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High and low pre-inoculation temperatures decrease the effectiveness of the *Lr20* and *Sri5* rust resistance genes in wheat

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Spring wheat seedlings containing *Lr20* and *Srl5* resistance alleles were raised at 30°C, prior to inoculation with leaf rust *(Puccinia recondita* race 76-2.3) and stem rust *(Puccinia graminis* f.sp. *tritici* race 343-1.2.3.5,6) pathogens, respectively. Infected plants were then grown at one of seven temperatures in the range 18°-30° C and infection types were scored at 10 days post-inoculation. These results were compared with those obtained for plants raised at a pre-inoculation temperature of 18 °C. In both 18 C and 30° C pre-grown plants, a progressive increase in infection type was observed on resistant lines as post-inoculation temperature increased.

However, resistant lines raised at 30°C had significantly higher infection types than plants raised at 18° C at all post-inoculation temperatures for which some degree of resistance was still evident in the plants raised at 18° C. The maximum temperature for expression of resistance was significantly higher for *Lr20* than for *Srl5, irrespective* of pre-inoculation temperature. A lowering of the resistance expression was also evident in Sr/J-bearing lines raised at a very low pre-inoculation temperature (4°C). The effects of low pre-inoculation temperature on resistance were assessed in both winter and spring wheat lines. These results are discussed in the light of current ideas concerning the host membrane location of pathogen recognition events.

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INTRODUCTION

Temperature effects on host-pathogen interactions in cereals were first noted in the late 1920s when a loss of rust resistance in wheat and oat seedlings was observed under warm summer conditions (Waterhouse, 1929). Later studies, in which environmental temperature was strictly controlled, confirmed these observations (Forsyth. 1956; Lange *el al.,* 1958; Bromfield, 1961: Zimmer & Schafer, 1961; Luig & Rajaram, 1972). Subsequently, in a study of oat seedlings infected with the stem rust pathogen *{Puccinia graminis* f.sp. *avenue).* Martens *el al.* (1967) demonstrated that temperature sensitivity is a specific property of particular resistance genes, and that the temperature at which resistance is lost varies for different resistance genes, independently of the genetic background in which they occur.

Not all temperature-sensitive resistance genes lose effectiveness as temperatures increase, A survey of leaf rust *(Puccinia recondita)* resistance [2]genes backcrossed into the wheat variety Thatcher indicated that several of these genes are effective at higher temperatures (20-25 C) but ineffective at lower temperatures (10—15^{C}C) (Dyck & Johnson, 1983). Similar temperature-dependent resistance against stripe rust *(Puccinia striiformis* f.sp. *tritici)* has also been reported (Gerechter-Amitai *et al.,* 1984; Park *et al.,* 1992)**.** While the potential of temperature-sensitive resistance genes for use as experimental material in mechanistic studies of cereal rust resistance was recognized quite early (Bromfield, 1961), relatively few studies have attempted to investigate this potential. Rather, investigations have concentrated on determining which genes exhibit temperature-dependent expression, and the range of post-inoculation temperatures within which they maintain their effectiveness (Bromfield. 1961; Martens *el al..* 1967; Dyck & Johnson. 1983). Perhaps the best studied are the wheat genes *Srl5* and *Lr20* (Jones & Deverall. 1977a. 1977b; Gousseau *el al,.* 1985; Gousseau *&* Deverall 1986, 1987). which confer resistance towards avirulent races of the stem rust and leaf rust pathogens, respectively. These resistance factors prevent growth of the respective avirulent races at 20°C, but are increasingly ineffective as post-inoculation temperatures approach 30°C. It is considered that the two characters are expressions of a single gene which is effective against avirulent races of both pathogens (Mclntosh, 1977; Deverall & McLeod, 1980).

While the effects of post-inoculation temperature on host-pathogen interactions in cereals have been widely recognized, there have been only a few reports of the effect of pre-inoculation temperature on these interactions (Sharp 1962; Jones & Deverall, 1977a; Gousseau & Deverall, 1987). These studies suggest that the effectiveness of certain temperature-sensitive resistance genes is lowered when seedlings are raised at high temperatures before inoculation. The effect of very low pre-inoculation temperatures on such disease interactions has not been reported.

In the present study we have systematically examined the effect of both high and low pre-inoculation temperatures on post-inoculation expression of the *Lr20* and *Srl5* genes in wheat. Our results indicate a significant sensitivity of gene expression in response to either extreme of pre-inoculation temperature.

