RESISTANCE IN *TRITICUM AESTIVUM* TO INFECTION BY *GAEUMANNOMYCES GRAMINIS* VAR *TRITICI*

by

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SUMMARY

Resistance in Triticum aestivum (wheat) to infection by Gaeumannomyces graminis var tritici (G.g.t.) was studied by comparing the extent to which roots of cultivars were colonised in controlled and field environments.

Two methods were employed to score early infection in wheats grown in cups in controlled environments. Severity of infection was more simply scored by the number of stelar lesions in roots than by directly measuring hyphae in sections, provided observations were made at least nine days from inoculation and before translocation had ceased. In preliminary experiments weight of seed was found to dissimilarly influence infection in two wheats. Thus, the extent to which hyphae had grown into the roots of cultivars did not uniquely indicate their resistance to infection by G.g.t. when plants were grown from a single weight of seed.

Wheats naturally infected with G.g.t. were studied at several sites. Though symptoms of infection with other pathogens were also found on roots, G.g.t. was the most damaging pathogen at the main sites of study. Wheats did not generally differ in the extent of infection with G.g.t. though highly significant differences in the incidence of deadheads were found, in association with differences in maturity of the cultivars studied. Maturity may largely account for differences in expression of disease caused by G.g.t. which are frequently noticed in the field in wheats that differ little in resistance to the pathogen.

Evidence was found of two forms of resistance to infection by G.g.t. One appeared in many tissues of wheat roots, was not of mechanical origin and may not be useful in improving field resistance to G.g.t., as it was not simply inherited and its effect on the colonisation of seminal and coronal roots was dissimilar. The other form of resistance was associated with cortical browning and appeared to limit damage to coronal roots infected with G.g.t. If this were to occur generally, cortical browning may prove useful in improving the resistance of cultivars to G.g.t. as it appeared to be simply inherited in a cross between a local and an exotic wheat.

The technique of associating controlled and field studies proved useful in investigating resistance to infection by G.g.t. in wheat. While strong levels of resistance were not found, the weaker levels of resistance detected may still prove sufficient to improve the field resistance of wheats grown in South Australia.

STATEMENT

This thesis contains no material which has been accepted for the award of any other degree or diploma in any university. To the best of my knowledge and belief, no material described herein has been previously published or written by another person except when due reference is made in the text.

L. Penrose

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CHAPTER 1

INTRODUCTION

Gaeumannomyces graminis (Sacc.) Arx and Olivier is a serious fungal pathogen of members of the Gramineae. Walker (1972) recognised three varieties of the fungus, of which var tritici is the most serious pathogen of cultivated wheat (Triticum aestivum L.). While the other varieties may also occur on wheat, var graminis is largely a pathogen of pasture grasses and var avenae a pathogen of oats (Avena sativa L.) (Walker, 1975). Symptoms of infection on wheat with Gaeumannomyces graminis var tritici (Walker) (subsequently referred to as G.g.t.) include blackening of the roots, crown nodes and subcoronal internode. The dysfunction of the root system that follows infection has been likened to the amputation of roots (Simmonds & Sallans, 1933) and can lead to reduced vigour or death of seedlings, premature death of tillers and heads, and shrivelling of grain.

G.g.t. is widely distributed in southern Australia where wheat is very susceptible, barley (Hordeum vulgare L.) less severely affected, and rye (Secale cereale L.) generally resistant. Many of the graminaceous weeds of cereal crops harbour the pathogen, of which the barley grasses (H. leporinum Link and H. glaucum Steud.) are the most significant (Banyer, 1966). Crops become infected with the pathogen by contact with hyphae from infected crop and weed residues from the previous and earlier years. Under favourable conditions the pathogen spreads along infected roots and to neighbouring plants via root contact, but is restricted by microbial antagonists (Rovira & Wildermuth, 1981) or

insufficient moisture (Cook, Papendick & Griffin, 1972). Early infection may be so severe as to cause stunting of seedlings, though often young crops show no symptoms of disease. Infection may spread through the crop as the season progresses with few additional symptoms other than a tendency for lower leaves to die prematurely (Warcup, pers. comm.). In a spring with above average rainfall, deadheads may become evident in infected crops, expressing loss of yield (Garrett, 1934). Detailed studies have not been conducted but observations suggest disease due to G.g.t. is expressed more frequently as deadheads than as stunted seedlings in crops of wheat in southern Australia (Price, 1970). Few studies have been made of loss of yield caused by the pathogen over extensive areas though losses of 0.9, 2.6 and 2.8% were estimated for wheat grown in England in the years 1977-79 respectively, (Polley and Clarkson, 1980). Surveys of similar detail have not been reported in Australia, though damaging infections are known to have occurred over large areas of South Australia in some years (Garrett, 1934) and losses of 66% determined in individual crops in Victoria (Price, 1970).

Resistance in wheat to infection by G.g.t. has been examined independently in histopathological studies and by observing infection in cultivars of wheat. Inherent in these avenues of study are separate perspectives of resistance. The histopathologist sees resistance as an impeding effect to the proliferation of a pathogen in its host, while the plant breeder is more demanding and considers resistance in relation to the economic fitness of a cultivar to an agricultural environment. Few have reported studies of resistance from both perspectives, as has Nilsson (1973).

