

Article

Multi-Pathotype Testing of Selected Kenyan Wheat Germplasm and Watkin Landraces for Resistance to Wheat Stripe Rust (*Puccinia striiformis* f. sp. *tritici*) Races

Mercy N. Wamalwa ^{1,2,*}, James Owuoche ², Joshua Ogendo ² and Ruth Wanyera ^{3,*}

¹ Department of Biological Sciences, Egerton University, P.O. Box 536, Njoro 20115, Kenya

² Department of Crops, Horticulture and Soil Sciences, Egerton University, P.O. Box 536, Njoro 20115, Kenya; owuoche@yahoo.com (J.O.); jogendo@egerton.ac.ke (J.O.)

³ Pathology Section, Kenya Agricultural and Livestock Research Organization, P.O. Box Private Bag, Njoro 20107, Kenya

* Correspondence: mercy.wamalwa@egerton.ac.ke (M.N.W.); wanyerar@gmail.com or wanyera@plantprotection.co.ke (R.W.)

Received: 16 October 2019; Accepted: 28 October 2019; Published: 18 November 2019



Abstract: Wheat stripe rust, caused by *Puccinia striiformis* f. sp. *tritici* (*Pst*), is one of the key diseases of economic importance in wheat worldwide. Host resistance, which follows the gene-for-gene hypothesis between the host and pathogen, has been used in wheat lines to resolve resistance specificities and postulate resistant genes. The objective of this study was to elucidate stripe rust resistance in a collection of Kenyan wheat lines and Watkin landraces to identify new sources of stripe rust (*Yr*) resistance. In this study, the resistance in twenty wheat lines was determined by comparing their infection type with those of twenty differential lines using isolates representing twelve *Puccinia striiformis* races from Kenya, Denmark, U.K., Sweden, and Eritrea at the seedling stage. Among the twenty wheat lines, none was resistant to all the twelve *Pst* races and isolate *DK02d/12* (“Kranich” race) was virulent on all the genotypes except wheat genotype “Kenya Tai.” This genotype (“Kenya Tai”) had the highest resistance as it was resistant to all the twelve stripe rust races used in this study. From this study, the introduction and utilization of wheat genotypes with adult plant resistant (APR) stripe rust genes, such as *Yr15*, are important in breeding wheat genotypes with effective resistance to wheat stripe rust in Kenya and worldwide.

Keywords: gene postulation; pathotype; *Pst*; resistance; stripe rust; wheat

1. Introduction

Wheat (*Triticum aestivum* L.) is the second-most important cereal crop in Kenya, after maize; however, there is inadequate annual production due to abiotic and biotic factors leading to a deficit of over 60% [1]. Wheat stripe rust caused by *Puccinia striiformis* f. sp. *tritici* (*Pst*) is one of the biotic factors that has been an important disease in Kenya for the past century [2,3] and its pathogenicity increases yearly [2,4,5]. The epidemiology of stripe rust is influenced not only by the deployment of resistance genes in wheat varieties, but also by climatic conditions that affect *Pst* infection and growth and by wind movements across areas where wheat is grown. Among the countries in East Africa, stripe rust is important in Kenya, Ethiopia, and Uganda [6]; hence, regular virulence analysis is vital for disease management and the incorporation of resistant genes [7]. A stripe rust race (pathotype) is the compatible or incompatible interactions between the host and pathogen by characterizing the *Pst* on a set of resistant (R) genes carrying wheat differentials, and associated race surveys are undertaken in

many countries [8,9]. In Kenya and Ethiopia, wheat cultivars with stripe rust genes *Yr9* and *Yr27* broke down due to the evolution of stripe rust races virulent to *Yr9* and *Yr27* genes, resulting to yield losses of up to 40% in commercial cultivars like “Paa,” which carried the *Yr9* gene [10,11]. These countries are at high risk of wheat rust’s diseases; therefore, it is necessary to identify and incorporate sources of resistance with improved durability in breeding programs to avoid major stripe rust epidemics [12,13].

The inheritance of resistance in wheat genotypes to wheat rust pathogens is due to R genes in the host plant and avirulent (Avr) genes in the wheat rust; thus, for every R gene in wheat, there is a corresponding Avr gene in the wheat rust pathogen [14,15]. Gene postulation has been used in several studies to identify stripe rust resistance genes in wheat lines from Nepal, Denmark, China, and Ethiopia [12,16–18]. Genes resistant to wheat rusts have been identified by testing genotypes and the differential sets (wheat lines with known genes) in different countries [19,20].

The aim of this study was to resolve the resistance specificities of major genes in Kenyan wheat genotypes by inoculating them with a panel of isolates of *Puccinia striiformis* of diverse origin and race at the seedling stage in the greenhouse. The isolates were selected from a previous study on virulence analysis of 54 *Pst* isolates collected within Kenya between 2009–2014 (unpublished data). The resistance spectra of the genotypes formed the elementary information for further gene postulation.

2. Materials and Methods

2.1. Genotypes

Twenty wheat genotypes consisting of Kenyan wheat varieties and Watkins landraces, together with twenty wheat differential lines, were used (Table 1). The Kenyan varieties were provided by the Kenya Agricultural and Livestock Research Organization (KALRO), Njoro, Kenya, and the Watkins lines were provided by John Innes Centre (JIC), Norwich U.K., through the Biotechnology and Biological Sciences Research Council under the Sustainable Crop Production Research for International (BBSRC SCPRID) award (BB/J012017/1). The virulence phenotyping of isolates was based on the infection type assessment of differential host genotypes with known yellow rust resistance genes. The differential sets consisted of European, world, avocet near isogenic lines, and others. The seeds of the differential sets were kindly provided by Prof. M.S. Hovmøller from the Global Rust Research Centre (GRRRC), Slagelse Denmark.

Table 1. Wheat (*Triticum aestivum* L.) genotypes and differential lines used for stripe rust (*Puccinia striiformis* (*Pst*)) virulence analysis.

Variety/Landrace	Year of Release	Pedigree/Accession Name	Seed Source
1190713	–	Watkin	JIC, U.K.
1190026	–	Watkin	JIC, U.K.
1190034	–	Watkin	JIC, U.K.
1190826	–	Watkin	JIC, U.K.
1190120	–	Watkin	JIC, U.K.
1109524	–	Watkin	JIC, U.K.
1190527 (Panjabi)	–	Watkin	JIC, U.K.
1190059	–	Watkin	JIC, U.K.
Kenya Korongo	2012	Emb16/CBRD//CBRD	KALRO, Njoro
Kwale	1987	Kinglet,Cm33089-W	KALRO, Njoro
Kenya Wren	2012	THELIN#2/TURUKU	KALRO, Njoro
Duma	1993	R674-AV/UP301//GLL/SX/3/PEW ‘S’/4/MAI ‘S’//PEW ‘S’-CM67245	KALRO, Njoro
NJBWII	2001	CM8181-12y-06PZ-4y-5m-0y-2AL-0AL-0M	KALRO, Njoro
Kenya Hawk12	2012	Equator-II/Kenya-Ploughman	KALRO, Njoro
Eagle10	2010	Babax/Lr42//Babax*2/4/SN1/TRAP#1/3/KAUZ*2/TR	KALRO, Njoro
Kenya Ibis	2012	Kwale/Duma	KALRO, Njoro
Kenya Kingbird	2012	TAM200/TUI/6/PVN/CAR422/ANA/5/BOW/CROW	KALRO, Njoro
Kenya Sunbird	2012	ND643/2*WBLL1	KALRO, Njoro
Kenya Tai	2012	ND643/2*WBLL1	KALRO, Njoro
Robin	2010	Babax/Lr42//Babax*2/3/Turuku	KALRO, Njoro
Host Differentials	Yellow Rust Genes (s)	Differential type/Grouping	Seed Source

Table 1. Cont.

