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POSTULATION OF STRIPE RUST RESISTANT GENES IN SOME AUSTRALIAN BREAD WHEAT CULTIVARS AND THEIR RESPONSE TO TEMPERATURE

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Abstract

Stripe rust caused by *Puccinia striiformis* f. sp. *tritici* (*Pst*) is an important constraint to wheat production in cool environments. Six pathotypes (*PST*) were used to identify resistance genes in 27 Australian spring wheat (*Triticum aestivum*) cultivars. Postulation of resistance at seedling growth stage showed that most cultivars carried *Yr17* alone or in combination with *Yr7* and or other undesignated resistance genes. The cultivar H45 carried *Yr7* Molecular markers were used to confirm the presence of stripe rust resistant gene *Yr17* in these cultivars. The 252-bp PCR product from primer VENTRIUP was observed in all Australian spring wheat cultivars tested and in positive check Avocet + *Yr17* NIL. The molecular marker for *Yr17* verified the results from rust testing. The effect of temperature on expression of resistance conferred by *Yr17* was also confirmed. Seedlings that possess *Yr17* expressed high levels of resistance under higher temperature (15-20ºC). Under lower temperature (12-15ºC) most cultivars possessing *Yr17* showed compatible or near-compatible reactions to the pathotype 134 E16 A^+ (avirulent to *Yr17*).

Introduction

Plant diseases are among the principal factors affecting the yield of wheat crops. The rust diseases of wheat have historically been one of the major biotic production constraints both in Asia and the rest of the world. There are more than 3000 rust species in the world (Laudon, 1973), three of which are pathogenic on wheat: *Puccinia graminis* f. sp. *tritici* (causal agent of stem rust), *P. striiformis* f. sp. *tritici* (causal agent of stripe rust) and *P. triticina* (causal agent of leaf rust). Stripe rust is principally an important disease of wheat during winter or early spring or at higher elevations (Roelfs *et al*., 1992). In most wheat producing areas, yield losses caused by stripe rust range from 10-70% (Chen, 2005).

Many control measures have been applied to minimize the losses incurred by stripe rust but use of resistant cultivars is found to be the most economical, efficient in environment and farmer friendly strategy (Ittu, 2000). Before the foundation of modern genetics by Mendel, clever farmers and early plant breeders empirically selected resistant plants. Systematic breeding for disease resistance started after the discovery of genetics of resistance (Biffen, 1905), pathogen specialisation (Stakman & Levine, 1944) and gene-for-gene resistance (Flor, 1956). At least 33 *Yr*-genes that confer resistance to yellow rust in wheat have been identified and incorporated into commercial wheat varieties (Mclntosh *et al*., 1995, Pathan *et al*., 2007)). Many of them are race-specific genes showing hypersensitive reaction and several are currently being used by breeders to

develop new cultivars. However, the resistance provided by these genes can be short-lived as new races of the pathogen, *Puccinia striiformis* f. sp. *tritici*, are continuously evolving and acquiring virulence to these genes (Stubbs, 1985). In addition, expression of seedling resistance conferred by some of these genes is claimed to be affected by environment (Wellings *et al*., 1988). A thorough understanding of the influence of environment on each yellow rust resistance gene will help to incorporate the desired genes into a cultivar for a particular agro-ecological zone.

Resistance gene postulation is a rapid means by which the resistance genes present in a host genotype can be hypothesized (Loegering *et al*., 1971). It is based on gene-for-gene specificity between host resistance genes and different avirulence genes. A well-characterized collection of pathogen pathotypes with different avirulence gene combination is used to postulate the resistance genes in host genotypes. However, interaction between resistance genes can obscure the gene postulation and this method is best suited for seedling resistance genes (Kolmer, 1996). Problems like gene interaction and plant growth stage at which genes are expressed can be overcome by using DNA-based markers to identify resistance genes (McCartney *et al*., 2005). The study was aimed to identify genes conferring seedling resistance in some Australian commercial wheat cultivars and their response to temperature at the seedling growth stage.

Materials and Methods

Multipathotype tests of wheat genotypes were conducted in Plant Breeding Institute, Cereal Rust Research Laboratory, Cobbitty University of Sydney, Australia in 2007 to postulate the presence of any seedling resistance rust genes and their expression to different temperature regime. The research consists of some greenhouse experiments at the seedling growth stage and use of molecular markers.