METHODOLOGY

Materials

Seed samples of spring wheat lines were obtained from the University of Sydney Plant Breeding Institute, Cobbitty, New South Wales, Australia. Fully susceptible lines (Federation and Chinese Spring) contained *lr20* and *srl5.* Lines containing *Lr20* and *Srl5,* of similar genetic background to susceptible lines, were Fedka (Federation*3/ Kenya W744) and Chinese Spring* 5/Axminster 7A. Winter wheat lines were obtained from the Australian Winter Cereals Collection, Tarnworth, New South Wales, Australia. Chancellor, a soft red winter wheat containing *srl5,* was chosen as the fully susceptible line. Lines with *Sr15* in a Chancellor background were Norka/ 8*Chancellor (AWCC accession 14160), Axminster/8*Chancellor (AWCC 14157) and Asll/ 8*Chancellor (AWCC 14159).

Uredospores of *Puccinia recondita* race 76-2,3 and *Puccinia graminis* f.sp. *iritici* race 343-1,2,3,5,6, avirulent with *Lr20* and *Sri* 5, respectively, were obtained from Dr R. Rees at the Queensland Wheat Research Institute, Toowoomba, Queensland, Australia. Adequate supplies of uredospores were maintained by increase on the susceptible cultivar Morocco and stored under liquid nitrogen until required.

Methods

Prior to inoculation, wheat seed was planted in pots 10 cm in diameter in a 1:1:1 vermiculite: peat: sand mixture and seedlings were raised at temperatures of either 18 °*C* or 30° C for 7 to 9 days in temperature-controlled growth cabinets. A 16'8-h day/night photoperiod was employed and light intensity ranged from 180 to 250 /^mol/ s/m^2 at soil level. Seedlings to be raised at 4° C were germinated at 18 C until coleoptile emergence and then transferred to a 4 °C glass-door refrigerator for approximately 24 days. Natural and incandescent light provided plants with a light intensity of 70 ^mol/s irr at soil level. In all cabinets, humidity levels were between 70% and 80%. Temperature was controlled to within 1°C of the set temperature and was monitored with a thermohygrograph.

Spores were mixed with pure talc in a ratio of approximately 1:20 by weight and applied to the underside of fully developed primary wheat leaves, using a small brush. Inoculated plants were placed in a perspex humidity chamber kept in a dark, air-conditioned room at 18°- 20° C for 16-18h. Infected seedlings were then transferred to growth cabinets set at one of nine post-inoculation temperatures (12. 15, 18, 20, 22. 24. 26, 28 or 30° C) for 10 days, with the exception of plants maintained at 12 ° or 15°C, which were grown

for 15 days, since at these temperatures symptom development was much slower.

Infection types were assessed 10 days (or 15 days) after inoculation, and typical leaves were photographed in order to ensure consistency of infection typing over time. The criteria used to classify stem rust and leaf rust infection types were based on those of Stakman *et al-* (1962), and are listed in Table 1. Leaves with necrosis present were regarded as expressing some degree of resistance despite the size or abundance of pustules also present. Fully susceptible responses had no associated necrosis on the leaf.

RESULTS

Table 1 *Criteria used to determine* ***the infection types of*** *Puccinia graminis f.sp. tritici and Puccinia recondita in wheat, 10 days after inoculation*[3]

Rating	Description
0	Immune host response, no macroscopic symptoms of infection
;	Small necrotic flecks
1	Small pustules surrounded by necrosis
2	Medium to large pustules surrounded by necrosis
3	Pustules with or without surrounding chlorosis
X	Mesothetic reaction, several infection types on the one leaf

[a] Where two or more ratings are given without separating punctuation, infection types listed appeared on the same leaf. A comma between types indicates differences between leaves of the same replicate, while a diagonal slash indicates differences in the infection types of replicates. The most frequent reaction type is underlined. Extensive chlorosis on the leaf is denoted by 'c', and plus or minus signs indicate that pustules were larger or smaller than the normal size limits

Expression of *Lr20*

Spring wheat lines with *Ir2t)* were susceptible to *P recondita* race 76-2.3 at all post-inoculation temperatures tested, regardless of pre-inoculation temperature (Table 2). Resistance to the leaf rust pathogen, conferred by *Lr20.* was observed as necrotic flecks on leaves in plants raised **at** 18 C before inoculation and at 18°, 20° and 22°C for 10 days after inoculation. Some uredia appeared on seedlings kept at post-inoculation temperatures of 24°C and 26°C, but were surrounded by necrotic tissue. On the leaves of Lr20-containing lines maintained at 28° C and 30°C, many large uredia were observed, with small necrotic flecks being associated with the margins of only a very few uredia (Table 2).

