6	64.6	55.4	60.0
Kenya Robin	69	72	71	77.3	86.3	81.8	12.3	11.2	11.8	20.1	10.9	15.5	56.2	45.2	50.7
Coolah	78	81	79	82.7	81.7	82.2	10.6	10.4	10.5	18.6	13.7	16.1	62.1	56.8	59.4
DS Faraday	76	78	77	78.4	83.4	80.9	11.6	10.0	10.8	19.7	11.4	15.6	61.0	55.7	58.3
Chara	76	77	77	76.3	65.9	71.1	9.4	8.5	9.0	13.1	9.2	11.1	56.6	49.4	53.0
LRPB Flanker	75	79	77	81.2	89.1	85.1	10.4	10.5	10.4	19.5	10.7	15.1	67.9	48.5	58.2
LRPB Reliant	76	79	77	95.6	84.3	89.9	10.5	10.1	10.3	22.9	16.8	19.8	71.8	66.9	69.4
Ninja	79	81	80	66.6	72.0	69.3	9.3	9.5	9.4	12.7	10.2	11.5	40.7	42.0	41.3
Sunmax	79	82	80	87.0	80.1	83.6	10.7	10.6	10.7	10.7	12.4	11.6	47.5	53.9	50.7
Tenfour	51	54	53	62.6	65.9	64.3	7.0	8.1	7.5	15.6	13.1	14.3	56.2	48.7	52.4
Tungsten	73	78	75	76.6	71.5	74.0	9.6	9.2	9.4	17.9	10.2	14.1	52.0	47.9	50.0
Axe	52	56	54	69.5	69.0	69.2	8.4	7.6	8.0	27.9	16.8	22.4	67.9	54.1	61.0
B53	74	78	76	81.1	75.0	78.1	10.7	9.2	9.9	17.6	8.4	13.0	59.4	52.0	55.7
Beckom	71	75	73	68.7	67.1	67.9	8.1	7.6	7.9	17.9	14.2	16.0	68.5	59.8	64.2
Bremer	74	77	76	78.4	73.2	75.8	9.8	8.9	9.4	16.5	8.2	12.4	58.4	46.8	52.6
Buchanan	70	73	72	87.8	86.3	87.0	9.9	10.0	9.9	21.9	13.4	17.7	62.7	52.8	57.7
Calingiri	81	83	82	81.6	72.7	77.2	8.9	8.5	8.7	14.4	8.9	11.7	51.0	42.7	46.8
Cobalt	69	72	71	79.8	80.4	80.1	10.4	9.9	10.1	14.5	9.5	12.0	56.3	52.7	54.5
Cobra	73	81	77	76.7	63.9	70.3	10.5	8.8	9.7	18.7	12.7	15.7	61.0	57.4	59.2
Condo	71	70	71	76.3	72.4	74.4	9.8	9.2	9.5	27.6	14.6	21.1	69.2	61.2	65.2
Corack	66	68	67	72.7	60.8	66.7	8.5	7.6	8.1	22.0	13.4	17.7	65.0	60.1	62.5
Correll	72	74	73	80.8	71.9	76.3	8.7	8.7	8.7	23.6	9.1	16.3	58.8	39.0	48.9
Cosmick	68	72	70	79.5	69.6	74.6	9.2	8.1	8.6	12.8	9.4	11.1	47.9	46.7	47.3
Cutlass	79	84	81	84.0	79.8	81.9	10.1	9.7	9.9	14.5	9.2	11.9	54.2	48.2	51.2
Dart	51	54	52	70.2	69.1	69.7	8.7	8.8	8.7	24.5	15.5	20.0	72.5	61.3	66.9
Derrimut	62	67	65	72.3	67.4	69.8	7.3	7.4	7.4	24.7	15.7	20.2	72.9	63.9	68.4
DS Darwin	69	73	71	77.3	72.5	74.9	8.4	8.6	8.5	27.7	14.9	21.3	73.6	64.0	68.8
DS Pascal	73	78	75	73.9	68.0	71.0	9.9	9.7	9.8	11.1	6.6	8.9	48.1	43.3	45.7
EGA Bounty	65	66	66	90.9	86.8	88.9	11.7	10.0	10.9	27.7	15.7	21.7	74.8	59.7	67.3
EGA Gregory	80	83	82	86.9	85.4	86.2	10.5	10.4	10.5	22.9	11.9	17.4	71.2	55.0	63.1
Baxter	69	74	71	73.7	79.1	76.4	9.0	8.8	8.9	24.4	16.3	20.4	75.3	69.0	72.2
Emu Rock	57	60	59	66.9	58.7	62.8	7.6	6.9	7.3	23.0	15.3	19.2	64.5	54.8	59.7

