

Inheritance of seedling resistance to stripe rust (*Puccinia striiformis* f. sp. *tritici*) in 'Otane' and 'Tiritea' wheat (*Triticum aestivum*)

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Abstract Two spring wheat (*Triticum aestivum*) cultivars, 'Tiritea' and 'Otane', were crossed and the reactions of the F₁-derived double haploid (DH) population to the stripe rust pathogen (*Puccinia striiformis* f. sp. *tritici*) at the seedling stage were examined in glasshouse and field experiments. Both parental cultivars were susceptible to stripe rust pathotype 106E139A⁺, but transgressive segregation in the DH progenies indicated that both parents possess resistance genes. The distribution of DH lines fitted a trigenic ratio with epistatic gene action. In a proposed model, resistant DH lines were produced when all three loci involved were homozygous recessive, moderately resistant DH lines were produced when the *t* gene was homozygous recessive with at least one recessive allele at either the B or the C locus, and susceptible DH lines were produced when either *t*, *b*, or *c* were present alone or had a dominant gene at the B and

the C loci. The susceptible phenotype of all F₁ progenies indicated that the resistance factors segregated were all recessive. The susceptible reaction of both cultivars could be attributed to the presence of genes that may suppress the expression of resistance genes when they coexist in the parents. The use of these cultivars as parents in different wheat breeding strategies is briefly discussed.

Keywords *Triticum aestivum*; *Puccinia striiformis*; disease resistance; resistance suppression; transgressive segregation; genetic models

INTRODUCTION

Stripe rust, caused by *Puccinia striiformis* West. f. sp. *tritici*, is one of the most important diseases of bread wheat (*Triticum aestivum* L.) in temperate areas of the world, particularly those with cool, maritime climates such as northern Europe and north-western America. This disease is also common at high altitudes in some subtropical regions, including parts of East Africa, Central and South America, and the Indian subcontinent. The disease has also been recorded in the warm and arid areas of Egypt, Iran, and Turkey (Russell 1978). Stripe rust was first detected in Australia in 1979 and in New Zealand in 1980 (Beresford 1982). Most stripe rust epidemics in eastern Australia begin in early to mid spring (Ash et al. 1991) when wheat crops are at the booting to heading stages of development, while the relatively mild winters in New Zealand create conditions that are ideal for earlier disease development (Johnson 1996). Stripe rust can seriously reduce grain yields, particularly when severe outbreaks develop before ear emergence; reports of losses in grain yield range from 20% (Doling & Doodson 1968) to 60% (Beresford 1982) and 84% (Murray et al. 1995) in susceptible wheat cultivars.

Despite the potential importance of stripe rust in New Zealand and other countries, very little research has explored the genetic diversity for host resistance in spring wheat cultivars. This diversity is important

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in breeding for resistance and interpreting variation in the races of the pathogen found on infected crops. Many investigations relating to the inheritance of host reaction have revealed the importance of understanding the genetic basis of host plant resistance to stripe rust when developing successful genetic resistance breeding strategies (Chilosi & Johnson 1990; Zwer & Qualset 1991). The New Zealand wheat 'Otane' has conferred a moderate level of adult plant resistance to stripe rust since its release in 1984 (Cromey & Munro 1992), but this cultivar is susceptible at the seedling stage. Therefore, the aims of this study were to detect the number and diversity of resistance genes operating at the seedling stage in 'Otane', and measure whether significant progress in the improvement of resistance can be achieved when two cultivars with susceptible seedlings are crossed.

MATERIALS AND METHODS

Plant material

Two wheat (*T. aestivum*) cultivars ('Otane' and 'Tiritea'), both of which are susceptible to stripe rust at the seedling stage of growth (Cromey 1992), were crossed. From the F₁ plants of the cross, 140 double haploid (DH) lines were produced using the wheat-maize technique (Laurie & Bennett 1988) and were developed by M. Forbes and S. Shorter of the New Zealand Institute for Crop & Food Research Limited. Both cultivars are spring-sown wheats selected from germplasm of the International Maize and Wheat Improvement Centre (CIMMYT), Mexico (Bezar et al. 1982; Sparks et al. 1987).