Z.r20-containing lines raised at 30° C prior to infection responded differently from lines raised at 18° C. Small to medium-sized pustules surrounded by

necrotic tissue were observed on the leaves of plants kept at 18°C and 20C for 10 days after inoculation, and a mixed response was evident on seedlings maintained at a temperature of 22°C after inoculation (Table 2). At post-inoculation temperatures of 24°, 26° and 28°C, uredia were abundant on leaves, and only a few of these were associated with necrotic flecks. Wheat lines with *Lr20* grown at 30°C before and after inoculation exhibited a completely susceptible infection type in response to *P. recondita* infection.

Expression of *Sr15*

All lines containing *srl5* were susceptible to *P. graminis* f.sp. *tritici* race 343-1,2,3,5,6, regardless of the temperature regime employed (Table 3). Both winter and spring wheat lines bearing the *Sri5* allele, raised at 18° C prior to inoculation, had necrotic flecks or very small pustules surrounded by necrosis on infected leaves at post-inoculation temperatures of 20° C and below. Larger pustules with associated rings of necrosis were present on leaves at 22° *C* and 24° C.

Table 2 *The development of Puccinia rccondita race 76-2,3 in spring wheat lines at different pre- and post-inoculation temperatures*

Line	Pre-inoculation temperature (°C)	Post-inoculation temperature (°C)						
		18	20	22	24	26	28	30
Chinese Spring	18	3	3	3	3	3	3	3
	30	3	3c	3	3	3	3c	3c
Chinese Spring 5* Axminster 7A	18	;	1– ;	;12 1–	12/1–	2 + 3/2	23/3	3c/2+
	30	;1–	1	1 2/X	23 2 + 3	2+ 2 + 3	3	3
Federation	18	3	3	3	3c	3c	3	3
	30	3	3	3	3c	3	3c	3
Fedka	18	;	;1	1/;1–	2,1 + 2	2	2 + 3	2 + 3/3
	30	12	12	2+, 23	2 + 3/X	3	3/X	3

Table 3 *The development of Puccinia graminis f.sp. tritici race 343-1,2,3,5,6 in spring wheat lines at different pre- and post-inoculation temperatures*

Line	Pre-inoculation temperature (°C)	Post-inoculation temperature (°C)								
		12	15	18	20	22	24	26	28	30
Chinese Spring	4	3	3	3-	3	3	3	3	3	nt
	18	3	3	3	3	3	3	3	3	3
	30	nt*	nt	3	3	3	3	3	3	3
Chinese Spring 5*/Axminster 7A	4	1–/1	1/1 + 2	1/2+	2++,3	3	3	3	3	nt
	18	;	;	;,;1	;1+/1 + 2	2 1	X	3c	3	3
	30	nt	nt	1	2,3	3	3	3	3c	3
Federation	4	3	3	3	3	3	3	3	3	nt
	18	3	3	3	3	3	3	3	3	3
	30	nt	nt	3	3	3	3	3	3	3
Fedka	4	;1/1	1,3/2	2	3	3	3	3	3	nt
	18	;	;	;1–	;1;1–	;1– 1,2–	1 3c	3	3	3
	30	nt	nt	;1+/12	X	3 X	3	3	3	3

*nt, not tested.

All lines were completely susceptible to the stem rust pathogen at and above temperatures of 26°C, 10 days after inoculation (Tables 3 and 4).
By comparison, 5r/5-bearing lines raised at a pre-inoculation temperature of 30°C were completely susceptible to *P. graminis* f.sp. *tritici* race 343-1,2,3,5,6 at post-inoculation temperatures of 22°C and above. At 18°C and 20° C. infected leaves developed pustules of restricted size which were surrounded by necrotic tissue (Table 3).

Wheat lines bearing *Srl5,* raised at 4°C before inoculation, were also completely susceptible to *P. graminis* f.sp. *tritici* race 343-1,2.3.5,6 at post-inoculation temperatures of 22° C and above (Tables 3 and 4). Symptoms on the same lines at post-inoculation temperatures of 12° C and 15° C were seen as small pustules surrounded by necrosis, 15 days after inoculation. Pustules with minimal associated necrosis were observed 10 days after inoculation on these lines at post-inoculation temperatures of 18°C and 20°C.