Host genotypes: Presence of genes for stripe rust resistance and their response to different temperature regime was assessed in 27 Australian bread wheat cultivars (Tables 5 & 6). Australian differential set of wheat genotypes for stripe rust (McIntosh *et al*., 1995) was also included as controls in the test.

Pathotypes: Well characterized pathotypes of *P. striiformis* f. sp. *tritici* used in the study, alongwith their virulence, are given in Table 1. These pathotypes were obtained from the Plant Breeding Institute Cereal Rust Collection.

Molecular marker: Molecular marker VENTRIUP-N2 (Table 2) was applied for identification of *Yr17* adopting the protocol of Helguera *et al*., (2003) in 20 Australian bread wheat cultivars, selected from seedling test (Fig. 1). The near isogenic line (NIL) Avocet *+Yr17* was used as positive check for *Yr17*.

Seedling tests: At least 6-10 seedlings were raised in 9cm diameter plastic pots in a coarse potting mix comprising composted pine bark and coarse sand in a ratio of 4:1. The potting mix was firmly pressed into pots and fertilized with a complete fertilizer (25 g Aquasol® $10L^{-1}$water for 200 pots. After fertilization, seeds were sown and covered with additional potting mix. Seedlings were inoculated with each rust pathotype at the two leaf growth stage. Rust urediniospores suspended in a light mineral oil (Pegasol®, 10 mg urediniospores 10 mL^{-1} oil for 200 pots) were atomised over seedlings with the help of a hydrocarbon propellant pressure pack. To avoid contamination, the spray equipment was

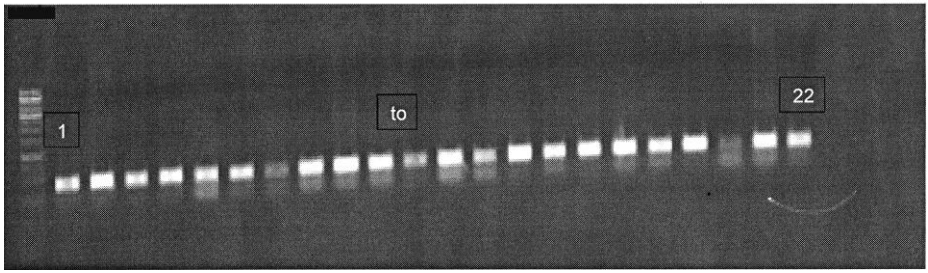

Fig. 1. PCR amplification of DNA extracted from Australian spring wheat cultivars1) Rudd, 2) Camm, 3) Ventura 4) EGA Eaglehawk, 5) Sunstate, 6) EGA Jaeger, 7) Sunzell, 9) Sunbri, 10) Sunlin, 11) Stylet, 12) Trident, 13) Bowie, 14) Qualges, 15) QAL 2000, 16) Gladius, 17) Derrimut, 18) Binnu, 19) Barham, 20) WA2713 21) Yenda, and 22) *Yr17* NIL with specific marker VENTRIUP. The bands in figure represent presence of *Yr17*.

washed in alcohol and rinsed in running tap water between successive inoculations. Inoculated seedlings were incubated for 24 hours on trolleys with in-built water baths, covered with polythene hoods, at 10-12°C in a dark temperature controlled room. Sufficient mist was generated under the hoods during incubation to allow infection. The seedlings were then transferred to temperature and irrigation-controlled growth rooms for disease assessments at the appropriate time. The temperature in these growth rooms was maintained within the range of 16-18ºC. After appearance of rust flecking on the leaves, the plants were fertilized with a nitrogenous fertilizer (Urea 25g $10L^{-1}$ water for 200 pots). Two weeks after inoculation, disease assessment was made using 0-4 infection type scoring system outlined by McIntosh *et al*., (1995). Infection types (IT) of 3 or higher were considered as compatible (high IT), whereas ITs lower than 3 were regarded as incompatible (low IT). The presence or absence of genes in test cultivars was postulated by correlating their responses to control differential genotypes against a given pathotype. A high IT on the test cultivar indicated that it did not have any of the resistance genes effective against the pathotype.