Appendix 6 continued

	Days to heading			Plant height (cm)			Spike length (cm)			1000-kernel weight (g)			Test weight (kg hL^{-1})		
Genotype	2019 O	2019 M	Mean	2019 O	2019 M	Mean	2019 O	2019 M	Mean	2019 O	2019 M	Mean	2019 O	2019 M	Mean
Espada	66	70	68	71.4	70.6	71.0	7.9	8.3	8.1	24.6	15.0	19.8	71.0	54.8	62.9
Estoc	72	74	73	67.7	71.1	69.4	7.8	8.4	8.1	21.4	12.3	16.9	66.4	51.2	58.8
Forrest	82	84	83	67.8	71.6	69.7	11.4	10.2	10.8	18.7	9.6	14.1	63.1	41.8	52.5
Gauntlet	77	75	76	72.0	66.9	69.5	9.1	8.9	9.0	23.2	17.3	20.2	74.5	68.7	71.6
Gazelle	74	76	75	84.4	69.8	77.1	9.2	8.7	8.9	11.4	7.2	9.3	47.8	37.6	42.7
Janz	64	69	67	78.2	74.2	76.2	8.6	8.3	8.5	24.0	13.0	18.5	74.2	69.2	71.7
Kiora	69	72	70	76.8	81.5	79.1	9.3	8.7	9.0	16.5	12.9	14.7	63.4	59.6	61.5
Lancer	76	78	77	70.4	69.2	69.8	8.3	9.0	8.6	25.4	20.8	23.1	77.4	70.9	74.2
Livingston	53	57	55	69.3	72.3	70.8	7.4	8.1	7.8	31.4	20.9	26.1	74.5	67.2	70.9
Mace	70	74	72	70.5	65.2	67.9	8.8	8.2	8.5	25.9	12.7	19.3	69.8	59.2	64.5
Magenta	68	77	72	80.1	71.8	75.9	8.6	8.7	8.7	31.2	19.2	25.2	76.4	69.6	73.0
Merlin	64	66	65	73.0	72.4	72.7	8.5	8.2	8.4	28.6	18.9	23.7	77.1	66.0	71.5
Mitch	73	78	76	82.5	76.0	79.3	11.3	9.6	10.4	12.0	10.3	11.1	48.1	46.2	47.2
Orion	71	75	73	83.3	74.1	78.7	10.6	9.8	10.2	20.0	14.3	17.1	57.1	51.7	54.4
Gladius	65	69	67	73.4	75.9	74.7	7.6	8.1	7.8	27.4	19.3	23.4	69.4	61.6	65.5
Preston	70	72	71	69.9	68.1	69.0	8.7	8.0	8.4	13.5	11.6	12.5	50.3	48.3	49.3
Scepter	50	55	52	66.4	73.0	69.7	8.0	8.9	8.4	32.9	23.9	28.4	71.4	70.6	71.0
Scout	72	76	74	74.0	73.9	74.0	9.6	8.9	9.3	19.7	10.5	15.1	67.6	51.5	59.6
Shield	74	79	76	73.3	67.6	70.5	8.0	8.1	8.1	26.3	15.2	20.8	71.2	56.4	63.8
Spitfire	62	65	64	78.2	70.7	74.4	8.5	8.5	8.5	27.8	18.0	22.9	76.1	69.7	72.9
Steel	73	77	75	80.9	84.5	82.7	9.4	9.8	9.6	16.3	9.0	12.7	59.8	43.7	51.7
Sunguard	77	82	79	80.4	75.9	78.2	8.1	8.3	8.2	23.3	24.1	23.7	73.1	76.1	74.6
Bolac	72	76	74	78.5	73.3	75.9	8.5	8.7	8.6	19.0	15.7	17.3	65.5	65.6	65.5
Suntop	66	69	68	76.1	91.1	83.6	9.9	10.0	10.0	26.5	18.8	22.6	73.2	68.1	70.7
Supreme	54	59	56	63.6	57.7	60.7	8.6	8.2	8.4	20.9	13.8	17.3	67.5	57.3	62.4
Trojan	74	78	76	77.2	69.9	73.5	10.1	9.1	9.6	17.0	11.7	14.4	65.8	56.0	60.9
Viking	71	75	73	83.4	74.0	78.7	9.5	8.8	9.2	22.0	16.7	19.4	72.2	66.1	69.1
Wallup	61	62	62	79.1	70.6	74.9	8.6	8.4	8.5	21.6	17.8	19.7	68.3	65.1	66.7
Westonia	60	63	62	72.6	71.6	72.1	9.9	8.3	9.1	20.9	15.9	18.4	70.9	64.7	67.8
Wyalkatchem	61	62	61	66.1	50.1	58.1	7.5	6.7	7.1	14.7	13.0	13.8	60.4	56.7	58.6
Yitpi	71	74	72	75.0	78.6	76.8	9.9	9.5	9.7	12.8	15.1	14.0	64.8	64.4	64.6
Zen	83	84	83	69.2	64.3	66.8	7.4	7.7	7.6	13.6	8.6	11.1	60.9	47.0	54.0