Fungal culture

Culture WYR 93/5 (pathotype 106E139A⁺) of *P. striiformis* f. sp. *tritici* used in this study was initially obtained from the rust culture collection of the New Zealand Institute for Crop & Food Research Limited, Christchurch, New Zealand, and was increased on the susceptible 'Tiritea'. Pathotype 106E139A⁺ was originally obtained from 'Karamu', which possesses the stripe rust resistance gene *Yr_A*. Pathotype 106E139A⁺ has virulence for the world differential varieties 'Lee' (*Yr₇*), 'Vilmorin 23' (*Yr₃*), 'Strubes Dickkopf' (*Yr_{SD}*), and 'Suwon 92/Omar' (*Yr_{SO}*); the European differential varieties 'Hybrid 46' (*Yr₄*), 'Reichersberg 42' (*Yr₇*), 'Nord Desprez' (*Yr₃*), and 'Heines VII' (*Yr₂*); and Australasian differential variety 'Avocet' (*Yr_A*), according

to the pathogenicity pattern described by Johnson et al. (1972) and Wellings & McIntosh (1990).

Inoculum production and inoculation

Ten seeds of the susceptible 'Tiritea' were sown in 10 cm pots containing potting mix. When the first leaf emerged, a water solution (0.2 g/litre) of maleic acid (1,2-dihydro-3, 6-pyridazinedione) was applied to the soil to keep the plants short, delay leaf senescence, and increase uredospore production (Stubbs et al. 1986). At the second leaf stage the plants were inoculated with *P. striiformis* f. sp. *tritici* pathotype 106E139A⁺. For inoculation, the spore suspension was made by mixing uredospores with light mineral oil (Pegasol, Mobil) in a vial. The plants were sprayed uniformly with the spore suspension using an atomiser, and 5–10 min later were sprayed lightly with water. Plants were maintained at 10 ± 1°C in a dark moist chamber for 24 h and then transferred onto glasshouse benches in isolation at 15 ± 1°C. Sporulation began 14–15 days after inoculation. Inoculum was collected several times by holding the sporulating plants over a sheet of aluminium foil and gently tapping them so that uredospores fell onto the foil. The uredospores were dried for 24 h in a desiccator containing silica gel before being sealed in plastic-lined, aluminium foil packets for storage in a freezer at –80°C. Before use, uredospores were heat shocked by immersing the packets in water at 42–44°C for 4 min to break dormancy (Stubbs et al. 1986).

Screening procedures

Three replicates of one seed of each DH line, two seeds of the F₁, and four seeds of both parents were sown on 20 August 1999 in individual pots (10 cm) containing potting mix and arranged in a glasshouse (16 h daily photoperiod at 15 ± 1°C) in a randomised block experimental design, with benches being blocks. The 16 h lighting included 8 h artificial light provided through ten 400 W lamps. Fourteen days after sowing the seedlings were inoculated with *P. striiformis* f. sp. *tritici* pathotype 106E139A⁺ as previously. Approximately 0.5 mg urediospores were applied per plant. Disease reactions, based on infection type (IT), were recorded 18 days after inoculation using a numerical scale of 0–9 (Line et al. 1974). Disease classes from 0 to 3 were classified as resistant (R), 4 to 6 as moderately resistant (MR), and 7 to 9 as susceptible (S) (Table 1). Since IT is a descriptive component of resistance, mean values were rounded up to whole figures following the general mathematics rule. The 140 DH lines and the

two parents were also sown on 3 August 2000 and inoculated with the pathotype 106E139A⁺ in the field following the experimental design and inoculation method described by Imtiaz et al. (2003) in an adult plant study. In the field, recording of IT data began at growth stage (GS) 21 (main shoot and one tiller stage) and continued at weekly intervals (a total of five assessments) until GS 30 (pseudo stem erection) (Tottman & Makepeace 1979). Maximum IT data were used for the analysis.

Statistical analyses

Chi-square (χ^2) analyses were used to test the goodness of fit of observed ratios to theoretical expectations. Contingency table χ^2 analysis was used to compare glasshouse and field data. One-, two-, and three-gene and gene-interaction models were tested.

RESULTS

As both parental cultivars were susceptible at the seeding stage, it was hypothesised that any segregation for resistance in the DH population would indicate the presence of gene interactions. The expectation was upheld as resistance was observed in the DH population. A genetic model hypothesis for the DH population was based on the classification

of three phenotypes, resistant (IT 0–3), moderately resistant (IT 4–6), and susceptible (IT 7–9). Establishing the relationship between parents, F₁ genotypes and infection types would allow the development of a genetic model incorporating the three classes.

Glasshouse studies

Both ‘Tiritea’ and ‘Otane’, as well as the F₁, exhibited a high IT (9) to the stripe rust pathotype 106E139A⁺ (Fig. 1). However, three distinct phenotypic resistance classes against stripe rust pathotype 106E139A⁺ were observed in the progeny as described above. Although a large proportion of the DH population was identified as susceptible, transgressive segregation was apparent in the progeny of the cross (Fig. 1). The mean DH population IT was 7.1, but there were DH lines with ITs ranging from 1 to 9 (Fig. 2). The numbers of DH lines exhibiting R, MR, and S infection types were 15, 39, and 86 respectively. This segregation fits a 1:2:5 ratio (Table 2), suggesting the involvement of three genes with epistatic gene action for resistance at the seedling stage in this population. This segregation pattern does not fit any two-gene models (1:0:3) or any other three-gene models for independently segregating factors (data not shown).