Histopathological studies show roots of wheat are colonised by thin-walled hyphae of G.g.t. (hyaline hyphae) growing from pigmented and thick-walled hyphae (runner hyphae) that colonise the rhizoplane (Fellows, 1928). Hyaline hyphae grow radially into seminal roots, cross the endodermis and invade the stele (Fellows, 1928) where they disrupt the flow of translocates and cause the death of the distal portion of root (Clarkson, Drew, Ferguson and Sanderson, 1975). Several characters are thought to retard the invasion of seminal roots by G.g.t. The endodermis appears to retard invasion into the stele (Skou, 1975) while lignitubers in the lower cortex, endodermis and vascular tissue of infected roots may act similarly (Skou, 1981). Black deposits in the stele may retard the growth of G.g.t. up vascular tissue and into the scutellar and coronal nodes (Robertson, 1932). Crown roots are colonised similarly to seminal roots (Fellows, 1928) but are more resistant to invasion by the fungus (Sivasithamparam & Parker, 1978). The subcoronal internode and the scutellar and coronal nodes are of considerable significance to the wheat plant, and are more resistant again to attack by G.g.t. The root system of wheat becomes more resistant to infection by G.g.t. with maturity, possibly through the lignification of aging endodermal and vascular tissues (Robertson, 1932). Lignification of tissues also occurs prematurely when young roots of wheat are infected with G.g.t., but a corresponding increase in resistance to infection by the pathogen is not observed (Fellows, 1928). The contradictory role of lignified tissues in resisting

infection may be due to differing chemical constituents of lignin in tissues that are mature but uninfected, and those that are young and infected.

Studies to detect resistance or tolerance to infection by G.g.t. in cultivars of wheat have been conducted in controlled environments and in the field and have been reviewed by Nilsson (1969) and Scott (1981). Strong repeatable differences between wheats have not been detected in either environment (Scott, 1981). A problem is that seedlings grown in small containers have been inoculated with G.g.t. and scored for infection in many ways, leaving comparisons of the findings of different workers unclear (Scott, 1981). Comparisons are further complicated by Russell's (1934) finding that source of seed influenced the infection of wheats more strongly than did effects due to cultivars.

Field resistance and tolerance to infection by G.g.t. have been most commonly studied by artificially infecting cultivars of wheat with the pathogen and measuring blackening of roots and yield of grain. These studies have been conducted in a wide range of environments, typically without regard for the microflora of soil at sites or the manner in which disease is most frequently expressed in the field. Moreover, inoculum level is frequently manipulated to obtain disease in years in which the pathogen is not naturally damaging. While significant differences in resistance and tolerance to the pathogen may appear in such trials, findings are not consistent from year to year, and suggest significant interactions with the environment (Scott, 1981). Interactions most probably

reflect the range of conditions under which trials have been conducted, and the complexity of environmental influences on the severity of disease. As disease that accounts for most loss of yield in the field is likely to occur within a narrower range of conditions than are used in artificially inoculated trials, wheats may more consistently exhibit field resistance and tolerance to G.g.t. than previous studies have indicated. Thus resistance and tolerance in wheat to infection by G.g.t. should be tested in conditions in which the disease causes most loss of yield. While experiments with wheats following natural infection with G.g.t. are rarely reported, consistent differences between cultivars in the degree of blackening of roots have then been found (Nilsson, 1969).

Disease has also been observed in wheat trials that have been by chance naturally infected with G.g.t. (Sims, Meagher & Millikan, 1961). Before providing evidence of resistance or tolerance to G.g.t., the pathogen must be shown to have caused disease in such trials, and the original experimental design allow statistical testing of differences between wheats in severity of infection. Though not tested

in this manner, observations of trials that are by chance infected with G.g.t. have led many wheat breeders to believe wheats differ in resistance to infection by the pathogen (Rathjen, pers. comm.).

This study sought to obtain evidence of resistance to infection by G.g.t. in wheat and to observe its influence on yields of wheats infected with the pathogen in the field. The inheritance of resistance was also briefly studied to assess the prospects of breeding wheat for resistance to the pathogen. Resistance in wheat will be most clearly demonstrated when wheats are shown to be similarly infected in controlled and field environments. This will be best

achieved where infection is measured directly in controlled environments, and where field trials are conducted in natural environments with disease that is of common occurrence. Factors that influence disease caused by G.g.t. in the field were studied to account for differences between wheats that have been noticed by wheat breeders. As little is presently known of ways in which wheat plants resist infection by G.g.t., wheats were not 'screened' for resistance to the pathogen . Thus, studies were restricted to wheats presently grown in southern Australia and to exotic wheats previously selected for tolerance to the pathogen (Simon & Rovira, 1985).

CHAPTER 2

MATERIALS AND METHODS

Genotypes of T. aestivum

Wheats were infected with G.g.t. as sets of genotypes in individual experiments in controlled and field environments. Of the five sets of wheats that were employed (Table 1), four were of cultivars and one was of families of genotypes, and each set was infected with G.g.t. for differing purposes.

Set 1. These cultivars comprised eight advanced lines and releases from Australian wheat breeding programs, and in being available as fresh clean seed, were used to study infection of wheats with three isolates of G.g.t. This set of cultivars was not used after the completion of preliminary experiments.

Set 2. These cultivars differed most in infection with G.g.t. in preliminary experiments, and were selected from exotic and local wheats that were themselves thought to differ in resistance to infection by the pathogen (Rovira and Rathjen, pers. comm.). Of cultivars grown in South Australia that were tested, Kite appeared least infected and Condor and Rac311 most infected with G.g.t. Condor also appeared particularly susceptible to G.g.t. in the field (Rovira, 1977) as did Rac311 (personal observation). Of the four exotic wheats, Aus1080, Chile909 and Chile911 were least infected with G.g.t. while Nebraska86 was most infected in preliminary experiments.