Appendix 7. Superiority measure (P_i), mean squares (MS) of genotype-by-season interaction (GSI) and b_i values for area under disease progress curve.

Rank[a]	Genotype	Mean	P_i (10^1)	MS(GSI) (10^1)	b_i	Rank[a]	Genotype	Mean	P_i (10^1)	MS(GSI) (10^1)	b_i
Minimum response		3	0.00	0.00	0.01	Minimum response		3	0.00	0.00	0.01
1	Lancer	4	0.00	0.00	1.00	33	DS Faraday	490	832.35*	64.03*	0.06
2	Sunguard	3	0.02	0.02	0.81	34	Viking	447	943.64*	348.08*	0.03
3	Gauntlet	12	0.17	0.17	4.33	35	Wallup	410	1000.80*	522.67*	0.02
4	Shield	101	27.94	0.67	-1.86	36	Zen	498	1090.06*	349.61*	0.03
5	Bolac	123	53.37*	12.33	0.13	37	Derrimut	542	1140.06*	256.11*	0.03
6	Magenta	116	60.40*	24.81	0.10	38	Mace	491	1159.02*	477.04*	0.02
7	Sunmax	192	114.54*	7.04	0.17	39	Suntop	633	1334.51*	10.40	0.14
8	Dart	185	142.24*	47.60*	0.07	40	LRPB Flanker	566	1392.31*	428.42*	0.03
9	Spitfire	164	156.94*	88.17*	0.05	41	Coolah	703	1545.95*	22.82	0.10
10	Janz	190	167.81*	59.54*	0.06	42	Scout	698	1632.29*	113.54*	0.05
11	EGA Bounty	222	174.47*	27.74	0.09	43	Chara	670	1703.37*	296.81*	0.03
12	Merlin	199	179.24*	74.20*	0.06	44	Axe	755	1928.33*	31.28*	-0.10
13	Emu Rock	210	204.99*	79.94*	0.06	45	Westonia	533	1961.03*	1305.38*	0.01
14	Cobra	198	228.14*	130.67*	0.04	46	Condo	692	2060.71*	737.04*	0.02
15	Beckom	283	275.12*	14.42	0.12	47	Tungsten	811	2120.04*	0.04	1.63
16	Gladius	265	279.95*	66.67*	0.06	48	Calingiri	664	2179.88*	932.51*	0.02
17	Forrest	238	333.01*	196.08*	0.04	49	DS Pascal	804	2259.80*	151.00*	0.04
18	Corack	265	334.26*	142.11*	0.04	50	Livingston	684	2265.84*	998.46*	0.02
19	Espada	322	352.69*	3.38	0.22	51	Ninja	747	2536.35*	922.56*	0.02
20	DS Darwin	319	355.85*	29.04	0.09	52	Correll	885	2669.26*	128.81*	0.04
21	Kiora	264	391.11*	219.62*	0.03	53	Mitch	835	2801.50*	580.17*	0.02
22	Scepter	279	391.41*	184.82*	0.04	54	B53	853	2891.22*	608.03*	0.02
23	Estoc	354	393.68*	9.88	0.14	55	Gazelle	968	2998.92*	31.74*	0.09
24	LRPB Reliant	293	426.93*	188.16*	0.04	56	Cobalt	1050	3414.85*	29.04	0.09
25	Yitpi	297	433.64*	169.60*	0.04	57	Cosmick	807	3467.21*	1683.38*	0.01
26	Trojan	353	457.01*	33.14*	0.08	58	Bremer	990	3554.66*	315.38*	0.03
27	Supreme	293	510.40*	292.60*	0.03	59	Preston	1054	4215.10*	861.60*	0.02
28	Baxter	293	554.54*	354.20*	0.03	60	Buchanan	1158	4818.94*	682.67*	0.02
29	EGA Gregory	408	558.47*	47.60*	0.07	61	Steel	1239	5553.62*	556.81*	0.02
30	Cutlass	386	696.88*	311.04*	0.03	62	Cacuke	1376	6069.48*	129.00*	0.04
31	Wyalkatchem	397	807.48*	365.04*	0.03	63	Robin	1465	6999.74*	88.94*	0.05
32	Orion	466	831.10*	161.20*	0.04	64	Tenfour	1562	7803.12*	0.24	13.00