Table 1 Assessment scale used to characterise stripe rust (*Puccinia striiformis* f. sp. *tritici*) infection types and classes (Line et al. 1974 with modification).

| Class | Number | Symptoms and signs of the disease |
|---------------------------|--------|---|
| Resistant (R) | 0 | immune; no sign or symptoms |
| | 1 | necrotic and/or chlorotic flecks, no sporulation |
| | 2 | necrotic and/or chlorotic blotches or stripes*, no sporulation |
| Moderately resistant (MR) | 3 | necrotic and/or chlorotic blotches or stripes, trace sporulation |
| | 4 | necrotic and/or chlorotic blotches or stripes, light sporulation |
| | 5 | necrotic and/or chlorotic blotches or stripes, intermediate sporulation |
| Susceptible (S) | 6 | necrotic and/or chlorotic blotches or stripes, moderate sporulation |
| | 7 | necrotic and/or chlorotic blotches or stripes, abundant sporulation |
| | 8 | chlorosis behind sporulating area, abundant sporulation |
| | 9 | no chlorosis or necrosis, abundant sporulation |

*Blotches occur on seedlings and stripes occur on older plants.

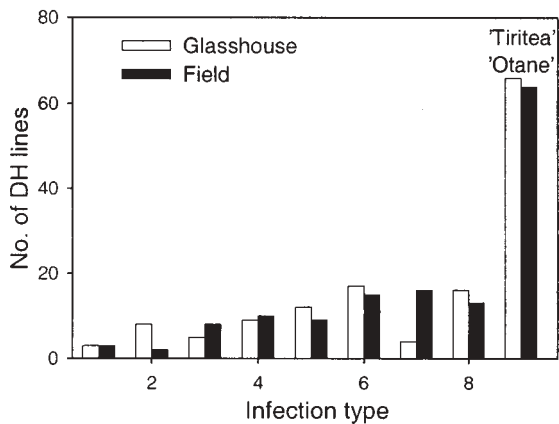


Fig. 1 Distribution of wheat (*Triticum aestivum*) double haploid (DH) lines for mean stripe rust infection types at the seedling stage in glasshouse and field experiments for a 'Tiritea' × 'Otane' population inoculated with *Puccinia striiformis* f. sp. *tritici* pathotype 106E139A⁺.

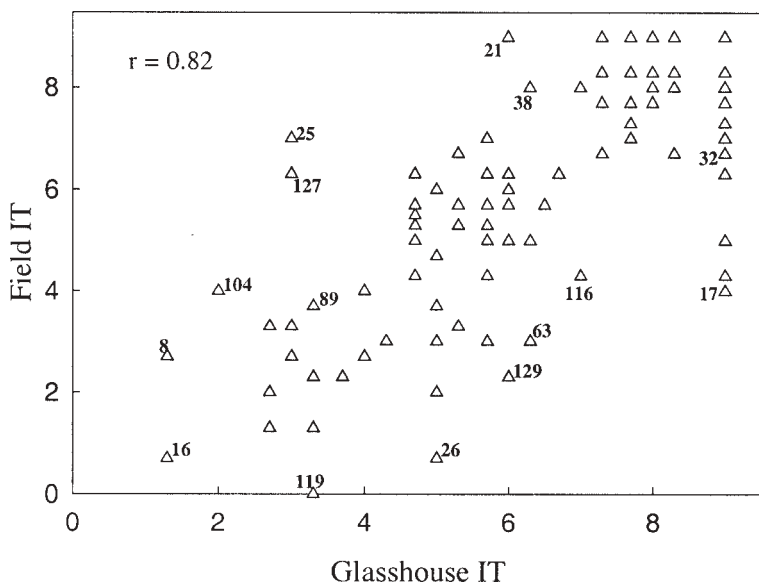


Fig. 2 Comparison of field and glasshouse infection type (IT) for double haploid (DH) lines through simple linear regression. Numbers identify DH lines with very different glasshouse and field resistance scores.