Molecular marker test: Leaf samples of seedling of each Australian genotype were collected and DNA was extracted from these bulked samples using a standard protocol. The samples were grinded in liquid nitrogen with a crushing machine, 800µl of warmed (65ºC) CTAB buffer was added to each tube and the resulting mixture was vortexed thoroughly to homogenize. Tubes were then incubated in a water bath for 40 minutes at 65ºC. Then the samples were left for five minutes at room temperature. In each tube 768µl chloroform and 32 µl phenol (24:1) was added and the solution was mixed gently by inverting tubes for two minutes. The samples were then centrifuged at 13,200 rpm for 15 minutes. The supernatant was taken and collected in new 1.5 µl Eppendorf tubes. Cold isopropanol (2/3rd volume of supernatant) was added to each tube and mixed gently. Samples were again centrifuged at 14,000 rpm for 10 minutes to pellet DNA. The supernatant was then discarded and the pellet dissolved in 500µl of washing buffer. The DNA pellets were dried at room temperature for 10 minutes. TE 100 µl (pH 8.0) and 1 µl of $10mg.ml^{-1}$ RNase was added to each tube and incubated for 1-2 hours.

PCR analysis: The polymerase chain reactions (PCR) were performed following the protocol of Helguera *et al*., (2003). The amplified DNA products were separated by electrophoresis on 2% (w/v) agarose gel and visualised under UV light after staining with Ethidium bromide. 1kb ladder (sigma) was loaded along with amplified DNA products on the gel.

Results and Discussion

Gene postulation by multipathotype test: Seedling responses of host genotypes, the Australian standard and supplementary differential set, alongwith the NILs for different *Yr* genes with well defined genotypes against 6 pathotypes of *P. striiformis* f. sp. *tritici* are presented in Table 3 (experiment 1) and 4 (experiment 2). The postulated resistance genes in 27 Australian cultivars against 6 pathotypes are shown in Tables 5 and 6. Cultivars Barham, Binnu, Bowie, Carinya, EGA Jaeger, Derrimut, Ellison, Gladius, QAL 2000, Stylet, Sunlin, Trident and Marombi displayed susceptibility (high IT) to the pathotype 134 E16 A^+ $Yr17^+$, whereas they showed low infection type (IT) with the remaining three pathotypes (Table 5). The result was reproduced and these cultivars showed again low IT for 104 E137 A^+ $Yr17^+$ and 134 E16 A^+ $Yr17^+$ in a second experiment with 6 pathotypes (Table 6). The result indicates that these genotypes carry *Yr17*. Bariana *et al*., (2003) also reported *Yr17* in Sunlin and Trident. They also claimed that these two cultivars also carried an additional adult plant gene for stripe rust. Qalbis showed a fully compatible reaction to pathotype 134 E16 A^+ $Yr17^+$ (Table 5) in the first experiment. In the second experiment, it expressed high compatibility to pathotypes 104 E137 A^+ $Yr17^+$ and 134 E16 A^+ $Yr17^+$ (Table 6). Its reaction for the remaining pathotypes was moderate. The result indicates that these cultivars also possess *Yr17*. The moderate IT of these cultivars might be due to low temperature because expression of *Yr17* is affected by temperature (Bariana & McIntosh, 1994). Braewood expressed moderate IT to pathotype 134 E16 A+ $Yr17^+$, whereas it was highly resistant to the remaining three pathotypes in both experiments (Tables 5 & 6). Sunbri and Sunvale showed lower IT for all pathotypes including 134 E16 A^+ $Yr17^+$ (Table 5), but in a repeated experiment it showed a moderate IT for pathotypes 104 E137 A^+ $Yr17^+$ and 134 E16 A^+ $Yr17^+$ whereas for remaining pathotypes they expressed incompatible reaction (Table 5 & 6). These findings suggest that the gene composition for these three cultivars is *Yr17* and Y*rCK* (Cook) because they were derived from a cross of wheat genotype Cook. Cook carries an unknown resistance *Yr* gene designated as *YrCK* (Personnel communication, Wellings). Due to the presence of *YrCK*, the pathotypes 134 E16 A^+ $Yr17^+$ and 104 E137 A^+ $Yr17^+$ could not express virulence to *Yr17*. According to Bariana *et al*., (2001) *YrCK* is a component of high level additive adult plant and durable stripe rust resistance in Australian wheat cultivar Cook, in addition to *Yr18*. The moderate IT of Sunvale might also be due to some adult plant resistance as Bariana *et al*., (2003) also reported adult plant resistance in Sunvale. In the first experiment, the cultivars Camm, Sunzell and Ventura gave high ITs with pathotype 134 E16 A^+ $Yr17^+$ whereas they showed low ITs to the other three pathotypes, suggesting *Yr17* in the cultivars (Table 5). A similar reaction of these genotypes was observed in a second experiment. Except with 134 E16 A^+ $Yr17^+$, these three cultivars were resistant to all pathotype tested (Table 6). The reaction to pathotype 134 E16 A+ was intermediate in these cultivars, suggesting the presence of *Yr17* and *Yr7*.