TABLE 1: Identity and origin of cultivars of wheat used experimentally

Wheat	Origin	Wheat	Origin
Cultivars of set 1			
* Aus1080	Aust. collection	Kite	N.S.W.
Condor	N.S.W.	* Kite 4A/2R	S.A.
Festiguay	N.S.W.	Rac311	S.A.
Halberd	S.A.	Warigal	S.A.
Cultivars of set 2			
* Aus1080	Aust. collection	Kite	N.S.W.
* Chile909	Chile	* Nebraska86	U.S.A.
* Chile911	Chile	Rac311	S.A.
Condor	N.S.W.		
Families of set 3			
* 40 F_2 families from the cross Aus1080(♀)xCondor(♂)			
Cultivars of set 4			
Eagle	N.S.W.	(MM*MMC)/59/W6	S.A.
Egret	N.S.W.	Rac357	S.A.
Festiguay	N.S.W.	Rac399	S.A.
M2335	N.S.W.	Rac415	S.A.
M2424	N.S.W.	Rac416	S.A.
Oxley	N.S.W.	(WW-15*MH-49)/36/W3	S.A.
Sun39A	N.S.W.	Millewa	Vic.
Sun41A	N.S.W.	MQ6	Vic.
Sun43A	N.S.W.	PD36	Vic.
Cook	Qld.	SD34	Vic.
K-2003-12	Qld.	Jacup	W.A.
LR/OXS 2730-4	Qld.	69W/237	W.A.
N10/TG2248-8	Qld.	69W/393	W.A.
Halberd	S.A.	69Z/401	W.A.
(MKR*Kite)/57/S14	S.A.		
Cultivars of set 5			
Aroona	S.A.	Miling	W.A.
Condor	N.S.W.	Millewa	Vic.
Cook	Qld.	Oxley	N.S.W.
Festiguay	N.S.W.	PF/41/W1	S.A.
Jacup	W.A.	Rac311	S.A.
Kite	N.S.W.	Warigal	S.A.
Lance	S.A.	Warimba	S.A.
MC/29/S5	S.A.		

* not grown in Australia

Set 3. These genotypes were not of homozygous cultivars, but of 40 heterogeneous families from the cross Aus1080 x Condor. Each family was derived from an F_2 individual, and maintained by bulking grain at the harvest of successive generations. Families of set 3 were employed in both controlled and field environments to study genetic control of factors influencing infection of roots with G.g.t. While there is considerable genetic variation within F_2 families, there was insufficient time to generate more advanced and less heterogeneous families for study.

Sets 4 and 5. These cultivars were used to investigate resistance to infection with G.g.t. in wheats currently grown in Australia. By chance these wheats were naturally infected with G.g.t. in two separate trials conducted by the South Australian Department of Agriculture. Set 4 comprised the 30 wheats of the 1981 Series B Interstate Trial and were found to be infested with G.g.t. at Nangari (Fig. 1) in 1981. Eight cultivars of set 4 were selected for differences in symptoms of infection with G.g.t. in a preliminary study in a waterbath (Appendix 1) and were further studied in the field. Set 5 consisted of another 15 advanced lines and recent releases from Australian wheat breeding programs, found to be infested with G.g.t. at Nangari and Perponda in 1981.

Isolates of G. graminis var tritici

The isolates used in this study are listed in Table 2 with source, date of isolation and site of origin. Those personally isolated were from blackened roots from infected

TABLE 2: Origin of isolates of *G. graminis* var. *tritici*

Isolate	Site	Host	Date of Isolation	Isolated by
19	Caliph	Wheat cv. Raven	1979	J. Harris
42	Palmer	Wheat cv. Kite 4A/2R	1981	L. Penrose
108	Windsor	Wheat cv. Condor	1981	L. Penrose
201	Waite	*H. leporinum*	1981	L. Penrose
226	Palmer	Wheat cv. Chile909	1983	L. Penrose

hosts, and were identified by characteristics of colonies on one sixth strength neutral dox yeast extract (NDY/6) agar (Warcup, 1955) and type of hyphopodia on the coleoptiles of seedlings of wheat (Walker, 1972). Isolates were maintained in tubes on NDY/6 agar under mineral oil at room temperature. The locations of sites from which isolates were obtained are depicted in Figure 1.

Field sites

Experiments were undertaken in the field over three years (1981-1983), but were not concluded in 1982, a year of severe and general drought. All sites were within 250 km of Adelaide (Fig. 1). Data in 1981 were from yield trials of cultivars of wheat at Nangari and Perponda which were established by officers of the South Australian Department of Agriculture and found to contain disease caused by G.g.t. Soils at both sites were calcareous sandy loams (Gc1.22: Northcote, 1979), with average annual rainfalls of 274 mm (Loxton Post Office, 1914-1983) and 334 mm (Karoonda Post Office, 1914-1983) respectively. Both sites were of gently undulating topography with trials near the bottom of slopes. Experiments in 1983 were undertaken at Palmer and Strathalbyn in soils found to have a high level of natural inoculum of G.g.t. in assays.

Experiments at Palmer were conducted on two adjacent soil types, a calcareous sandy loam (Gc1.22) on a slope, and a sand over clay (Dr4.63: Northcote, 1979), at the foot of the slope. Average annual rainfall at Palmer is 343 mm (farm homestead, 1932-1982). Experiments at Strathalbyn were in a sand over clay (Dg4.81: Northcote, 1979) on flat topography. Average annual rainfall at Strathalbyn is 445 mm (farm