Variety/Landrace	Year of Release	Pedigree/Accession Name	Seed Source
Cartago	None		Flak10
Chinese 166	Yr1	World	Flak10
Kalyansona	Yr2,+	Others	Flak08
Vilmorin 23	Yr3,+	World	Flak06
Hybrid 46	Yr4,+	European	Flak07
Heines Kolben	Yr6,+	World	Flak06
Avocet Yr6	Yr6,AvS	Avocet NIL	Flak09
Lee	Yr7	World	Flak09
Avocet Yr8	Yr8	Avocet NIL	Flak09
Avocet Yr9	Yr9,AvS	Avocet NIL	Flak12
Moro	Yr10	World	Flak09
Coatez	Yr15	Others	Flak09
VPM1	Yr17,+	European	Flak12
Avocet Yr17	Yr17,AvS	Avocet NIL	Flak09
TP 981	Yr25,+	Others	Flak09
Opata	Yr27,+	Others	Flak10
Carstens V	Yr32,Yr25,+	European	Flak06
Avocet S	AvS,+	Avocet NIL	Flak12
Ambition	Amb	Others	Flak12
Avocet SP	SP,AvS	Avocet NIL	Flak13

JIC—John Innes Centre, KALRO—Kenya Agriculture and Livestock Research Organization, NIL—near isogenic lines. Flak seed was sourced from the Global Rust Research Centre (GRRC).

2.2. Stripe Rust Isolates

Stripe rust isolates used in this study were provided by Prof. M.S. Hovmøller of GRRC, Denmark. A total of twelve stripe rust isolates of diverse origins from five countries (Kenya, Denmark, U.K., Sweden, and Eritrea) were used (Table 2). The isolates were selected due to their broad spectrum in terms of virulence, and because they represent different world regions that have been collected from for the last 40 years. Urediniospores used in this study had been previously (in a different study on virulence analysis, unpublished data) purified and developed on the susceptible wheat genotype “Cartago,” and subsequently stored over liquid nitrogen (−196 °C) at GRRC’s rust isolate bank in accordance with References [8,21,22].

2.3. Inoculation and Incubation of the Wheat Genotypes

Virulence assessment of the races on the resistance of the wheat genotypes based on their infection type (IT) was carried out on the Kenyan and Watkins lines, and differential sets. Seven seeds of each genotypes were sown in 6.5 cm × 6.5 cm square plastic containers filled with approximately 144 g of peat moss (PINDSTRUP MOSEBRUG A/S 8550 RYOMGARD, Denmark) and placed on a tray measuring 35 cm × 44 cm. The sown seeds were placed in a spore-proof greenhouse maintained at a temperature of 17 °C/12 °C day/night cycle. The stripe rust isolates were removed from the liquid nitrogen (−196 °C) and heat-shocked in a water bath at 40 °C for 2 min. At the two-leaf stage (feekes stage 1) [23], the wheat genotypes were inoculated with urediniospores (15 mg) of each isolate suspended in Novec™ 7100 using an air brush spray gun [24], then incubated in the dew chamber at 100% humidity and 10 °C for 24 h in total darkness. The inoculated plants were transferred to spore-proof greenhouse cabins with a 16-h/8-h day/night cycle, maintained at 17 °C/12 °C day/night cycle.

2.4. Field Experiment

Field experiments were conducted at the Kenya Agriculture and Livestock Research Organization (KALRO) in Njoro (0°20′ S, 35°56′ E, 2185 m above sea level), Mau Narok (01°55′ S, 35°52′ E, 2400 m above sea level), and Eldoret (0°41′ S, 36°31′ E, 2065 m above sea level) during the cropping cycle in 2016 and 2017. Each entry was sown in double rows measuring 0.2 × 0.75 m separated by 0.3 m and 0.5 m wide alleyways within and between the blocks, respectively. To facilitate inoculum build-up and uniform dissemination within the nursery, a continuous row of wheat variety “KS. Mwamba,”

susceptible to three types of rust, was planted perpendicular to all entries. The disease occurred naturally in all three environments.

2.5. Data Collection and Analyses

Infection types (IT) were noted 16 days after inoculation based on a 0–9 scale [25]. ITs of 0–4 were classified as resistant, ITs of 5–6 as intermediate, and ITs of 7–9 as susceptible. The resistance spectrum was based on a gene-for-gene hypothesis where the ITs produced by the *Pst* races on the wheat genotypes (Kenyan and Watkins lines) was compared with the ITs produced by the same races on the tester lines with known genes (differential lines) [16,26]. When the pathogen was avirulent, the response IT was classified as resistant; however, when the races were virulent on the host, the response IT was classified as susceptible and the intermediate reactions were classified as moderately resistant and moderately susceptible. The spectrum was comprised of resistant (R), moderately resistant (MR)/susceptible (MS), and susceptible (S) reactions. The disease in the field experiment was assessed during the peak of the wheat rust infection, immediately after heading. The host reaction and rust severity were taken from each entry. Estimates of disease severity were based on a modified Cobb scale [27,28] incorporating both the percentage of leaf area affected and the host response.

3. Results

3.1. Confirmation of Virulence Phenotype of the Twelve *Yr* Isolates and the Response of the Differential Lines to the Isolates

Collectively, the twelve *Pst* isolates were virulent on the following yellow rust (*Yr*) genes: *Yr1*, *Yr2*, *Yr3*, *Yr4*, *Yr6*, *Yr7*, *Yr8*, *Yr9*, *Yr10*, *Yr17*, *Yr25*, *Yr27*, *Yr32*, *YrAmb*, and *YrAvS*, and avirulent to *Yr15* and *YrSp* (Table 2). Isolates from Kenya and Denmark made up the majority of virulent isolates, consisting of 41.67% and 33.33%, respectively. The Kenyan isolates had a common virulence for yellow rust genes *Yr2*, *Yr6*, *Yr7*, and *YrAvS*, except *KE86102Yr9*, which lacked virulence for *Yr7* and *Yr8*, but was the only Kenyan isolate with virulence for *Yr17*. Virulence for *Yr17* was present in the European isolates. Two pathotypes, *KE70063* and *KE131/14*, had additional virulence for *Yr25* and *Yr27*; moreover, the latter also had virulence for *Yr1* and *Yr9*. The isolate from Eritrea had virulence for *Yr2*, *Yr6*, *Yr7*, *Yr8*, *Yr9*, *Yr25*, *YrAvS*, and an additional virulence for *Yr10* that was absent in the Kenyan isolates (Table 2). The Danish isolates (*DK01/09*; *DK71/93*) had virulence for 6 *Yr* genes, whereas isolates (*DK09/11SP*; *DK02d/12*) had virulence for 12 *Yr* genes, thereby representing isolates with a wide virulence spectrum. The other isolates from Europe (Sweden and United Kingdom) differed in virulence, *SE100/09* being virulent on only five *Yr* genes and *UK94/519* had virulence for seven *Yr* genes. Interestingly, the Swedish isolate and one Kenyan isolate were avirulent to *YrAvS*.