In the first experiment, EGA Eaglehawk was resistant to pathotypes 104 E137 A^+ and 134 E16 A^+ but the result was not available for pathotypes 108 E141 A+ and 134 E16 A^+ $Yr17^+$ (Table 5). In a second experiment, this cultivar was found to be highly resistant to all pathotypes tested except for pathotype 134 E16 A^+ $Yr17^+$. Although its IT was not fully compatible (Table 6) with pathotype 134 E16 A^+ $Yr17^+$, EGA Eaglehawk seemed to carry *Yr17* and possibly an unknown additional gene.

The cultivar Peak seemed to carry *Yr17* and possibly *Yr7*, as in both experiments it was susceptible to the pathotype 134 E16 A^+ $Yr17^+$ and it was not fully compatible to the pathotype 238 E143 A^+ carrying virulence to *Yr7*. It gave a low IT with pathotypes 104 E137 A^+ and 104 E137 A^+ $Yr17^+$, whereas it showed a mixed reaction with pathotype 108 E141 A^- (Tables 5 & 6).

Table 3. Response of different pathotypes of *P. striiformis* f. sp. *tritici* on Australian differential genotypes of wheat and NILs used in experiment 1.

International set	*Yr* Gene	108 E141 A$^-$	134 E16 A$^+$	134 E16 A$^+$	134 E16 A$^+$ Yr17$^+$	104 E137 A$^+$
Chinese 166	*Yr1*		0	0	0	0
Lee	*Yr7*	CN	3	3	3+C	0,(1P2)
Heines Kolben	*Yr6*	3+	3+	3	3+C	2+3
Vilmorin 27	*Yr3*	3+	1-C	2C	0	2CN(1P3C)
Moro	*Yr10*	0	0	0	0	0
Strubes Dickkopf		3+	1C	1+C	CN/1+C	23CN
Suwon 92/Omar		3+	0	0	3C	3+
Clement	*Yr9*+		2+C	2++3	CN	0;C(1P1)
T.spelta album	*Yr5*	0	0	0	0;C	0;C
European set						
Hybrid 46	*Yr4*	22+C	0	/1+	0;C	2+CN
Reichersberg 42	*Yr7*+	CN	2++C	2+C	2+3NC	CN(1P1C)
Heines Peko	*Yr6*+	3+	2+3	3	CN/2C	1CN(1P3C)
Nord Desprez		3+C	1-NC	1-	2+C	2+3CN
Compair	*Yr8*	0;C	3	3	2+CN	0;C
Carstens V	*Yr32*	0	0	0	0	0;C
Spaldings Prolific		CN(1P2+)	0	1-	0	0;C(2P2+3)
Heines VII	*Yr2,25*+	2+3	2+	2++3	23+C	2+CN
Supplementary set						
Avocet R	*YrA*	22+C	2+	3	3+C	3+C
Kalyansona	*Yr2*	3++	3+	3	3+C	2+3(1P;N)
Trident	*Yr17*	N1-		/2+	2+3C	0;NC
Yr15 NIL	*Yr15*	0	0	0	0	0
Hugenoot	*Yr25*	1+2	1++C	1-	2+CN	NC1-
Selkirk	*Yr27*	2C3	1++CN	1+C	0;(1P1CN)	NC
Fed4*/ Kavkaz	*Yr9*	0	3+	2++	3+C	2+
Federation		3+	3	3	3+C	3+C
Gregory	*Yr33*	2CN	1+2CN	2C	N(1P3C)	1CN,2+C
Gladius	*Yr17*	12-CN	2++3	2++3	2+3CN	NC,2+C
Binnu	*Yr17*		2+C	2+	3C	NC,(1P1+)
NILS						
Yr1 / 6* Avocet S		0	0	0	0	0
Yr5 / 6* Avocet S			0	0CN	0	0
Yr6 / 6* Avocet S		3+	3+	3	2+3C	2+CN
Yr7 / 6* Avocet S		1-	3+	3	2+3C	2+C
Yr8 / 6* Avocet S		(1P;2)	2+C	2+3C	;NC(1P1-)	0
Yr9 / 6* Avocet S		0	3	3	2+3C	2+C
Yr10 / 6* Avocet S		0	0	0	_	0
Yr15 / 6* Avocet S		0	0	0	0	0
Yr17 / 6* Avocet S		0	0	0	_	0
Yr18 / 6* Avocet S		3++	3+	3	CN1-1	23CN
Yr24 / 6* Avocet S		N	N	CN	0;CN	0;
Yr26 / 3* Avocet S		1+/;N	N1-	CN	CN	2+/;
Yr27 / 6* Avocet S		2C	N1-	CN	0;(1P1-)	0;(1P2+)
Yr32 / 6* Avocet S		NC	NC	CN	0;(1P1-)	0
YrSp / 6* Avocet S			0	0	0	1+/0
Jupateco R		3+	3	3	2CN	2
Jupateco s		3	3	3	3+C	3+C
Avocet R		3	3+	3+	3+C	3+C
Avocet S		3++	3	3+	3+C	3C