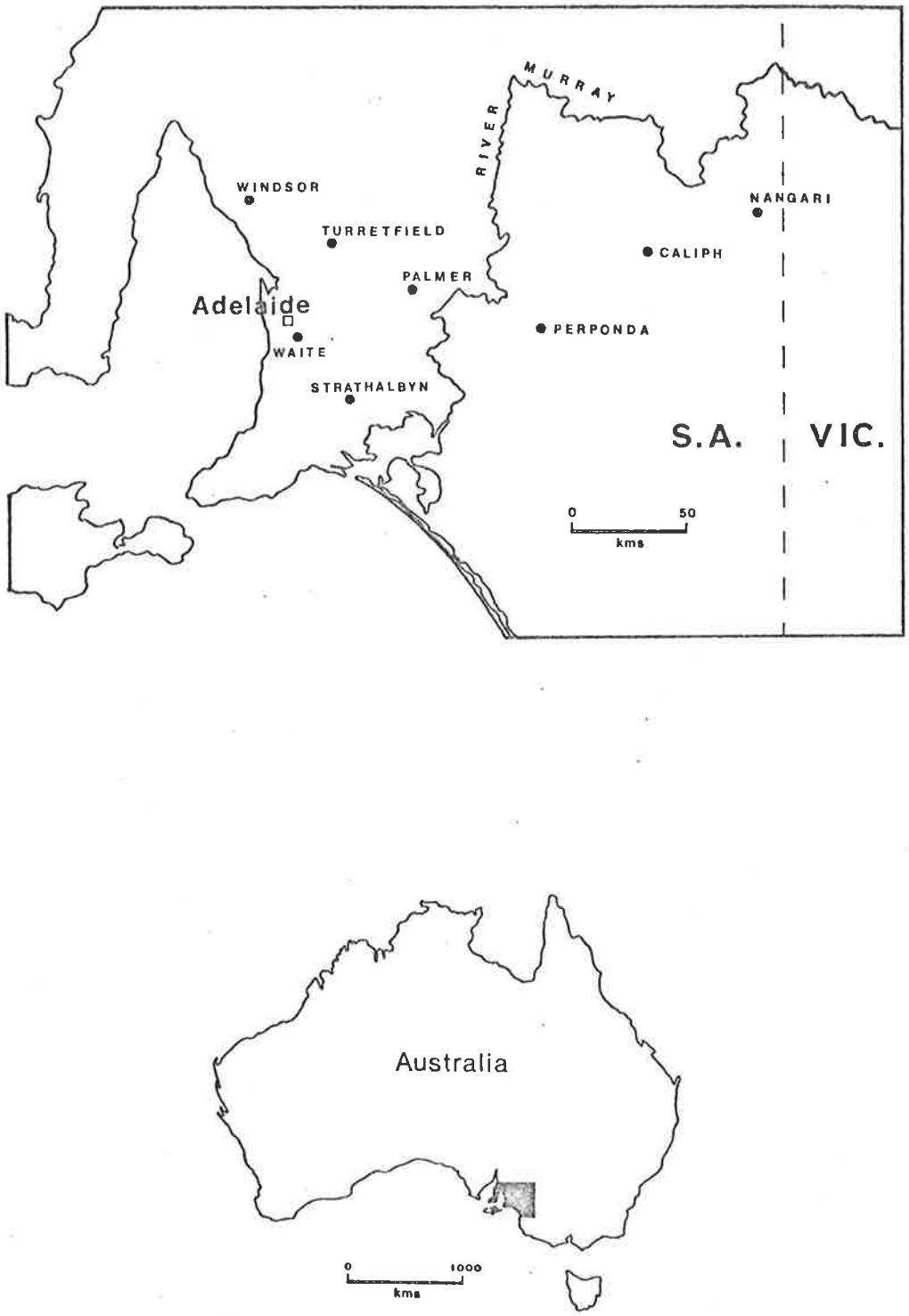

FIGURE 1: Location of experimental sites in South Australia and origin of isolates of G.g.t.

homestead, 1962-1982).

Wheats were grown in experiments according to the cultural practises of local farmers. Wheat is typically grown at Nangari, Perponda and Palmer after 18 months of grassy pasture and six months of bare fallow. The soil is worked after the first rains of autumn and sown with grain and fertilizer (2:1 by weight of superphosphate and ammonium sulphate at 140 $kg.ha^{-1}$) when germination is assured in late April or early May. At Strathalbyn wheat is grown in rotation with clover for seed and clover and grass pasture. The soil is first worked two weeks before being sown with grain in June and therefore several weeks after the opening rains. Fertilizer is broadcast before sowing (superphosphate, 90 $kg.ha^{-1}$).

Plots differed in size in different experiments. At Nangari and Perponda plots were four drill rows x 20 m and were originally intended to measure yield of grain. Smaller plots were employed to measure the incidence of disease at Palmer and Strathalbyn and were four drill rows x 3.5 m except for families of set three of which seed was limited and plots were one drill row x 2.5 m. Seed was sown five cm deep at Nangari, Palmer and Perponda and two cm deep at Strathalbyn with equipment designed to sow small plots of cereals at a rate (70 $kg.ha^{-1}$) similar to that of adjacent crops. Dates of sowing, sampling and harvesting of experiments are given in Table 3. Mean monthly rainfalls of the nearest official recording stations to sites are presented in Appendix 2 with monthly rainfalls for years in which experiments were conducted.

TABLE 3: Dates of sowing, sampling, scoring deadheads and harvesting field experiments

Experiment		Sowing	Sampling		Scoring deadheads	Harvesting
Cultivars	Site		tillering	anthesis		
set 2	Palmer	5/5/83	12/7/83	13/9/83	22/10/83	1/12/83
	Strathalbyn	1/6/83	20/7/83	21/9/83	25/10/83	22/12/83
set 3	Palmer	5/5/83	21/6/83	31/8/83	20/10/83	1/12/83
set 4	Nangari	5/6/81	-	-	30/10/81	15/12/81
	Palmer	5/5/83	29/6/83	8/9/83	22/10/83	1/12/83
set 5	Nangari	5/6/81	-	-	30/10/81	15/12/81
	Perponda	16/6/81	-	-	29/10/81	10/12/81
	Strathalbyn	1/6/83	21/7/83	21/9/83	-	22/12/83

Controlled Environment Studies

(1) Inoculation with G. graminis var tritici and maintenance of seedlings.

Throughout the controlled environment studies a single method was employed to infect seedlings with G.g.t. Seedlings were inoculated with the pathogen following the method of Garrett (1936) and maintained at constant temperature in plastic cups in a waterbath. Seed from a common source and of equivalent weight was surface sterilised in a 1% available chlorine solution of sodium hypochlorite for 20 min, rinsed in sterilised distilled water and pre-germinated aseptically on moistened sterilised filter paper for 24 h at 25°C. Single pre-germinated seeds were placed on an inverted inoculum disc, 14 mm in diameter, cut from the edge of an actively growing colony on one-sixth strength potato dextrose agar. Although agar media are slightly toxic to seedlings of wheat (Rovira, pers. comm.) and infected agar is an artificial source of inoculum, this method of inoculation allows control of the nutritional status and inoculum potential of the pathogen. Four seed and inoculum discs were placed in each cup (300ml) three quarters filled with sterilised coarse (<2mm) river sand and covered by a further 1 cm of sterile sand. Distilled water was added to saturate the bottom 2 cm of sand, and the cups placed in a waterbath at 15°C and watered at 4-day intervals with 5 ml of distilled water per cup. At harvest roots of seedlings were gently washed free of sand and until scored, were preserved in 2% formaldehyde at 5°C.