Wheat genotypes “Cartago” (susceptibility check) and “Kalyansona” (*Yr2*) were susceptible to all the twelve isolates (Table 3). “AvocetS” (*AvS*) was also susceptible to all the isolates except *KE70063* and *SE100/09* from Kenya and Sweden, respectively. On the other hand, “Cortez” (*Yr15*) was resistant to all the isolates, followed by “AvocetSP” (*SP*, *AvS*). “Hybrid46” (*Yr4+*), “Moro” (*Yr10*), and “Ambition” (*YrAmb*) were resistant to ten out of twelve isolates, while “Opata” (*Yr27+*) and “CarstansV” (*Yr32*, *Yr25*) were resistant to nine isolates (Table 3). “VPM1” (*Yr17*) and “Chinese166” (*Yr1*) were resistant to eight and seven isolates, respectively, and “AvocetYr17” was resistant to six isolates. Three varieties—“Vilmorin23” (*Yr3+*), “AvocetYr8,” and “AvocetYr9”—were resistant to five isolates. “AvocetYr6,” “Lee’ (*Yr7*),” and “Tp981” (*Yr25*) were resistant to four isolates. “Heines Kolben” (*Yr6*) and “AvocetS” (*YrAvS*) were resistant to three and two isolates, respectively.

3.2. Wheat Genotypes and Resistance Responses to Stripe Rust Isolates Representing Distinct Races

Interpretation of the results for each cultivar was based on the race-specific responses when challenged by twelve isolates (races), which produced 20 unique resistant spectra. Some spectra appeared to represent single *Yr* resistance genes, whereas others represented two or more genes

(often unknown). The susceptibility check “Cartago” proved to be susceptible to all isolates, whereas “Kalyansona” (Yr2+) showed an intermediate reaction (“MS”) to SE100/09 (race “Triticale 2006”) (Table 3). Thus SE100/09 had partial virulence to “Kalyansona,” either due to Yr2 or the additional “unknown” gene in this differential. “Vilmorin23” (Yr3+) showed resistance (R or MR) to seven isolates and susceptibility (S) to five isolates, with the former confirming avirulence to Yr3+ in these seven isolates and virulence to Yr3+ in the latter. “Hybrid46” showed resistance to 10 isolates and susceptibility to DK09/11SP, i.e., only the latter two isolates had virulence to Yr4+ (Table 3). Despite two differentials having carried Yr6, they revealed different spectra due to an uncharacterized R gene in AvocetYr6+. Thus, only resistance spectrum 6 (conferred by *Heines Kolben*) were considered for the postulation regarding Yr6 in this study.

Table 2. Pathotypes of *Puccinia striiformis* (*Pst*) used in seedling screening of the Kenyan varieties and Watkins landraces for resistance.

Pathotype ^a	Sampling Year	Country	Virulence ^b
KE79067	1979	Kenya	Yr2, Yr6, Yr7, Yr8, YrAvS
KE82017	1982	Kenya	Yr2, Yr6, Yr7, Yr8, YrAvS
KE70063	1970	Kenya	Yr2, Yr6, Yr7, Yr8, Yr25, Yr27
KE86102 Yr9	1986	Kenya	Yr2, Yr6, Yr9, Yr17, YrAvS
KE131/14	2014	Kenya	Yr1, Yr2, Yr6, Yr7, Yr8, Yr9, Yr25, Yr27, YrAvS
ER02/03	2003	Eritrea	Yr2, Yr6, Yr7, Yr8, Yr9, Yr10, Yr25, YrAvS
SE100/09	2009	Sweden	Yr2, Yr6, Yr7, Yr8, Yr10
DK01/95	1995	Denmark	Yr2, Yr3, Yr4, Yr9, Yr25, YrAvS
DK09/11SP	2011	Denmark	Yr1, Yr2, Yr3, Yr4, Yr6, Yr7, Yr9, Yr17, Yr25, Yr32, YrAmb, YrAvS
DK71/93	1993	Denmark	Yr1, Yr2, Yr3, Yr25, Yr32, YrAvS
DK02d/12	2012	Denmark	Yr1, Yr2, Yr3, Yr6, Yr7, Yr8, Yr9, Yr17, Yr25, Yr32, YrAmb, YrAvS
UK94/519	Unknown	United Kingdom	Yr1, Yr2, Yr3, Yr9, Yr17, Yr25, YrAvS

^a GRRC pathogen coding system; ^b Yellow rust virulence tested on differential genotypes carrying the resistant genes Yr1, Yr2, Yr3, Yr4, Yr6, Yr7, Yr8, Yr9, Yr10, Yr15, Yr17, Yr25, Yr27, Yr32; and resistance specificities in Ambition, Avocet SP, and Avocet S.

Tester *Heines Kolben* and *AvocetS* had a narrow resistance spectrum, where they showed resistance to *Pst* races DK71/93, UK94/519, DK01/95, KE70063, and SE100/09. “AvocetYr6,” “Lee,” and “TP981” showed resistance to *Pst* races KE82017, KE86102Yr9, DK09/11SP, KE79067, DK71/93, SE100/09, UK94/519, and DK01/95, and could not distinguish from the resistance spectrum of other testers. In the present study, Yr2 and Yr25 were the most frequent because the resistance spectrum of “Kalyansona” and “TP981” was covered by the resistance spectra of other testers. The testers “Vilmorin23,” “AvocetYr8,” and “AvocetYr9” showed resistance to *Pst* races KE82017, KE86102Yr9, DK09/11SP, KE131/14, KE79067, KE70063, DK71/93, SE100/09, UK94/519, DK01/95, and ER02/13. The tester genotype “Chinese166” showed resistance to *Pst* races KE82017, KE86102Yr9, KE79067, KE70063, SE100/09, DK01/95, ER02/13; “Vilmorin23” showed resistance to the same *Pst* races in “Chinese166” except DK01/95; and “TP981” showed resistance to *Pst* races in “Vilmorin23” except KE70063 and ER02/13. The resistance spectrum of testers “Vilmorin23” and “TP981” were covered by the resistance spectrum of another tester (Table 3).

Spectrum 1 (Yr1) consisted of differential “Chinese166” with infection types (ITs) 0–1 and 6–7 for the incompatible and compatible reactions, respectively. Spectrum 2 (None and Yr2) consisted of “Cartago,” “Kalyansona,” “1190120,” “K. Hawk12,” “Eagle10,” “K. Sunbird,” and “Robin” with ITs 6–8 and 4–7, which indicated the presence of an additional gene for Yr2 (Tables 3 and 4). Genotypes “1190120,” “K. Hawk12,” “Eagle10,” “K. Sunbird,” and “Robin” could have had the Yr2,+ unknown gene. Spectrum 3 (Yr3,+) was comprised of a differential “Vilmorin 23” with ITs of 6–7 for compatible reactions and 0–4 for the incompatible reaction, which was an indication of the presence of a second gene to Yr3. Spectrum 4 (Yr4,+) was comprised of “Hybrid 46” with all the incompatible reactions 0; 0–1, then 5–7; 7 for compatible reactions, which is an indication of an additional gene to Yr4.

Table 3. Seedling reaction of twenty testers with known yellow (*Yr*) genes to twelve isolates of *Puccinia striiformis* collected from European and East African countries.