Table 4 *The development of Puccinia graminis f.sp. tritici race 343-1,2,3,5,6 in winter wheat lines at different pre- and post-inoculation temperatures*

Line	Pre-inoculation temperature (°C)	Post-inoculation temperature (C)							
		12	15	18	20	22	24	26	28
Chancellor	4	3	3	3	3	3	3	3	3
	18	3	3	3	3	3	3	3	3
Norka/*8	4	1	12	2+/2	3−	3	3	3	3+
Chancellor	18	;	;1−/;	;1−/1+	;/1	1+/1 + 2	3	3	3
Axminster/*8	4	2	2	2,3/2+	3−	3	3	3	3
Chancellor	18	;	;/;2	0/1,1+	;/1+	1 + 2	3	3	3
ASII/*8	4	1	1,2/1−	3/2+	3−	3	3	3	3
Chancellor	18	;	;1	1+,2/;1	;/1 + 2	1+/2	3	3	3

Plants grown at 4°C were raised at lower light intensities (70/xmol/s/m^2) than the plants maintained at 18°C and 30°C, due to lighting limitations in the cold cabinet that was used. This did not appear to have any significant effect on the symptoms observed, since plants that were raised at 18°C under low light intensities (70 ^mol/s/m^2) before inoculation, and then transferred to 18° C and normal light conditions after inoculation, gave similar results to plants raised at 18^{C}C at the higher light intensity (180-250 /jmol/s/m^2) used for all other temperatures (data not shown).

DISCUSSION

Our studies represent the first systematic investigation of the effects of pre-inoculation temperature on post-inoculation expression of the *Lr20\Srl5* complex. For lines with *Lr20* raised at a pre-inoculation temperature of 18° C, the transition from resistance to susceptibility was almost complete at the highest post-inoculation temperature tested (30°C) (Table 2). The transition from resistance to susceptibility for the Sr/5-stem rust reaction occurred at post-inoculation temperatures of 24°-26°C (Table 3). These findings are

consistent with those of previous studies (Jones & Deverall, 1977a; Gousseau *et al.,* 1985) and establish that this 4°C difference between the stability of *Lr20* resistance expression and that of *Srl5* occurs regardless of pre-inoculation temperature (Tables 2 and 3).

The change from resistance to susceptibility, in seedlings with *Lr20/SrI5* raised at 30°C before inoculation, occurred at post-inoculation temperatures significantly lower than those recorded for plants grown at 18°C prior to inoculation (Tables 2 and 3). Furthermore, in plants raised at 30°C, there is a comparative decrease in the degree of resistance conferred by *Lr20* and *Sr15* at all post-inoculation temperatures at which resistance is normally expressed in plants raised at 18°C. Previously, Jones & Deverall (1977a) observed that the growth of avirulent *P recondita* colonies on the wheat cultivar Thew *(Lr20j Sr15)* at 26° C was more extensive on plants raised at a pre-inoculation temperature of 30-5°C than on those raised at 205'°C. Similarly, Gousseau *&* Deverall (1987) observed that the development of avirulent *P. graminis* f.sp. *tritici* colonies in the presence of the *Sr15* resistance gene was more extensive at a post-inoculation temperature of 18°C when cv. Thew seedlings were raised at 30°C than when they were raised at 18°C prior to infection.

Both genetic mapping (The & Mclntosh, 1975) and mutational analysis of *Lr20* and *Sr15* (Mclntosh, 1977) provide strong evidence that a single host gene product recognizes the avirulence gene product from each of the two rust pathogens. If one host gene is indeed responsible for both resistance characters, then the finding that recognition of the *P. recondita* avirulence gene product is less sensitive to temperature-induced changes than recognition of the *P. graminis* f.sp. *tritici* avirulence gene product implies that the products of the two avirulence genes are different. This view is supported by the observation that, in mutant lines, the degree of change in resistance expression against the two pathogens was not the same (Mclntosh, 1977).

The results obtained for resistant lines grown at a low temperature (4°C) indicate a loss of subsequent *Srl5* expression that is at least as marked as that observed in the same lines when raised at high temperatures (30°C) (Table 3). To our knowledge, this is the first report of low pre-inoculation temperatures altering resistance gene expression in cereals.

Several hypotheses have been proposed to explain the effects of temperature on some disease resistance genes. Ellingboe (1976) suggested that temperature sensitivity is due to denaturation at high temperatures of thermal-labile proteins, which are products of the resistance genes. This view is consistent with the observed loss of *Lr20 Sr15* resistance at high post-inoculation temperatures, and its relative decrease following elevated pre-inoculation temperatures. However, instances where resistance is less effective at lower post-inoculation temperatures (Dyck & Johnson, 1983; Park *et* a/.. 1992), and the findings of the present study, **that** seedlings raised at 4°C before inoculation show reduced resistance, are not readily explained by protein thermolability alone.