According to the three gene model, resistant DH lines are produced when three loci (*tbc*) were homozygous recessive (*tbc*), moderately resistant DH lines are produced when the *t* gene was homozygous recessive with at least one recessive allele at either the B or the C locus (*tbC* or *tBc*), and susceptible DH lines are produced when any of the recessive genes *t*, *b*, or *c* were present alone or had a dominant gene at the B and C loci or two recessive genes from one parent (Tbc; Table 2). The *tbc* represent genes involved in seedling resistance in this study, where "T" stands for the susceptible, and "b" for the resistance gene of 'Tiritea', and "r" designates the resistance gene of 'Otane' (Table 2). The hypothesised genotypes were TTbb and ttBB for

'Tiritea' and 'Otane' respectively whereas the third resistance gene "c" could be from either parent.

Field studies

Both parental cultivars were susceptible in the field, but transgressive segregation was evident in the DH population (Fig. 1). The mean IT in the field was 6.7 in comparison to the mean glasshouse IT of 7.1 (Fig. 2). The number of DH lines in the R group increased from 15 to 21, whereas the number of the DH lines in the MR group decreased slightly from 39 to 35. The S group in the field consisted of 84 lines in comparison to 86 in the glasshouse. DH-25 was classified as R in the glasshouse but S in the field (Fig. 2), while six other lines also changed from MR

in the glasshouse to S in the field. DH-89, DH-104, and DH-127 were R in the glasshouse but MR in the field. Of 39 MR DH lines in the glasshouse, 10 were R in the field, and 7 out of 86 DH lines that were S in the glasshouse were MR in the field (Fig. 2).

Although there was a change in the resistance level of some DH lines (Fig. 2), the Chi-square for independence ($\chi^2 = 121.70$ with 4 d.f.) and the correlation ($r = 0.82$) between glasshouse and field data was significant ($P < 0.001$). The segregation under field conditions was 21R:35MR:84S, which did not deviate significantly from the 1:2:5 trigenic ratio (Table 2).

DISCUSSION

Seedling resistance to stripe rust has largely been non-durable. However, it is still important to understand the nature of any diversity in seedling resistance so that desirable plant breeding materials can be distinguished and hybrid populations can be constructed for further selection. Both parents (‘Tiritea’ and ‘Otane’) were susceptible at the seedling stage, but transgressive segregation was observed for resistance and susceptibility (Fig. 1), a result also reported by

other researchers (Johnson & Wilcoxon 1979; Wallwork & Johnson 1984; Zwer & Qualset 1991; Ghannadha 1993). The transgressive segregation, whether for resistance or susceptibility, clearly originated from combinations of the genetic components from both parents.

The F₁ generation from the cross between ‘Tiritea’ × ‘Otane’ was susceptible, indicating that the resistance observed in the DH population was recessive. Segregation data for DH lines both under glasshouse and field conditions fitted the three-gene model. The presence of transgressive segregation suggests that both ‘Tiritea’ and ‘Otane’ possess recessive genes for resistance to stripe rust. It appears that ‘Otane’ possesses a gene (*yr_l*) that alone did not confer resistance to pathotype 106E139A⁺. However, in combination with other resistance genes, this gene acted positively in increasing the level of resistance in the DH population. ‘Tiritea’ also appeared to possess seedling resistance genes, which supplemented the resistance of ‘Otane’ in this study, but alone they were unable to confer any resistance. Both cultivars contribute at least one gene to cause the transgressive segregation in the DH population. The third gene segregating in this population most probably comes from ‘Tiritea’ because molecular studies (Imtiaz 2002) have

Table 2 Stripe rust (*Puccinia striiformis* f. sp. *tritici*) infection types for wheat (*Triticum aestivum*) ‘Tiritea’/‘Otane’, F₁ and double haploid (DH) lines grouped into R (resistant), MR (moderately resistant, and S (susceptible) and the probability of fit to hypothesised gene models (1:2:5) for wheat seedlings inoculated with *P. striiformis* f. sp. *tritici*, pathotype 106E139A⁺ under glasshouse and field conditions.

| Cultivar/ generation | Phenotype | Observed infection types | Hypothesised genotype | Expected proportion | Observed no. | χ^2 | Prob. |
|-----------------------------------|-----------|-----------------------------|--|------------------------|-----------------|----------|-------|
| Tiritea (glasshouse and field) | S | 9 | TTbbcc | all | | | |
| Otane (glasshouse and field) | S | 9 | ttBBCC | all | | | |
| F ₁ (glasshouse) | S | 9 | TtBbCc | all | 2 | | |
| DH lines (glasshouse) | R | 1, 2, 3 | tbbcc | 1/8 | 15 | 0.84 | 0.65 |
| | MR | 4, 5, 6 | ttbbCC, ttBBcc | 2/8 | 39 | | |
| | S | 7, 8, 9 | TTBBCC, TTBBcc, TTbbCC, TTbbcc, ttBBCC | 5/8 | 86 | | |
| DH lines (field) | R | 1, 2, 3 | tbbcc | 1/8 | 21 | 0.84 | 0.65 |
| | MR | 4, 5, 6 | ttbbCC, ttBBcc | 2/8 | 35 | | |
| | S | 7, 8, 9 | TTBBCC, TTBBcc, TTbbCC, TTbbcc, ttBBCC | 5/8 | 84 | | |