[a]Ranking of genotypes was based on Pi.

Appendix 8. Rainfall and temperature for KALRO, Njoro from 2009 to 2020[a].

Year	Parameter	Jan.	Feb.	Mar.	Apr.	May	Jun.	Jul.	Aug.	Sep.	Oct.	Nov.	Dec.
2009	Rainfall (mm)	21.7	5.7	24.8	62.7	173.8	13.6	42.2	56.3	45.1	74.8	62.2	76.7
		(12)	(4)	(5)	(15)	(17)	(9)	(9)	(9)	(12)	(18)	(10)	(16)
	Max Temp (°C)	25.0	27.0	28.0	25.6	24.0	24.0	23.0	24.9	25.0	22.0	23.0	23.4
	Min Temp (°C)	8.8	8.0	9.0	9.4	9.4	8.0	7.0	9.13	8.0	10.0	9.0	9.8
2010	Rainfall (mm)	42.9	157.0	184.1	140.4	180.8	51.8	166.1	240.0	172.2	109.9	53.1	14.3
		(9)	(14)	(21)	(15)	(15)	(11)	(18)	(22)	(21)	(21)	(12)	(4)
	Max Temp (°C)	24.0	25.0	23.0	23.0	22.0	22.0	21.0	20.0	22.0	22.0	22.6	23.6
	Min Temp (°C)	9.0	10.0	10.0	10.0	11.0	9.0	8.0	8.0	8.0	10.0	8.4	8.1
2011	Rainfall (mm)	3.9	9. 5	130.3	28.9	120.5	177.7	158.6	124.9	145.4	102.1	165.3	104.6
		(1)	(3)	(14)	(11)	(13)	(18)	(19)	(18)	(19)	(14)	(17)	(12)
	Max Temp (°C)	25.0	26.0	26.0	25.0	23.0	21.0	18.0	15.0	22.0	22.0	20.0	16.0
	Min Temp (°C)	8.0	8.0	9.0	9.0	9.0	11.0	11.0	12.0	11.0	11.0	11.0	15.0
2012	Rainfall (mm)	0.0	13.6	11.0	295.0	183.7	62.1	87.3	174.7	174.9	98.3	28.0	112.7
		(0)	(4)	(4)	(26)	(22)	(13)	(18)	(14)	(22)	(18)	(6)	(14)
	Max Temp (°C)	23.0	18.0	22.0	24.0	22.0	22.0	20.0	20.0	22.0	23.0	21.0	21.0
	Min Temp (°C)	10.0	16.0	18.0	14.0	12.0	12.0	10.0	10.0	9.0	11.0	14.0	13.0
2013	Rainfall (mm)	37.8	2.5	57.5	238.3	110.9	142.7	150.1	110.6	173.3	73.9	60.6	137.5
		(6)	(1)	(9)	(21)	(14)	(16)	(17)	(13)	(20)	(13)	(17)	(11)
	Max Temp (°C)	23.0	25.0	24.0	20.0	23.0	21.0	21.0	22.0	23.0	21.0	22.0	23.0
	Min Temp (°C)	9.0	13.0	15.0	14.2	9.0	11.0	9.0	8.0	9.0	10.0	10.0	10.0
2014	Rainfall (mm)	9.4	30.9	80.6	61.5	102.3	96.4	85.8	160.4	50.2	74.2	48.5	39.3
		(4)	(4)	(12)	(13)	(7)	(14)	(12)	(16)	(9)	(9)	(14)	(8)
	Max Temp (°C)	23.7	23.6	23.9	24.3	24.1	19.3	23.2	22.1	20.7	23.5	22.9	23.5
	Min Temp (°C)	7.7	9.5	9.2	9.2	13.3	12.4	10.0	9.6	12.4	9.6	11.0	13.0
2015	Rainfall (mm)	1.0	13.9	16.1	168.2	213.4	83.0	54.5	53.1	78.1	67.3	117.4	86.2