(2) Assessment of infection

The first formed seminal root was scored for symptoms of infection with G.g.t. and for colonisation of tissue by hyphae. As roots were inoculated close to the scutellar node, symptoms of infection were distal to the inoculum disc and were scored at 50x magnification under a binocular microscope. The following measurements were made (Fig. 2).

(a) The number of stelar lesions (LN)

(b) The largest stelar lesion ranked for length (LL)

(c) The mean of all stelar lesions ranked for length (ML)

(d) The length of root with the cortex discoloured brown (CL) and intensely discoloured brown (CI)

(e) The extent of hyphal growth (mm) down the exterior of the root (ECTG).

Infected roots were then sectioned transversely by hand 2 mm below the inoculum disc, and stained for 10 min in 0.1% aqueous trypan blue and mounted in poly-vinyl alcohol (Omar, Bolland & Heather, 1979). Hyaline hyphae stained dark blue and host tissue pale blue or yellow brown. Sections were examined at 200x magnification and the percentages of the following cells that contained hyphae were recorded.

(f) Cortical cells adjacent to the endodermis (CCOL)

(g) Endodermal cells (ECOL)

(h) Stelar cells bounded by the endodermis (SC)

Field Studies

(1) A bioassay of inoculum of G. graminis var tritici

Sites suitable for experiments were selected for a

FIGURE 2: Symptoms of infection with G.g.t. in seminal roots of wheat and their measurement

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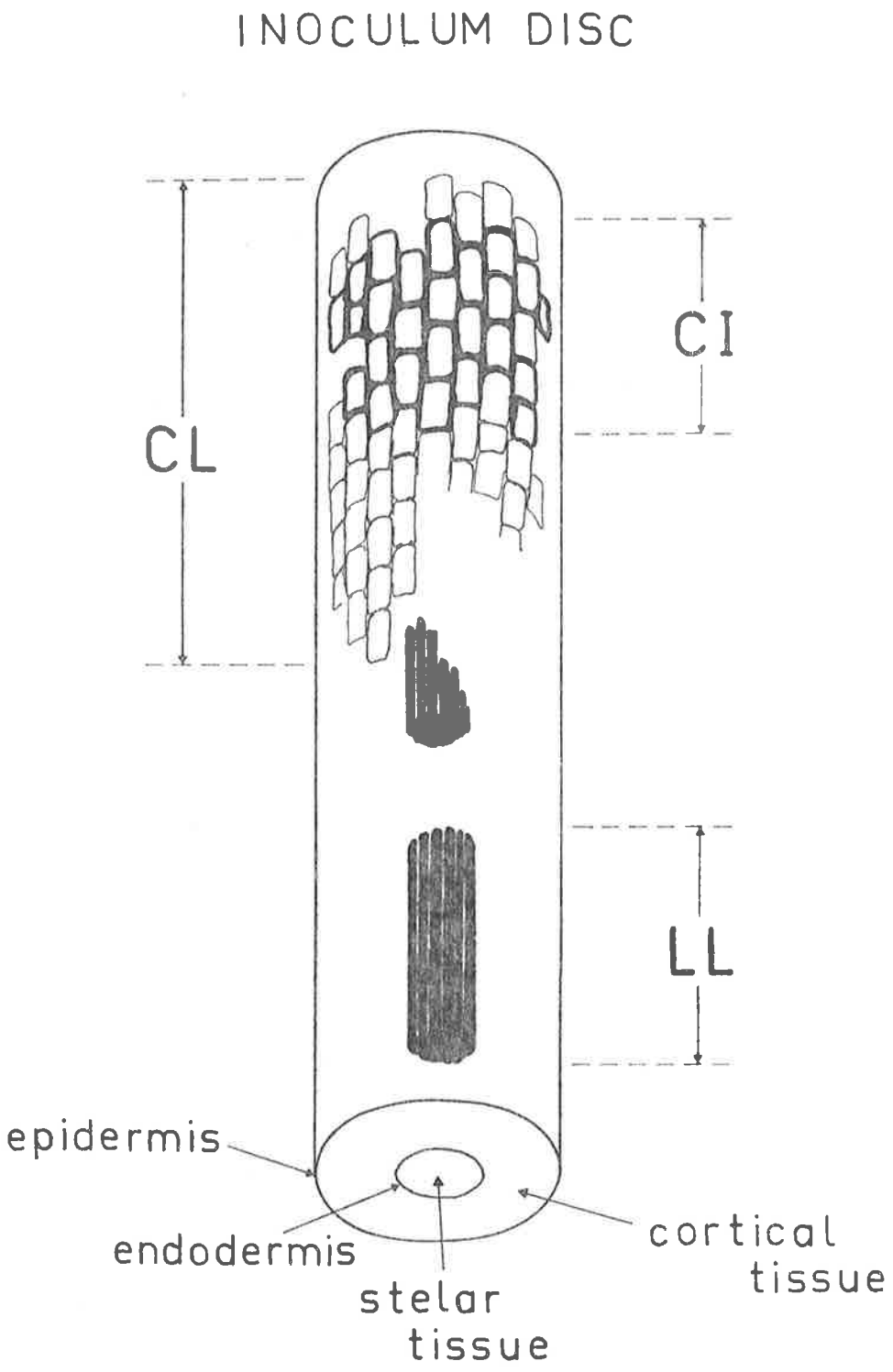
LL Length of the largest stelar lesion