Host Differentials	Resistant Gene(s)	KE82017		KE86102 Yr9		DK09/11SP		KE131/14		KE79067		KE70063		DK71/93		DK02d/12		SE100/09		UK94/519		DK01/95		ER02/13		Resistance Spectrum
		a	b	a	b	a	b	a	b	a	b	a	b	a	b	a	b	A	b	a	b	a	b	a	b	
Cartago	None	6–7	S	6–7	S	6–7	S	7	S	7	S	7	S	7	S	7	S	7	S	7	S	7	S	7	S	1
Chinese 166	<i>Yr1</i>	0	R	0	R	6–7	S	6–7	S	0	R	0	R	7	S	7	S	0	R	7	S	1	R	0	R	2
Kalyansona	<i>Yr2,+</i>	6–7	S	6–7	S	7	S	6–7	S	6–7	S	6–7	S	7	S	7	S	7	S	6–7	S	7	S	5–7	S	1
Vilmorin 23	<i>Yr3,+</i>	0–2	R	0–4	R	6–7	S	0–4	R	0	R	0–1	R	7	S	7	S	1	R	7	S	7	S	2	R	3
Hybrid 46	<i>Yr4,+</i>	0	R	0	R	5–7	S	0	R	0	R	0	R	1	R	0–1	R	1	R	7	S	1	R	1	R	4
Heines Kolben	<i>Yr6,+</i>	6–7	S	6–7	S	6–7	S	5–7	S	6–7	S	6–7	S	2–3	R	7	S	7	S	2	R	2–3	R	7	S	5
Avocet Yr6	<i>Yr6, AvS</i>	6–7	S	6–7	S	6–7	S	7	S	6–7	S	0–3	R	2–3	R	7	S	4–5	MS	2–3	R	2–5	R	6–7	S	5
Lee	<i>Yr7</i>	6–7	S	2–3	R	5–7	S	6–7	S	7	S	6–7	S	2	R	7	S	7	S	2	R	2	R	7	S	6
Avocet Yr8	<i>Yr8</i>	4–7	MS	0–3	R	0	R	5–7	S	4–6	MS	6–7	S	2	R	7	S	7	S	2	R	2	R	5–7	MS	7
Avocet Yr9	<i>Yr9, AvS</i>	0	R	6–7	S	6–7	S	6–7	S	0	R	0	R	1–2	R	7	S	0–1	R	6–7	S	7	S	7	S	8
Moro	<i>Yr10</i>	1–2	R	0	R	0	R	0–2	R	0–1	R	0–1	R	1–2	R	0–1	R	6–7	S	1	R	1	R	7	S	9
Coatez	<i>Yr15</i>	0	R	0	R	0	R	0–3	R	0	R	0	R	0–1	R	0	R	0	R	0	R	0	R	0	R	10
VPM1	<i>Yr17</i>	0–1	R	0–3	R	6–7	S	3–5	MR	0	R	0–1	R	1–2	R	6–7	S	1	R	6–7	S	2	R	1	R	11
Avocet Yr17	<i>Yr17, AvS</i>	4–5	MS	6–7	S	6–7	S	0–4	R	0–4	R	0	R	2–3	R	6–7	S	1	R	6–7	S	2	R	2–5	R	11
TP 981	<i>Yr25</i>	0–3	R	1–4	R	5–7	S	6–7	S	0–3	R	6–7	S	7	S	7	S	2	R	7	S	7	S	7	S	12
Opata	<i>Yr27,+</i>	0–4	R	0–4	R	0–3	R	7–8	R	2–3	R	6–7	S	2	R	2	R	2	R	2	R	2	R	2–4	R	13
Carstens V	<i>Yr32,25,+</i>	0–1	R	0–1	R	5–7	S	0–3	R	0	R	0–1	R	6–7	S	7	S	1	R	2	R	2	R	1–3	R	14
Avocet S	<i>AvS</i>	7	S	6–7	S	6–7	S	7	S	6–7	S	0	R	7	S	7	S	1–4	R	7	S	7	S	5–7	S	15
Ambition	<i>Amb</i>	0	R	0–3	R	5–7	S	0–3	R	0–3	R	0	R	2	R	8	S	2	R	3	R	2	R	2–3	R	16
Avocet SP	<i>SP, AvS</i>	0	R	0	R	6–7	S	0	R	0	R	0	R	2	R	0	R	0	R	2–3	R	2	R	0	R	16

a and b are infection type (ITs) and the reaction of genotype to the stripe rust isolates. R (resistant); MR (moderately resistant); MS (moderately susceptible); S (susceptible). ITs are given according to the scale of 0–9 [25]. The resistance response spectrum was according to Hovmöller [16].

Table 4. Seedling reaction of twenty wheat genotypes to twelve isolates of *Puccinia striiformis* collected from European and East African countries.

Wheat Varieties	KE82017		KE86102 Yr9		DK09/11SP		KE131/14		KE79067		KE70063		DK71/93		DK02d/12		SE100/09		UK94/519		DK01/95		ER02/13		Resistance Spectrum
	a	b	a	b	a	b	a	b	a	b	a	b	a	b	a	b	a	b	a	b	a	b	a	b	
1190713	5-7	S	1-3	R	2-6	M	2-3	MS	0	R	0-3	R	6-7	S	6-7	S	0	R	6-7	S	7	S	3-4	MR	22
1190026	3-5	MR	6-7	S	6-7	S	7-8	S	3-6	MS	6	S	7	S	7	S	1-2	R	7	S	7	S	7	S	15
1190034	0-2	R	1	R	0-3	R	0-2	R	0	R	0	R	5-7	MS	3-5	MS	1	R	6-7	S	6	S	2	R	21
1190826	1-5	M	5-6	MS	6-7	S	7-8	S	3-6	MS	6	S	5-7	MS	7-8	S	2-3	R	7	S	7	S	7	S	20
1190120	3-5	MS	6-7	S	7	S	7-8	S	4-6	MS	6-7	S	7	S	7-8	S	5-7	MS	7	S	7	S	7	S	1
1190524	6-7	S	5	MS	5-7	MS	6-7	S	5-7	S	5-6	MS	2-4	R	6-7	S	0	R	2	R	3-5	MS	7	S	22
1190527 (P)	0-1	R	0-1	R	0-3	R	6-7	S	0-3	R	0-1	R	7	S	7	S	5-6	S	2-3	R	7	S	4-5	MS	23
1190059	2	R	2	R	0-5	M	2-4	R	1	R	5-6	MS	7	S	4-7	MS	2	R	7	S	7	S	2	R	12
K. Korongo	4-5	MS	0	R	6-7	S	4-6	MS	5	MS	0-3	R	7	S	6	S	4-6	MS	7	S	7	S	6	S	24
Kwale	0	R	0-1	R	5-6	MS	7-8	S	0	R	0	R	0-1	R	7	S	0-1	R	2	R	2	R	7	S	25
K. Wren	6-7	S	0-4	R	6-7	S	5-6	MS	4-5	MS	5-6	MS	6-7	S	4-6	MS	7	S	6-7	S	7	S	6-7	S	24
Duma	0-3	R	0-2	R	5-7	MS	3-6	MR	1-6	M	5-7	MS	3	R	3-7	M	0	R	2	R	2-3	R	7	S	18
NJRBW2	0	R	3-4	MR	5-7	MS	7	S	0	R	0	R	1	R	7	S	0	R	7	S	7	S	2-4	R	25
K. Hawk 12	6-7	S	4-6	MS	4-6	MS	4-6	MS	6-7	S	6-7	S	7	S	4-6	MS	7	S	7	S	6-7	S	7	S	1
Eagle 10	6-7	S	4-6	MS	6-7	S	7	S	7	S	6-7	S	7	S	7	S	6-7	S	7	S	7	S	6-7	S	1
K. Ibis	0-1	R	0-1	R	0-4	R	0-3	R	0	R	0	R	0-1	R	7	S	0-1	R	6-7	S	2	R	2	R	21
K. Kingbird	5-7	MS	0-3	R	4-7	S	4-5	MS	5-6	MS	4-6	MS	1-3	R	4-6	MS	1-2	R	2	R	1-2	R	0-1	R	18
K. Sunbird	7	S	4-5	MS	6-8	S	7	S	7	S	6-7	S	7	S	6-7	S	7	S	7	S	7-8	S	7	S	1
K. Tai	0-3	R	0-1	R	0-1	R	3-4	MR	3	R	1-3	R	3	R	2	R	2-3	R	2	R	2	R	2-3	R	19
Robin	7	S	4-6	MS	5-6	MS	5-7	MS	4-6	MS	4-7	MS	7	S	5-6	MS	5-7	MS	6-7	S	7	S	7	S	1

a and b are infection type (ITs) and the reaction of genotype to the stripe rust isolates. R (resistant); MR (moderately resistant); MS (moderately susceptible); S (susceptible). ITs are given according to a scale of 0-9 [25]; The resistance response spectrum was according to Hovmöller [16].