		(2)	(4)	(4)	(14)	(16)	(11)	(9)	(5)	(8)	(12)	(23)	(8)
	Max Temp (°C)	25.0	28.0	23.2	25.0	22.0	23.0	23.0	24.0	25.0	24.0	23.0	22.0
	Min Temp (°C)	11.2	12.6	12.4	11.0	10.0	10.0	10.0	11.0	8.0	11.0	11.0	13.0
2016	Rainfall (mm)	70.2	15.8	49.5	244.0	125.7	105.5	98.8	128.5	105.9	65.8	46.8	4.7
		(9)	(2)	(7)	(13)	(12)	(9)	(16)	(13)	(12)	(6)	(6)	(2)
	Max Temp (°C)	24.0	25.0	27.0	24.0	23.0	23.0	22.0	23.0	24.0	25.0	22.0	25.0
	Min Temp (°C)	10.0	10.0	11.0	11.0	11.0	9.0	9.2	8.0	8.0	13.0	9.0	7.0
2017	Rainfall (mm)	10.5	23.2	40.8	46.4	124.1	27.3	271.3	152.9	86.5	107.1	54.5	2.3
		(2)	(6)	(5)	(9)	(13)	(6)	(17)	(18)	(12)	(13)	(10)	(1)
	Rainfall (mm)	28.0	27.0	28.0	25.0	23.0	24.0	23.0	23.0	23.0	24.0	22.0	18.0
	Max Temp (°C)	9.0	8.0	17.0	12.0	12.0	10.0	10.0	9.0	10.0	10.0	12.0	17.0
2018	Rainfall (mm)	7.2	10.0	46.7	235.3	153	191.6	55.9	102.2	24.5	43.5	11.2	67.6
		(2)	(2)	(13)	(20)	(18)	(18)	(9)	(13)	(7)	(7)	(2)	(15)
	Max Temp (°C)	18.4	18.9	21.0	23.0	23.0	22.1	22.0	23.0	25.0	25.0	25.0	24.0
	Min Temp (°C)	17.6	17.9	14.0	12.0	11.0	10.0	9.0	8.0	8.0	10.0	9.0	12.0
2019	Rainfall (mm)	8.1	14.3	14.2	42.4	53.5	230.6	106.8	132.6	34.7	152.8	101.7	289.4
		(2)	(5)	(3)	(6)	(11)	(19)	(13)	(10)	(7)	(20)	(12)	(17)
	Max Temp (°C)	24.0	27.0	28.0	27.0	24.0	22.0	22.0	22.0	24.0	23.0	23.0	22.0
	Min Temp (°C)	10.0	10.0	8.0	10.0	10.0	12.0	9.0	9.0	9.0	10.0	11.0	12.0
2020[b]	Rainfall (mm)	77.9	37.4	91.0	265.0	129.8	50.0	-	-	-	-	-	-
		(9)	(10)	(10)	(19)	(17)	(20)	-	-	-	-	-	-
	Max Temp (°C)	23.0	23.0	25.18	24.0	23.0	23.5	-	-	-	-	-	-
	Min Temp (°C)	10.0	12.0	10.0	11.0	11.0	10.0	-	-	-	-	-	-

[a]Bracketed values are the number of days in the month with rainfall.

[b] - Missing value.

Source: KALRO Njoro Meteorological Station No. 903502 (1) (2020).

Appendix 9. Research permit.

REPUBLIC OF KENYA

NATIONAL COMMISSION FOR SCIENCE,TECHNOLOGY & INNOVATION

Ref No: **676980**

Date of Issue: **29/January/2020**

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