Table 7. Effect of temperature on expression of Yr resistance genes.

Cultivar	134 E16 A$^+$ (12-15ºC)	134 E16 A$^+$ (15-20ºC)	134 E16 A$^+$ Yr17$^+$ (12-15ºC)	134 E16 A$^+$ Yr17$^+$ (15-20ºC)
Barham	3+	CN(1P1=)	3+	3
Binnu	23C	CN1=	3+	3CN
Bowie	3	CN	3+	3
Braewood	;N	CN	3+	3CN
Camm	3+	3+CN	3+	3+
Carinya	3CN	CN	3+	3+
Derrimut	3	1-CN(1P2-CN)	3+	3+
EGA Eaglehawk	12-CN	CN	3C	3+
EGA Jaeger	2+3CN	CN(1P2-)	3+	3CN
Ellison	3+(1P 1-CN)	1CN2-	3+	3C
Gladius	3+	1=CN	3+	3+
H46	3+	3+	3+	3+
QAL 2000	3+	CN	3+	3C
Rudd	0	0	0(1P3)	0 ES
Stylet	3	CN1	3+	3C
Sunbri	1-(2P23CN)	CN	3	23CN
Sunlin	CN(11+CN)	CN	3	3C
Sunvale	3(2P2+CN)	(few week p)	3	22+CN
Sunzell	23CN	N1=N	3	3+
Trident	2CN,3	CN	3+	23CN
Sunland	2+3N	12+CN	2+3N	23CN
Ventura	2+3CN	1-1(1P2-CN)	3	3+
Yenda	3+	2+3CN	3+	3+
Young	3+	0(1P1+C)	3	3
Qalbis	3+	22+C	3+	3+
Peake	3+	;CN1(1P3)	3+	3+
Marombi	23CN	0;N	3+	3

The cultivar Sunland showed moderate susceptibility to only one pathotype, 134 E16 A+ Yr17$^+$ and its IT was not fully compatible. So, the resistance gene(s) in this genotype cannot be specified (Tables 5 & 6). Similarly, resistance gene(s) in the cultivar Rudd could not be specified from multipathotype tests because it gave very low ITs to all pathotypes, however, molecular marker analyses demonstrated the presence of *Yr17* in this cultivar (Fig. 1). So it can be concluded that this cultivar carries additional undesignated gene(s) with *Yr17*. *Yr7* was postulated in H45 because it was susceptible to only the pathotypes virulent for *Yr7* (Tables 5 & 6). Bariana *et al*., (2003) and Bariana *et al*., (2007) also reported adult plant resistance gene *Yr18* in H45.

Postulation of rust resistance genes by molecular markers: The list of DNA marker linked with rust resistance gene *Yr17* with their primer sequences and expected size of PCR products are given in Table 2. The 252-bp PCR product from primer VENTRIUP (Helguera *et al*., 2003) was observed in all Australian spring wheat cultivars tested and in positive check Avocet + Yr17 NIL (Fig. 1). This result showed distinctive and strong signals for presence of stripe resistance gene *Yr17* in Australian cultivars. The amplified DNA from 21 Australian cultivars and *Yr17* NIL (Positive check) confirmed that all the cultivars tested carry *Yr17*.