Spectrum 5 (*Yr6*, +) was comprised of differential sets “Heines Kolben” and “Avocet *Yr6*” with ITs of 6–7 for all the compatible reactions. Spectrum 6 (*Yr7*) was comprised of differential “Lee” with ITs of 2–3 and 6–7 for incompatible and compatible reactions, respectively. Spectrum 7 (*Yr8*) was comprised of differential “Avocet *Yr8*” with ITs of 6–7 for compatible and 0–3 for incompatible reactions. Spectrum 8 (*Yr9*, *AvS*) consisted of differential “Avocet *Yr9*” that had ITs of 0–1 and 6–7. Spectrum 9 (*Yr10*) consisted of differential “Moro” that had ITs of 0–2 and 6–7 for the incompatible and compatible reactions, respectively. Spectrum 10 (*Yr15*) was comprised of differential “Coatez,” which was resistant to all the isolates with ITs of 0–3. Spectrum 11 (*Yr17*, +) was comprised of differentials “VPM1” and “Avocet *Yr17*” ITs of 6–7 and 0–3 in response to isolate *KE86102Yr9*, which indicates an additional gene that conferred resistant to isolate *KE86102Yr9* in “VPM1.” Spectrum 12 (*Yr25*, +) consisted of differential “TP981” and genotype “1190059” with ITs of 6–7 for a compatible reaction; however, “1190059” had ITs of 0–3 for isolates *DK09/11SP*, *KE70063*, and *UK94/519* that were virulent on “TP981” ITs of 6–7, which was an indication of an additional gene in “1190059.” Spectrum 13 (*Yr27*, +) consisted of differential “Opata,” which was resistant to 10 out of 12 isolates with ITs of 0–4 for incompatible reactions and 6–8 for compatible reactions. Spectrum 14 (*Yr32*, 25, +) consisted of differentials “Carstens V” with ITs of 6–7, which was susceptible to three Danish isolates (*DK09/11SP*, *DK71/93*, and *DK02d/12*), which was all due to the warrior race that has virulence for *Yr1*, *Yr2*, *Yr3*, *Yr4*, *Yr6*, *Yr7*, *Yr9*, *Yr17*, *Yr25*, *Yr32*, *YrSP*, *YrAvS*, and *YrAmb*. Spectrum 15 (*AvS*) was comprised of differential “AvocetS,” which was susceptible to all the isolates except isolates *KE70063* and *SE100/09* with ITs of 0–4 and 6–7. Spectrum 16 (*Amb*, *Sp*, *AvS*) was comprised of differentials “Ambition” and “Avocet SP,” which were resistant to all the isolates except *DK09/11SP* and *DK02d/12*, respectively. Spectrum 17 (*Yr1*, *Yr3*, +) was comprised of differentials “Chinese166” and “Vilmorin” with ITs of 0, 0–4, and 6–7, which is an indication of three or more resistance genes being present. Spectrum 18 (*Yr7*, *Yr8*, +) was comprised of “Lee,” “Avocet *Yr8*,” “Duma,” and “K. Kingbird” (Table 3; Table 4). “Duma” had a similar reaction to “Lee,” except that it was resistant to isolates *KE82107* and *SE100/09*. Genotype “K. Kingbird” was resistant to *ER02/13*; however, this isolate was virulent on “Lee.”

Spectrum 19 (*Yr15*, *Yr27*, +) was comprised of differentials “Coatez,” “Opata,” and “K. Tai.” “Coatez” (ITs 0–3) was resistant to all twelve isolates tested, while “Opata” (ITs 0–4) was resistant to 10 out of 12 isolates and “K. Tai” with intermediate reactions (ITs 0–3, 3–4) could have an additional gene to *Yr15* (Tables 3 and 4). Spectrum 20 (unknown) was comprised of genotype “1190826” that was susceptible to all isolates except *SE100/09*. Spectrum 21 (unknown) consisted of genotype “1190034” with ITs of 0–3, 5–7, and 6–7 that was susceptible to two isolates *UK94/514* and *ER02/13* (Table 4). This genotype could be carrying three genes, among which, one is an APR gene due to an intermediate reaction. Spectrum 22 (unknown) consisted of genotype “1190713” and “1190524” with ITs of 0–3, 5–7, and 6–7, which could have been carrying two to three unknown genes. Spectrum 23 (unknown) consisted of genotypes “1190524” with ITs of 0–2, 3–5, and 6–7. The intermediate ITs of 3–5 and 5–7 could have been due to APR genes. Spectrum 24 (unknown) consisted of “K. Korongo” with ITs of 0–3 and intermediate reactions (4–5, 4–6), which is an indication of APR genes.

Wheat genotypes “K. Korongo” and “K. Wren” have unknown APR gene(s) as the disease reaction and infection type were moderately resistant (Table 5). Genotypes postulated to have *Yr1*, *Yr7*, *Yr9* (“K. Ibis,” “Kwale,” “NJBW2,” “Duma,” “K. Kingbird,” and “K. Tai”) exhibited low disease reactions in the field at Njoro and Eldoret ranging from 0–20%, while the genotypes with *Yr2* (“1190120,” “K. Hawk12,” “Eagle10,” “K. Sunbird,” and “Robin”) had disease severity ranging from 10–50%. Genotype “K. Korongo” had a combination of unknown all stage resistance (ASR) and adult plant resistance (APR) due to moderately resistant to moderately susceptible (M) reactions observed in the field experiments (Table 5).

Table 5. Resistance response spectrum, postulated stripe rust (Yr) genes, and field resistance of twenty wheat genotypes to *Puccinia striiformis* across three environments.

Genotype	Pedigree/Accession name	Resistance Spectrum	Postulated Yr Genes	Yellow Rust Field Resistance of the Genotypes in Three Locations		
				Eldoret	Njoro	Mau Narok
1190713	Watkins	22	Unknown	40MSS-50S	15MS-30S	50S
1190026	Watkins	15	Unknown	50MSS	20-35MSS	65-80S
1190034	Watkins	21	Unknown	0-10MSS	30-60S	5-25M
1190826	Watkins	20	Unknown	0-50MS	30MSS	5MS-70S
1190120	Watkins	1	None/Yr2	10-50S	30MSS-60S	30MSS-75S
1190524	Watkins	22	Unknown	50S	25MSS-30S	25MS-35S
1190527	Watkins	23	Unknown	50S	15-20MSS	5-20M
1190059	Watkins	12	Unknown	5S	10MS-20MSS	15MS-50S
K. Korongo	Emb16/CBRD//CBRD	24	Unknown	5-10M	0	5-20MR
Kwale	Kinglet,Cm33089-W	25	Yr9, +	TMS-5M	15-20MSS	45-55S
K. Wren	THELIN#2/TURUKU	24	Unknown	10MR	0-10MS	30-40S
Duma	R674-AV/UP301//GLL/SX/3/PEW 'S'/4/MAI "S"//PEW 'S'-CM67245	18	Yr7, +	5-10MR	5-15MS	35MS-60S
NJBW2	CM8181-12y-06PZ-4y-5m-0y-2AL-0AL-0M	25	Yr9, +	TMR-20M	5-20MSS	45-60S
K. Hawk12	Equator-II/kenya-Ploughman	1	None/Yr2	TR-5MR	5-10MS	10-20MS
Eagle10	Babax/Lr42//Babax*2/4/SN1/TRAP#1/3/KAUZ*2/TR	1	None/Yr2	40-50S	10-15MS	5R
K. Ibis	Kwale/Duma	21	Yr1,+	TMR-15M	0-10MS	10-30MS
K. Kingbird	TAM200/TUI/6/PVN/CAR422/ANA/5/BOW/CROW	18	Yr7,+	0-10M	5-10MS	25-30MS
K. Sunbird	ND643/2*WBLL1	1	None/Yr2	10MR	0-15MS	15-20M
K. Tai	ND643/2*WBLL1	19	Yr15,+	TMR-5MR	0	20MS-50S
Robin	Babax/Lr42//Babax*2/3/Turuku	1	None/Yr2	15-20MS	0-10S	35S