Stelar lesion	Rank
none	0
indistinct	1
LL ⩽ 0.5 mm	2
0.5 mm < LL ⩽ 1.0 mm	3
LL > 1.0 mm	4

CL Length of root with the cortex discoloured brown or

CI intensely discoloured brown

Brown discoloration	Rank
none	0
(CL/CI) ⩽ 2.0 mm	1
2.0 mm < (CL/CI) ⩽ 4.0 mm	2
4.0 mm < (CL/CI) ⩽ 6.0 mm	3
(CL/CI) > 6.0 mm	4


INOCULUM DISC
CL
CI
LL
epidermis
endodermis
stelar tissue
cortical tissue
ROOT TIP

naturally high incidence of inoculum of G.g.t., in fields to be sown with wheat in the coming season. Assays for inoculum of G.g.t. were conducted in January and employed a baiting technique similar to that of Hornby (1969). Twenty samples of the topmost 10 cm of soil were taken from each site. From each sample, seven subsamples of 30 gm each were placed in containers of P.V.C. conduit (2.6 cm internal diameter and 12.5 cm long) one third filled with washed sterilised sand (120° C for 60 min). A further 2 cm of sterile sand was added to conduits which were then placed on trays containing a 3 cm layer of potting soil, and each conduit moistened with 30 ml of sterile distilled water. Even sized seeds of Warigal were surface sterilised (1% active chlorine, for 20 min), washed in sterile water and pre-germinated aseptically. Seed were placed singly in conduits in a growth cabinet. Soil in conduits was kept moist by the addition of 10 ml of distilled water at 5-day intervals. Seedlings were harvested at four weeks and scored for stelar blackening. The incidence of inocula in each sample of soil was expressed as the number of seedlings of seven with symptoms of infection with G.g.t. Sites with the highest levels of inoculum in soil samples, were selected for the location of experiments in 1983.

(2) Pathogens of roots infecting T. aestivum in field experiments

The pathogens responsible for damaging roots were identified from symptoms of infection, and were only recorded for the topmost 6 cm of roots. Three distinct symptoms of

pathogenic infection were commonly observed in field experiments. Symptom of infection with G.g.t. in roots was a blackened stele with an intact cortex. Coronal roots that were infected with the pathogen did not always have blackened steles, but cortical tissue was then discoloured brown or black, with dark runner hyphae present. The pathogen was readily isolated from fresh roots. The symptom of infection with Rhizoctonia solani Kuhn was a flaccid water soaked appearance of cortical tissue, which may have eventually rotted away leaving the stele either discoloured brown or unchanged. The stele may have eventually broken away to leave a pointed stub (Samuel & Garrett, 1932). The characteristic hyphae of R. solani were often but not always visible.

A symptom of infection not typical of G.g.t. or R. solani was an irregular constriction of roots that were discoloured yellow (Fig. 3). Tissues of affected roots were not differentially rotted or discoloured. While Pythium irregulare Buisman was a pathogenic fungus isolated from such affected segments of roots (P. Pittaway, pers. comm.), the causal organism was not investigated further as it was clearly not G.g.t. Other symptoms of infection in roots were rare, occurred in short segments of root and were not recorded.

(3) Assessment of Disease

The course of root disease was determined at sites by observing the influence of infections on the growth and yields of plants sampled from within experiments. Wheats were sampled at tillering and anthesis (Table 3) by the random selection of single plants from within even sized subdivisions of sown plots.

FIGURE 3: Roots of wheat showing symptoms of infection with an unidentified pathogen at anthesis at Strathalbyn

The following measurements were made of plants.

(a) The number of seminal roots (SEM)

(b) The percentage of seminal roots infected with G.g.t. (GSEM)

(c) The percentage of seminal roots infected with R. solani (RSEM)

(d) The number of coronal roots (COR)

(e) The percentage of coronal roots infected with G.g.t. (GCOR)

(f) The percentage of coronal roots infected with R. solani (RCOR)

(g) The percentage of coronal roots infected with the unidentified pathogen (OCOR)

(h) The length (cm) of the subcoronal internode (SCI)

(i) The length (cm) of blackened subcoronal internode (GSCI)

(j) The number of tillers (TIL)

(k) The weight of oven dried shoots (120° C for 24 hr) (DW)

The number of dead and empty heads of wheat (DH) and the number of fertile tillers (NFT) were recorded in plots near ripening (Table 3). After harvest plots were scored for yield of grain (PYD) and weight of a random sample of 100 grains (GRW)

Statistical analyses

Experiments in cups were of completely randomised design and in field trials were of randomised complete

block design and both were analysed by analysis of variance. Though some variates were not continuous, equivalent non-parametric statistical procedures are not available or universally accepted. Where necessary transformations were conducted on data to remove heteroscedasticity in residual terms, as assessed by plots of fitted versus residual values. Where interaction terms were tested, analyses were conducted and presented without transformation, but where applicable, data were transformed and reanalysed for comparison with original findings. As differences between treatments within experiments were more important to the interpretation of findings than differences between individual treatments, results of analyses were summarised in tables as levels of significance.

Tests of association were conducted extensively to test effects not under experimental control. While not as rigorous in interpretation as ANOVA's, these tests were useful in indicating effects within data. Though some variates were not continuous, associations were tested with product moment correlation coefficients, which enabled the use of procedures not available with non-parametric tests of association. Associations were determined within cultivars to exclude effects due to differences between wheats. Differences between cultivars in associations were tested and when not significant, associations were pooled over all wheats and tested for significance (Snedecor & Cochran, 1967). As these procedures become unreliable when the number of associations to be pooled increases, correla-

tions were over all individuals and not within families for data of wheats of set 3. Moreover, disease was not uniformly expressed over the experimental area of wheats of set 3 at Palmer, so that associations were tested and pooled from within replicates.

Unless stated otherwise, a probability of five percent ($P<0.05$) was adopted throughout for significance of statistical tests.