More recently, Gousseau *et al.* (1985) have suggested that changes in plant cell membrane properties which occur in response to changes in environmental temperature may alter the tertiary protein structure of membrane-associated resistance gene products, thus modifying their ability to recognize corresponding pathogen avirulence gene products. Such modifications could explain both increases and decreases in resistance as post-inoculation temperatures are increased.

Research on both chilling injury and high temperature stress in plants has revealed that acclimation to persistent low or high temperature brings about changes in both the composition and the fluidity of plasma membranes (reviewed in Raison, 1985). These changes can also affect the conformation and activity of membrane-bound proteins (Raison, 1980). Such changes may be responsible for the lowered resistance expression *o(Lr20/Srl5* in seedlings acclimatized to very low or very high pre-inoculation temperatures (Tables 2, 3 and 4).

The concept that post-inoculation temperatures alter the sensitivity of membrane-located recognition events has been developed further by Judelson and Michelmore (1992). They propose that altered membrane properties restrict signal transduction rather than, or in addition to, directly restricting the recognition function of resistance gene products. If signal transduction is the critical temperature-labile event, then loss of resistance should occur at the same temperature for both avirulent pathogens, given that trans-membrane signal transduction is likely to be the same for all recognition events conditioned by *Lr20/Srl5.* This is clearly not the case. Rather, our results are consistent with temperature-induced alterations to the membrane-located *Lr20/Srl5* product differentially affecting its ability to bind with the leaf rust and stem rust avirulence products, with recognition of the stem rust product being more sensitive to these changes. An earlier study in wheat (Gousseau *et al.,* 1985) showed that the temperature sensitivity of *Sr9b* resistance varied towards two avirulent strains. This again suggests that the primary recognition event is sensitive to temperature variation, rather than the subsequent signal transduction pathway.

Studies of the adaptation of thylakoid membranes of rye and wheat to low temperatures have suggested that, in varieties which differ in cold hardiness, membrane fluidity differs at the end of the hardening process (Vigh *et al.,* 1979, 1987). We tested several sources of *Sri5* back-crossed into either a spring wheat (Chinese Spring, Federation) or a winter wheat (Chancellor) background in order to investigate whether possible plasma membrane-related differences between cold-acclimatized spring and winter wheat backgrounds could affect the subsequent expression of *SrJ5.* No gross differences were observed between the responses of resistant lines, suggesting that, while low pre-inoculation temperatures reduce the effectiveness of *Sr15,* this effect is independent of any variation in cold-adapted membrane properties which may occur between these spring and winter lines. Indeed, throughout this study any differences in genetic background between resistant host lines appeared to have negligible effects

on expression. There is one report (Dyck & Johnson, 1988) that, in a study of four European wheat cultivars bearing *Lr20,* the resistance of one cultivar (Sicco) displayed a much higher temperature sensitivity than the others tested. The authors suggested that variation in the *Lr20* allele might be responsible, rather than differences in genetic background. If this hypothesis is correct, then it is entirely consistent with the sensitivity of *Lr20/ SrI5* resistance expression to small alterations in gene product conformation indicated both by our results and by the earlier mutation studies (Mclntosh, 1977).

The present study clearly demonstrates that the well-recognized post-inoculation temperature sensitivity of the expression of the *Lr20! Srl5* gene complex can be significantly modified by the pre-inoculation temperature conditions. In particular, very low pre-inoculation temperatures have been observed to lower the resistance expression of the *Srl5* gene significantly. Furthermore, if one accepts that *Lr20* and *Sri5* are expressions of the same gene (Mclntosh, 1977), the observation that the phenotypic expression of *Lr20* is stable at significantly higher temperatures than that of *Srl5* implies that the avirulence gene products from the two fungal species are different. Thus we have an example of gene-for-gene complementarity (Dixon & Lamb, 1990; Keen, 1990) in which a single resistance gene product recognizes two structurally different avirulence gene products from related fungal pathogens. Given that changes in lipid composition and membrane fluidity have been shown to occur at both high and low temperatures (Raison, 1985), these results support an important role for the plasma membrane in host-pathogen interactions. Further experimental manipulation of temperature-dependent gene complexes, such as *Lr20/SrJ5,* may provide valuable insights into the biochemical and physiological mechanisms whereby the expression of resistance genes can be modified *in vivo.*

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