Gene postulation was based on the resistance spectrum from the seedling results in Tables 3 and 4. Resistance genes of the genotypes were postulated by comparing resistance spectra of the genotype to twelve races with those of wheat genotypes possessing known Yr resistance genes (differential lines) [9]. R (resistant); MR (moderately resistant); MS (moderately susceptible); M (moderately resistant to moderately susceptible); S (susceptible). Disease severity was based on a modified Cobb scale [28].

4. Discussion

4.1. Confirmation of the Virulence Phenotype of the Twelve Yr Isolates and Response of the Differential Lines to the Isolates

The *Pst* isolates used had virulence for *Yr1*, *Yr2*, *Yr3*, *Yr4*, *Yr6*, *Yr7*, *Yr8*, *Yr9*, *Yr10*, *Yr17*, *Yr25*, *Yr27*, *Yr32*, *YrAmb*, and *YrAvS*, but were avirulent to *Yr15* and *YrSp*. There was virulence for *Yr1*, *Yr2*, *Yr6*, *Yr7*, *Yr8*, *Yr9*, *Yr17*, and *YrAvS* on *Pst* isolated collected before the year 2000 and after the year 2000 in Kenya (unpublished data). Virulence for *Yr17* has not been common in Kenya; however, most of the European pathotypes were virulent on *Yr17*; this could be due to the presence of *Pst1* and *Pst2* strains, which are also present in East Africa [29]. The varieties deployed in these countries, such as “Bill,” “Biscay,” and “Bandit” from Denmark and “Equinox” from England have been postulated to have *Yr17* [16].

The difference in virulence for pre-2000 and post-2000 Danish isolates is due to the evolution of the new races, such as the “warrior” race, that was detected in Europe in 2011 infecting many wheat varieties and triticale (*Triticum secale*), where the isolate *DK09/11SP* used in this study belongs to the warrior race, which is virulent to *Yr2*, *Yr6*, *Yr7*, *Yr8*, and *Yr10* [21]. From the results, we can group the isolates’ pathotypes into two different groups according to the regions of East Africa (Eritrea and Kenya) and Europe (Denmark, Sweden, and U.K.), which brings out the geographical distribution of *Pst*, and the Middle East and East African population were grouped together as Middle East-East African as they did not differ much from each other [29]. *Pst1* and *Pst2* were present in East Africa (Kenya) and the same strains were present in Europe in addition to the “warrior” race. This brings out the evolution and similarity in *Pst* strains, which has been observed in Europe (Denmark) and East Africa (Kenya and Ethiopia), both from different continents with common virulences for *Yr2*, *Yr6*, *Yr7*, and *Yr8* [21,30,31]. Wheat genotypes with resistant genes *Yr2*, *Yr6*, and *Yr7* have been extensively used in Kenya and the same virulence to these genes were also found in isolates obtained from Hungary and Canada [29]. The pathotypes from different countries shared common virulence but differed in the degree of their virulence due to differences in the aggressiveness of the races against the deployed resistant genes in the regions [32].

Gene pyramiding is effective in conditioning resistance as “CarstensV” with resistant genes (*Yr32*, *Yr25*) was resistant to more isolates than “TP981” (*Yr25*). The difference in resistance of *Yr17* in “VpM1” and in “AvocetS” was the same as the resistance to *Yr6* in the “Heines Kolben” and “AvocetS” background, which brings out the interaction of genes in different background conditioning differences in gene expression and resistance.

Virulence to *Yr5* and *Yr15* were not detected in this study. This is because *Yr5* and *Yr15* are resistant to a wide range of isolates of yellow rust from different geographical regions [26]. Therefore, these genes, in combination with other race non-specific genes, can be deployed in wheat breeding programs to condition resistance [27,30].

4.2. Wheat Genotypes and Resistance Responses to Stripe Rust Isolates Representing Distinct Races

The genotypes with a broad resistance spectrum possessed resistant genes, such as *Yr4*, *Yr10*, *Yr15*, *Yr32*, *YrAmb*, and *YrSp*, which are still effective. Among the *Yr* genes that could be present in the genotypes under study, *Yr17* is still effective in Kenya. Several researchers have employed a gene postulation for identifying *Yr* genes in wheat genotypes. For instance, Sharma [17] reported *Yr* genes *Yr2*, *Yr6*, *Yr8*, *Yr9*, *Yr10*, *Yr15*, *YrA*, and *YrSu* in 52 advanced emmer wheat lines. Hovmöller [9] postulated *Yr1*, *Yr2*, *Yr3*, *Yr4*, *Yr6*, *Yr9*, *Yr15*, *Yr17*, *Yr25*, and *Yr32* in 98 Danish wheat cultivars. In China, they have postulated several genes, such as *Yr1*, *Yr2*, *Yr3*, *Yr4*, *Yr6*, *Yr7*, *Yr8*, *Yr9*, *Yr1*, *Yr20*, *Yr21*, *Yr22*, *Yr23*, *Yr24*, *Yr26*, *Yr27*, *Yr32*, *YrHV*, *YrSD*, *YrV23*, *YrRes*, *YrC591*, *YrClein*, *YrSel*, and *YrSdin* at the seedling level in their wheat varieties [20,33].

Based on the multi-pathotype tests conducted, the wheat lines could have had different stripe rust resistance genes, with some similar to the tester lines and others being unknown. Yellow rust genes

Yr2, *Yr7*, *Yr9*, and *Yr15* were postulated in the Kenyan line tested. “K. Korongo” and four Watkins lines (“1190713,” “1190026,” “1190826,” “11900034,” and “1190524”) did not match any spectra of the testers, indicating the presence of unknown resistance genes in these lines.

Stripe rust *Yr1* gene was not postulated in the wheat lines tested in this study; however, the same gene was postulated in Chinese landraces “Zhengmai366,” “Xinmai208,” and “Luomai21”, and in cultivar “Buster” [9]. Gene *Yr2* was postulated in several studies in Nepal, Europe, and China [17,27,33]. The stripe rust gene (*Yr2*) is no longer effective against the *Yr* pathotypes tested and it might be present in wheat varieties “1190120,” “K. Wren,” “K. Hawk12,” “K. Sunbird,” and “Robin,” which explains why these varieties were susceptible to most of the pathotypes used. This gene (*Yr2*) found in the differential line “Kalyansona” is no longer effective; therefore, the lines with a single *Yr2* need to be improved by incorporating resistant genes such as *Yr15*, *YrSP*, *Yr4*, *Yr10*, and *YrAmb* to attain durable resistant.