CHAPTER 3

STUDIES IN A CONTROLLED ENVIRONMENT

Experiments in controlled environments have not given consistent results on the resistance of cultivars of wheat to infection with G.g.t. (Scott, 1981). Inconsistent findings may be due to ambiguous definitions and measures of resistance, and to effects related to the specific environmental conditions in which wheats are infected. It is therefore necessary to directly measure infection in roots or score characters that indicate resistance to colonisation in the host's tissues, and to infect wheats with G.g.t. in conditions comparable to those in the field.

G.g.t. is most damaging when colonising the vascular tissue of roots of its hosts. Thus resistance to radial invasion by hyphae into roots of wheats was of greatest interest, though the spread of hyphae along the surface of roots was also considered. The relationships between radial invasion by hyphae into roots and symptoms of infection with G.g.t. were studied in preliminary experiments. Further studies examined conditions in which to infect wheats with G.g.t.

Use of symptoms to measure infection of roots with G. graminis var tritici

The extent of radial invasion into roots is most directly measured by observing hyphae in cells in sections. However, the method is laborious and radial invasion more simply measured using symptoms that are host responses to infection. To effectively measure infection in roots, symptoms must be

closely related to colonisation of tissue by hyphae. In previous studies roots have been scored when colonisation was extensive and host tissue necrotic. However, necrotic symptoms of infection with G.g.t. are not distinct and do not delineate colonisation by the pathogen. Further, symptoms of infection that are host responses to invasion by the pathogen, only occur in living cells and can only delineate infection of tissue that is incomplete. To more accurately measure the extent of colonisation with G.g.t., symptoms that are host responses to infection were investigated before roots were extensively colonised. In particular, two major symptoms of infection, blackening of the stele (Fellows, 1928) and the brown discoloration of the cortex (Holden, 1976) have been studied here.

(1) Stelar blackening

Stelar lesions have been frequently used to measure the extent of infection with G.g.t. in roots of wheat (Nilsson, 1969). However, their failure to indicate the extent of colonisation of distal segments of roots in some studies (Deacon & Henry, 1978), has caused doubt of their value as measures of infection (Brown, 1981). Gilligan (1980a) suggested stelar blackening requires a supply of translocates which is lost when hyphae invade the vascular tissue in the proximal segment of a root. This hypothesis can account for the findings of Deacon and Henry (1978). However, stelar blackening should still occur distally to a point of infection if translocation is not completely

disrupted, or proximally where inoculation has been at a distance from seed. Therefore it is premature to dismiss stelar lesions as useful indicators of the colonisation of roots of wheat with G.g.t., especially in the field where roots are generally infected some distance from the seed.

To study the association between the early colonisation of tissues with hyphae of G.g.t., and the formation of small black stelar lesions in lightly infected roots, seedlings of the cultivars Aus1080, Condor and Kite were infected with either isolate 19 or 201 and harvested at ten days. Sections through small black stelar lesions (<1 mm in length) were prepared and observed at 200x magnification. Blackening was seen only in stelar and endodermal cells. Affected areas were bounded by the endodermis and overlain by extensively colonised cortical cells (Fig. 4). However, hyphae of G.g.t. were also observed in stelar tissue without blackening of cells. These observations suggest stelar lesions form when the endodermis is breached by vigorously growing hyphae of G.g.t. and that lesions increase in size with further colonisation of the stele (Robertson, 1932), subject to a supply of translocates. Moreover, the several stelar lesions which develop in a root inoculated with G.g.t. at a point, probably reflect independent sites of stelar invasion by hyphae growing from above the endodermis (Deacon & Henry, 1978). Thus the general rate of radial growth of hyphae into a segment of root, and resistance to that growth, will be reflected in the number and size of stelar lesions that are scored before impairment of translocation through the

FIGURE 4: Stelar lesions in séminal roots of wheat infected with G.g.t. in cups for 10 days

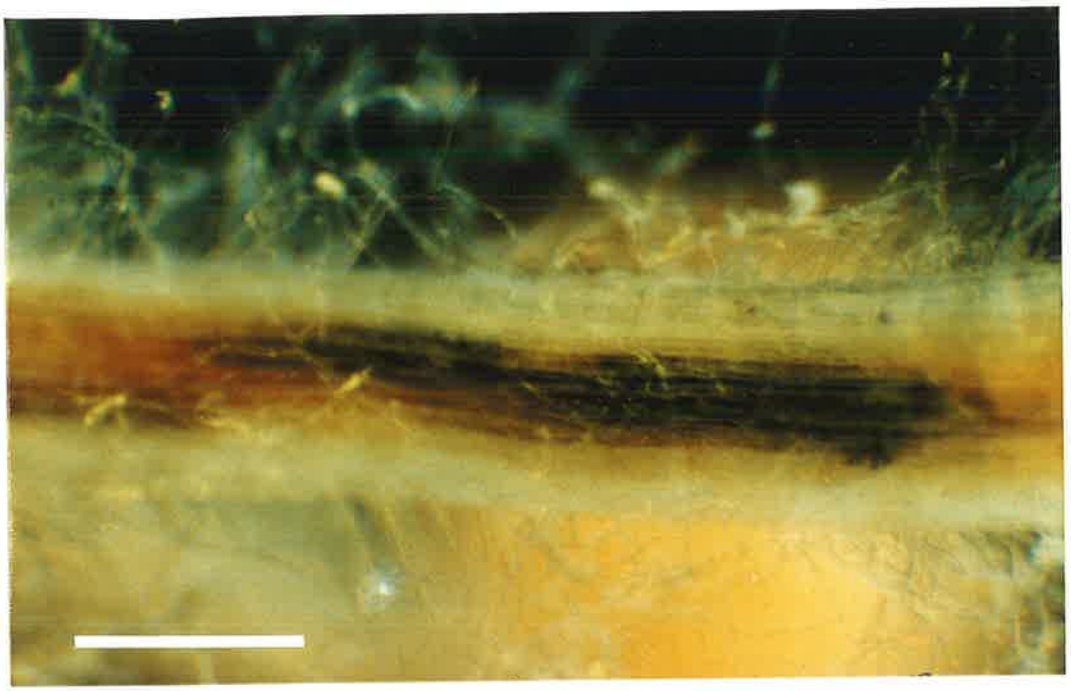
A. An exterior view

(Scale bar = 1 mm)