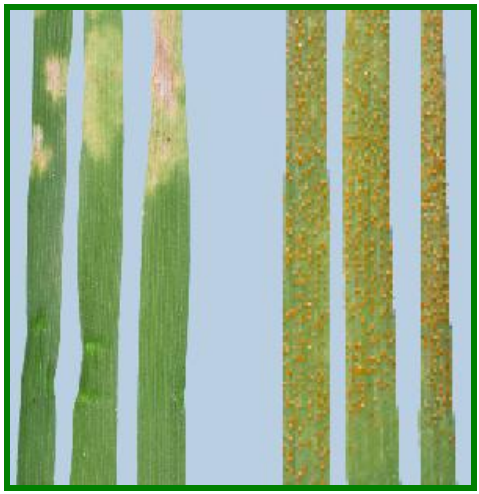

Fig. 2a. Seedling response of *Yr17* possessing cultivars A) Binnu, B) Gladius and C) Trident to *P. striiformis* pt. 134E16A$^+$ (left) and 134E16A$^+$*Yr17* (right) at 15-20^{o}C

Fig. 2b. Seedling response of *Yr17* possessing cultivars A) Binnu, B) Gladius and C) Trident to *P. striiformis* pt. 134E16A$^+$ (left) and 134E16A$^+$*Yr17* (right) at 12-15^{o}C.

Influence of temperature on expression of *Yr17*: Two temperature regimes (low temperature = 12-15^{o}C, high temperature = (15-20^{o}C) were maintained in separate green houses. The seedling responses of all cultivars including the reference cultivars Gladius, Binnu and Trident varied from lower IT to high IT different temperature regimes (Table 7 & Fig. 2). The cultivars Barham, Bowie, Carinya and QAL 2000, produced compatible reactions (IT 3/3+) for pathotype 134 E16 A$^+$ (Table 7) at low temperature (12-15^{o}C), whereas at higher temperature (15-20^{o}C), they showed a low IT (CN). Similarly, Stylet and Peake had a compatible reaction (3+) at lower temperature whereas they showed a low IT (CN1/1=CN) at higher temperature. The IT produced by Gladius and Derrimut were 3+/3 and 1=CN/1-CN at 12-15^{o}C and 15-20^{o}C, respectively. Ellison also showed a compatible reaction at lower temperature with an IT of 3+ and low IT (12-CN) at higher temperature. Similarly, Young exhibited full compatibility (IT 3+) at the lower temperature whereas it showed a decreased IT (0, 1p1+C) at the higher temperature. The cultivar Sunvale indicated high IT (3) and low IT (;) at lower and higher temperatures, respectively. The reaction of Marombi was 23CN at lower temperature while it showed a very low IT (0;N) at higher temperature. The IT of cultivar EGA Eaglehawk was "12-CN" and ";CN" at lower and higher temperature, respectively. EGA Jaeger produced 2+3CN IT at lower temperature whereas they produced ";CN" reaction at higher temperature. Sunzell produced IT, 23CN and ";CN1" at lower and higher temperature respectively. Sunland and Ventura indicated intermediate reaction (IT, 2+3N/2+3CN) at lower temperature whereas they produced reduced IT (12+CN/1-2-CN) at higher temperature. Similarly, Yenda and Qalbis indicated full compatible reactions (IT, 3+) at lower temperature and intermediate reaction (IT, 2+3CN/22+C) under higher temperature. Sunbri and Sunlin demonstrated less variation in their responses to temperature. Furthermore, Braewood demonstrated consistent reactions (IT, ;N or ;NC) under both temperature regimes.

Almost all of the above cultivars produced compatible ITs for pathotype 134 E16 A^+ $Yr17^+$ under both temperatures. Rudd showed a low IT (0 or ;) for both pathotypes (134 E16 A^+ and 134 E16 A^+ $Yr17^+$) at both temperature regimes, indicating additional gene(s) which is less sensitive to temperature variation. However, H46 was susceptible (IT, 3 or 3+) under both temperatures for both pathotypes, suggesting the absence of *Yr17* gene.

These results demonstrated the effect of temperature on expression of resistance conferred by *Yr17*. Seedlings that possess *Yr17* expressed higher level of resistance under higher temperature (15-20ºC). Under lower temperature (12-15ºC) most cultivars possessing *Yr17* showed compatible or near-compatible reactions to the pathotype 134 E16 A^+. These results are in contradictory with those of Bayles & Herron (1986) who found seedling susceptible and adult plant resistance reaction to yellow rust in a wheat cultivar Redezvous, which carries *Yr17*. They might have used a culture that was virulent for *Yr17* and additional genes might have conferred the adult plant resistance. In their experiment the environmental conditions might not be conducive for appearance of *Yr17* in the seedling tests. However, the results were in conformation to the findings of Bariana & McIntosh (1994), who reported that under low light and low temperature conditions (10ºC), seedlings carrying *Yr17* showed susceptible responses to avirulent pathotype. So it is concluded that the expression of stripe rust resistance conferred by *Yr17* is affected by environmental conditions and genetic background.

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