revealed that 'Tiritea' contributed to the resistance from more than one region of the genome, whereas 'Otane' contributed to the resistance from only one region. Thus, 'Tiritea' appears to possess two genes (Yr_b, Yr_c). To further validate this model, the F_1 progeny will need to be backcrossed to both parents to study segregation of the genes. However, these findings support the studies of Ghannadha (1993) who used 'Tiritea' in different crosses, and tested the F_2 and F_3 families against different stripe rust pathotypes. He found transgressive segregation in almost all crosses and was of the opinion that 'Tiritea' had some resistance genes. Similarly, he crossed 'Otane' with 'Briscard', 'Ruapuna', and 'Domino' and found transgressive segregation in the F_2 of all crosses, indicating the presence of resistant genes(s) in 'Otane'.

Seedling disease reaction to *P. striiformis* f. sp. *tritici* has been widely studied (Allan et al. 1966; Lewellen et al. 1967; Singh et al. 1990; Ma & Singh 1997) and is assumed to follow a gene-for-gene relationship (McIntosh & Wellings 1986). In most of these instances transgressive segregation for resistance and susceptibility, and in some instances involvement of a recessive gene in control of resistance, was reported. Also in these studies, gene accumulation for resistance was observed even when two susceptible parents were crossed. This present study agrees with these earlier studies.

Johnson (1996) was of the view that transgressive segregation could arise from additive effects, or interactions of race-specific genes, or from the transfer of race-specific genes from a suppressive to an expressive background. Singh & Mujeeb-Kazi (1995) reported the suppression and expression phenomenon of resistance; resistance of a donor parent was expressed in a synthetic hexaploid wheat only when the corresponding suppressor was absent in the second parent. These suppressors appeared to be specific to resistance genes. The inhibition of seedling resistance genes has been previously reported (Hooker 1967). In the present study both 'Tiritea' and 'Otane' were susceptible at the seedling stage; susceptibility gene *T* probably inhibits the expression of recessive resistant genes in 'Tiritea', while the *t* gene of 'Otane' is also suppressed in its present genetic background. Therefore, inhibition of seedling resistance and transfer of a gene from a suppressive to an expressive background probably prevailed in this study. Kema & Lange (1992) reported similar suppression of race-specific

resistance in hexaploid wheat to stripe rust and also noted the influence of host growth stage on resistance suppression.

Knox et al. (1998) considered that segregation ratios for the same genetic material can differ because of different growing conditions, chance deviations, misclassification of lines, continued segregation within families, and the techniques used to develop the lines. Despite this, DH lines can be repeatedly tested without variation contributed by segregation (Knox et al. 1998). While only one seed of each DH line was sown per replicate, occasional off-types were readily detected through morphological differences (plant height, time to flowering, presence or absence of awns). In our trials there were shifts from resistant and moderately resistant in the glasshouse to susceptible under field conditions. Stakman (1954) also reported such a shift for stem rust susceptibility with increasing temperature. Other possible explanations could be disease escape, misclassification of DH lines, and/or naturally occurring pathotypes with virulence to these lines. The increase in resistant DH lines and decrease in moderately resistant lines were probably the result of better expression of resistance in the field, while a complete shift in a few lines either from resistant to susceptible or vice versa could be because of misclassification. Although there was a shift, significant independence χ^2 tests and goodness-of-fit tests for the infection types recorded under glasshouse and field conditions separately revealed that the same number of genes are probably involved. This also suggests that these genes are not largely affected by environment, indicating that inheritance of resistance at the seedling stage is not prone to significant genotype-environment interactions.

This study has demonstrated that both 'Tiritea' and 'Otane' possess seedling resistance genes that interact with each other to provide increased resistance to stripe rust. However, the new recombinants (DH lines showing resistance) obtained in this study against *P. striiformis* f. sp. *tritici* pathotype 106E139A⁺ should also be tested against a wide range of *P. striiformis* pathotypes to judge their suitability for use in any future breeding programmes. Furthermore, the use of 'Tiritea' as a susceptible parent in studies of the inheritance of resistance to stripe rust in a particular cultivar is not recommended because its resistance complicated the inheritance studies in target cultivars.

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