Gene *Yr6* was postulated in Australia wheat lines categorized under group six (*YRG6*) [28] and in wheat cultivars “Hunter,” “Lynx,” and “Rialto” [9]. In our study, no genotype was postulated to have *Yr6*. Gene *Yr7* could be present in “Duma” and “K. Kingbird,” where this gene is linked to *Sr9g* in cultivar “Cadenza” and “Tonic” [34]. Gene *Yr9* postulated in “Kwale” and “NJBW2” is pleiotropic or linked to *Sr31/Lr26*. However, virulence to *Sr31* has been present in Kenya since the emergence of *Sr31* virulence in Uganda [9]; therefore, the gene is ineffective and cannot be deployed in Kenyan wheat lines unless in combination with other resistant genes. Genotype “K. Tai” was postulated to have *Yr15* plus other unknown genes. *Yr15* is a *T.-dicoccoides*-derived stripe rust resistant gene with all stage resistance (ASR) located on chromosome 1BS [35] and was found in 25 wheat lines from Iran [20]. The genotypes that had an unknown gene postulated include all the Watkins lines except “1190120,” exhibiting more than 50S in either one of the locations tested, which is an indication of the lack of effective genes against the pathotypes present in that environment.

The effectiveness of stripe rust genes against the new *Pst* pathotypes is not clear and the genetic background plays a big role in this; hence, it is important to have a diverse germplasm for testing [36–38]. *Yr15* and *Yr65* are among the genes that are still effective, while *Yr9* and *Yr10* are among the ineffective genes [39].

5. Conclusions

Evaluation of Kenyan and Watkins lines with twelve races from Kenya, Eritrea, Denmark, Sweden, and U.K. indicated that race *DK02d/12* from Denmark was more virulent on the genotypes than any other race except genotype “K. Tai,” which was resistant to these races and others; hence it is useful for breeding wheat cultivars with resistance to stripe rust. The twenty wheat genotypes that exhibited similar infection type responses as the tester lines could have had the same genes as the tester lines, but this needs further research to confirm the genes present.

Author Contributions: Conceptualization, M.N.W.; investigation, M.N.W.; methodology, M.N.W. and R.W.; resources, R.W.; supervision, J.O. (James Owuoche) and J.O. (Joshua Oendo); writing—original draft, M.N.W.; writing—review and editing, J.O. (James Owuoche), J.O. (Joshua Oendo), and R.W.

Funding: This study was supported by a Sustainable Crop Production Research for International (SCPRID) award (BB/J012017/1) funded by the Biotechnology and Biological Sciences Research Council (BBSRC) and The Global Rust Research Centre (GRRC), Denmark.

Acknowledgments: We are grateful to Global Rust Research Centre (GRRC), Denmark for providing the facilities and resources to carry out this study. Acknowledgements go to Hovmøller, J. Rodriguez-Algaba, Janne H. Hansen, Ellen Jorgenses, and Steen Meier. We are indebted to KALRO Njoro and the following members for providing some of the samples used in this study: Hannington Wanga, Phelister Kinyanjui, Eric Githinji, Festus Muchika, and Leonard Wolukao. We also wish to acknowledge the John Innes Centre (JIC) for the Watkins collections used in this study.

Conflicts of Interest: The authors declare no conflict of interest.

References

1. FAO. *Current World Fertilizer Trends and Outlook to 2016*; Food and Agriculture Organization of the United Nations (FAO): Rome, Italy, 2012.
2. Daniel, D.L. *Aspects of Durable Resistance in Wheat in Yellow Rust*; Wageningen Agricultural University: Wageningen, The Netherlands, 1994; 144p, ISBN 90-5485-236-4.
3. Thorpe, H.C. Wheat breeding in Kenya. In Proceedings of the First International Wheat Genetics Symposium, Winnipeg, MB, Canada, 11–15 August 1956.
4. Kurt, J.L. Black Stem Rust Biology and Threat to Wheat Growers. USDA, ARS Cereal Disease Laboratory, 2001. Available online: <http://www.cdl.umn.edu> (accessed on 25 June 2019).
5. Wanyera, R. The status of rust diseases of wheat in Kenya. In *Breeding for Disease Resistance with Emphasis on Durability, Proceedings of the a Regional Workshop for Eastern, Central and Southern Africa, Njoro, Kenya, 2–6 October 1994*; Daniel, D.L., Ed.; CIMMYT: Mexico City, Mexico, 1994; pp. 222–225.
6. Wellings, C.R. Global status of stripe rust: A review of historical and current threats. *Euphytica* **2011**, *179*, 129–141. [[CrossRef](#)]
7. Wan, A.M.; Chen, X.M.; Yuen, J. Races of *Puccinia striiformis* f. sp. *tritici* in the United States in 2011 and 2012 and comparison with races in 2010. *Plant Dis.* **2016**, *100*, 966–975. [[CrossRef](#)] [[PubMed](#)]
8. Hovmöller, M.S.; Rodriguez-Algaba, J.; Thach, T.; Sørensen, C.K. Race typing of *Puccinia striiformis* on wheat. In *Wheat Rust Diseases. Methods in Molecular Biology*; Periyannan, S., Ed.; Humana Press: New York, NY, USA, 2017; Volume 1659. [[CrossRef](#)]
9. Pretorius, Z.A.; Singh, R.P.; Wagoire, W.W.; Payne, T.S. Detection of virulence to wheat stem rust resistance gene *Sr31* in *Puccinia graminis* f. sp. *tritici* in Uganda. *Plant Dis.* **2000**, *84*, 203. [[CrossRef](#)] [[PubMed](#)]
10. Kisana, S.N.; Mujahid, Y.M.; Mustafa, Z.S. *Wheat Production and Productivity 2002–2003. A Technical Report to Appraise the Issues and Future Strategies*; Coordinated Wheat, Barley and Triticale Programme, National Agriculture Research Centre, Pakistan Agriculture Research Council: Islamabad, Pakistan, 2003.
11. Saari, E.E.; Prescott, J.M. World distribution in relation to economic losses. In *The Cereal Rusts Volume II, Diseases, Distribution, Epidemiology and Control*; Roelfs, A.P., Bushnell, W.R., Eds.; Academic Press: Orlando, FL, USA, 1985; pp. 259–298.
12. Dawit, W.K.; Flath, W.E.; Weber, E.; Schumann, M.S.; Röderand, S.; Chen, X. Postulation and mapping of seedling stripe rust resistance genes in Ethiopian bread wheat cultivars. *J. Plant Pathol.* **2012**, *94*, 403–409. [[CrossRef](#)]
13. Kang, Z.S.; Zhao, J.; Han, D.J.; Zhang, H.C.; Wang, X.J.; Wang, C.F.; Han, Q.M.; Guo, J.; Huang, L.L. Status of wheat rust research and control in China. In Proceedings of the BGRI 2010 Technical Workshop, St Petersburg, Russia, 30–31 May 2010.
14. Flor, H.H. The complementary gene system in flax and flax rust. *Adv. Genet.* **1956**, *8*, 29–54. [[CrossRef](#)]
15. Flor, H.H. Current status of the gene-for-gene concept. *Ann. Rev. Phytopathol.* **1971**, *9*, 275–296. [[CrossRef](#)]
16. Hovmöller, M.S. Sources of seedling and adult plant resistance to *Puccinia striiformis* f. sp. *tritici* in European wheats. *Plant Breed.* **2007**, *123*, 225–233. [[CrossRef](#)]
17. Sharma, S.; Louwers, J.M.; Karki, C.B.; Snijders, C.H.A. Postulation of resistance genes to yellow rust in wild emmer wheat derivatives and advanced wheat lines from Nepal. *Euphytica* **1995**, *81*, 271–277. [[CrossRef](#)]
18. Xia, X.C.; Li, Z.F.; Li, G.Q.; Singh, R.P. Stripe rust resistance in Chinese bread wheat cultivars and lines. In *Wheat Production in Stressed Environments*; Buck, H.T., Nisi, J.E., Salomon, N., Eds.; Springer: Amsterdam, The Netherlands, 2007; Volume 17, pp. 77–82. [[CrossRef](#)]
19. Singh, D.; Park, R.F.; McIntosh, R.A. Postulation of leaf (brown) rust resistance genes in 70 wheat cultivars grown in the United Kingdom. *Euphytica* **2001**, *120*, 205–218. [[CrossRef](#)]
20. Xiaodan, X.; Jing, F.; Ruiming, L.; Hussain, K.; Shichang, X.; Feng, L. Postulation of stripe rust resistance genes in 44 Chinese wheat cultivars. *Int. J. Agric. Biol.* **2011**, *13*, 665–670.
21. Hovmöller, M.S.; Walter, S.; Bayles, R.A.; Hubbard, A.; Flath, K.; Sommerfeldt, N.; Leconte, M.; Czembor, P.; Rodriguez-Algaba, J.; Thach, T.; et al. Replacement of the European wheat yellow rust population by new races from the centre of diversity in the near—Himalayan region. *Plant Pathol.* **2016**, *65*, 402–411. [[CrossRef](#)]
22. Sørensen, C.K.; Thach, T.; Hovmöller, M.S. Evaluation of spray and point inoculation methods for the phenotyping of *Puccinia striiformis* on wheat. *Plant Dis.* **2016**, *100*, 1064–1070. [[CrossRef](#)] [[PubMed](#)]
23. Large, E.C. Growth stages in cereals illustration of feekes scale. *Plant Pathol.* **1954**, *3*, 128–129. [[CrossRef](#)]

24. Thach, T.; Ali, S.; De Vallavieille-Pope, C.; Justesen, A.F.; Hovmøller, M.S. Worldwide population structure of the wheat rust fungus *Puccinia striiformis* in the past. *Fungal Genet. Biol.* **2016**, *87*, 1–8. [[CrossRef](#)]
25. McNeal, F.H.; Konzak, C.F.; Smith, E.P.; Tate, W.S.; Russell, T.S. *A Uniform System for Recording and Processing Cereal Research Data*; USDA-ARS Bull: Washington, DC, USA, 1971; pp. 34–121.
26. Dubin, H.J.; Stubb, R.W.; Johnson, R. Postulated genes for resistance to stripe rust in selected CIMMYT and related wheat. *Plant Dis.* **1989**, *73*, 472–475. [[CrossRef](#)]
27. McIntosh, R.A.; Wellings, C.R.; Park, R.F. *Wheat Rusts: An Atlas of Resistance Genes*; CSIRO: East Melbourne, Victoria, Australia, 1995; p. 157.
28. Peterson, R.F.; Campbell, A.B.; Hannah, A.E. A diagrammatic scale for rust intensity on leaves and stems of cereals. *Can. J. Res.* **1948**, *26*, 496–500. [[CrossRef](#)]
29. Walter, S.; Ali, S.; Kemen, E.; Nazari, K.; Bahri, B.A.; Enjalbert, J.; Hansen, J.G.; Brown, J.K.M.; Sicheritz-Ponten, T.; Jones, J.; et al. Molecular markers for tracking the origin and worldwide distribution of invasive strains of *Puccinia striiformis*. *Ecol. Evol.* **2016**, *6*, 2790–2804. [[CrossRef](#)]
30. Hovmøller, M.S.; Yahyaoui, A.H.; Milus, E.A.; Justesen, A.F. Rapid global spread of two aggressive strains of a wheat rust fungus. *Mol. Ecol.* **2008**, *17*, 3818–3826. [[CrossRef](#)]
31. Milus, E.A.; Kristensen, K.; Hovmøller, M.S. Evidence for increased aggressiveness in a recent widespread strain of *Puccinia striiformis* f.sp. *tritici* causing stripe rust of wheat. *Phytopathology* **2009**, *99*, 89–94. [[CrossRef](#)]
32. Sharma-Poudyal, D.; Chen, X.M.; Wan, A.M.; Zhan, G.M.; Kang, Z.S.; Cao, S.Q.; Jin, S.L.; Morgounov, A.; Akin, B.; Mert, Z.; et al. Virulence characterization of international collections of the wheat stripe rust pathogen, *Puccinia striiformis* f. sp. *tritici*. *Plant Dis.* **2013**, *97*, 379–386. [[CrossRef](#)] [[PubMed](#)]
33. Tian, Y.; Zhan, G.; Chen, X.; Tungruentragoon, A.; Lu, X.; Zhao, J.; Huang, L.; Kang, Z. Virulence and simple sequence repeat marker segregation in a *Puccinia striiformis* f. sp. *tritici* population produced by selfing a Chinese isolate on *Berberis shensiana*. *Phytopathology* **2016**, *106*, 185–191. [[CrossRef](#)] [[PubMed](#)]
34. Singh, D.; Park, R.F.; McIntosh, R.A.; Bariana, H.S. Characterization of stem rust and stripe rust seedling resistance genes in selected wheat cultivars from the United Kingdom. *Plant Pathol.* **2008**, *90*, 553–562.
35. Sun, G.L.T.; Fahima, A.B.; Korol, T.; Turpeinen, A.; Grama, Y.; Ronin, I.; Nevo, E. Identification of molecular markers linked to the *Yr15* stripe rust resistance gene of wheat originated in wild emmer wheat *Triticum dicoccoides*. *Theor. Appl. Genet.* **1997**, *95*, 622–628. [[CrossRef](#)]
36. Ellis, J.G.; Lagudah, E.S.; Spielmeier, W.; Dodds, P.N. The past, present and future of breeding rust resistant wheat. *Front. Plant Sci.* **2014**, *5*, 641. [[CrossRef](#)]
37. Mukhtar, S.; Khan, M.A.; Paddar, B.A.; Anjum, A.; Zaffar, G.; Mir, S.A.; Naseer, S.; Bhat, M.; Kamaluddin, A. Molecular characterization of wheat germplasm for stripe rust resistance genes (*Yr5*, *Yr10*, *Yr15* & *Yr18*) and identification of candidate lines for stripe rust breeding in Kashmir. *Indian J. Biotechnol.* **2015**, *14*, 241–248.
38. Rahmatov, M.; Otambekova, M.; Muminjanov, H.; Rouse, M.N.; Hovmøller, M.S.; Nazari, K.; Steffenson, B.J.; Johansson, E. Characterization of stem, stripe and leaf rust resistance in Tajik bread wheat accessions. *Euphytica* **2019**, *215*, 55. [[CrossRef](#)]
39. Pretorius, Z.A.; Ayliffe, M.; Bowden, R.L.; Boyd, L.A.; DePauw, R.M.; Jin, Y.; Knox, R.E.; McIntosh, R.A.; Park, R.F.; Prins, R.; et al. Advances in Control of Wheat Rusts. In *Achieving Sustainable Cultivation of Wheat, Volume 1: Breeding, Quality Traits, Pests and Diseases*; Langridge, P., Ed.; Burleigh Dodds Science Publishing: Cambridge, UK, 2017; pp. 295–343.