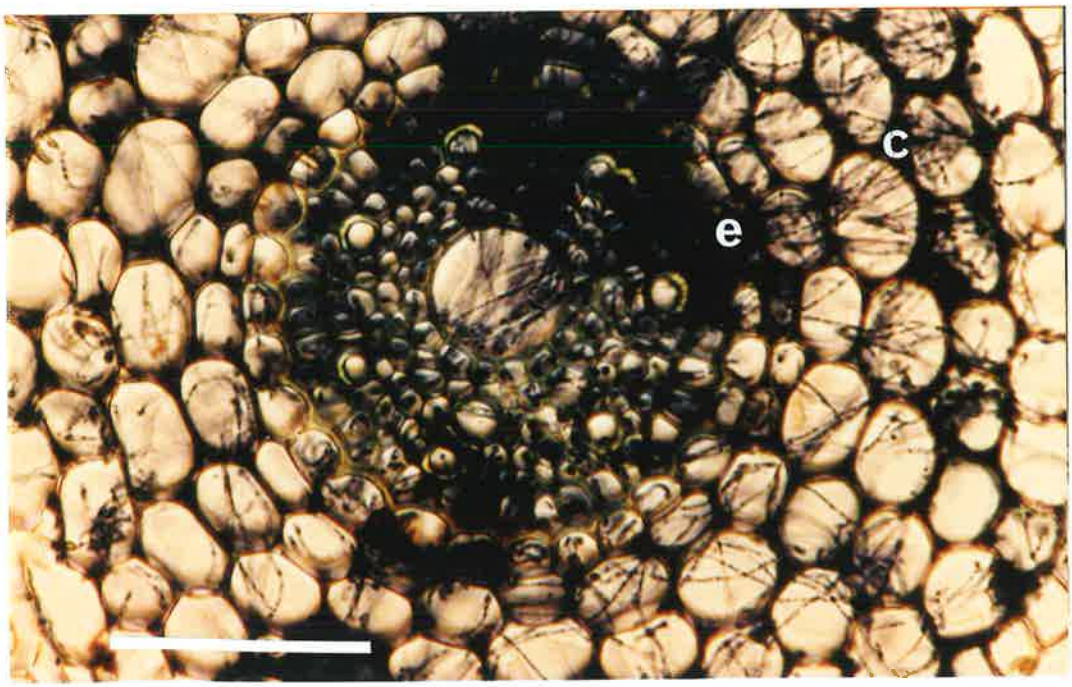
B. A transverse section showing the endodermis (e) and extensively colonised cortical tissue (c)

(Scale bar = 0.2 mm)

A



B



proximal portion of root. In data published by Gilligan (1980a), stelar blackening was not observed in steles colonised with G.g.t. until about nine days after inoculation, which was supported by observations in the current study. Since Gilligan inoculated tips of roots as in the present study, blackening may have been inhibited till nine days by either the immaturity of hyphae or, more likely, the immaturity of the stelar tissue of the host. Thus, for black lesions to reflect the rate of colonisation of steles of roots, observations must be made at least nine days after inoculation but before infection of proximal segments of roots is extensive.

(2) Brown discoloration of the cortex

The brown discoloration of the cortex of roots infected with G.g.t. has often been overlooked. Holden (1976) observed the discoloration in the topmost 4 mm of seminal roots and in all but the outermost cells of the cortex.

The development of the brown discoloration of the cortex was studied over time by infecting seedlings of the cultivars Aus1080, Condor and Kite with isolate 201 of G.g.t., and harvesting at seven, ten and thirteen days. The length of the first seminal root that was discoloured brown was measured at 50x magnification (Fig. 2). Thirteen replicates were employed for each treatment and data analysed by two factor analysis of variance (Table 4). Discoloration did not vary with day of observation, while significant differences between cultivars ($P<0.001$) were

TABLE 4: Effect of infection with G.g.t. isolate 201 on cortical browning (as ranks, page 17) in the first three seminal roots of three cultivars of wheat at 7, 10 and 13 days

Cultivar	Day			ANOVA (P)		
	7	10	13	cultivar	day	interaction
Aus1080	0.90	1.00	1.42	0.001	n.s.	n.s.
Condor	0.00	0.17	0.17			
Kite	0.11	0.42	0.00			

data represent means of 13 replicates

detected. The interaction term was not significant.

Sections through the discoloured cortex of roots were also made and stained as before. As observed by Holden (1976), affected cortical sections contained thickened and discoloured walls of cells and intercellular spaces (Fig. 5) which were associated with the immediate presence of hyphae. Walls of cells had been uniformly thickened and discoloured. When infected with more virulent isolates of G.g.t. than 19 or 201 (e.g. isolate 226, Table 8) walls of cortical cells are not discoloured and thickened. These observations suggest discoloration does not occur when the cortex is rapidly invaded.

Overall, findings suggest the thickening and discoloration of walls of cortical cells was a response by the host to infection which occurred as living cells were approached by hyphae. The response had largely occurred seven days after young tissue was infected with the pathogen, but did not occur where cortical cells were quickly killed by vigorous invasion by hyphae. As cortical cells were frequently colonised by hyphae but not discoloured, the discoloration does not delineate infection of the root and therefore cannot be used to measure severity of infection with G.g.t. Nevertheless, the lengths of roots with the cortex discoloured brown were recorded to test the hypothesis that thickening and discoloration retard further infection of cortical tissue.

FIGURE 5: Cortical browning in seminal roots of wheat infected with G.g.t. in cups

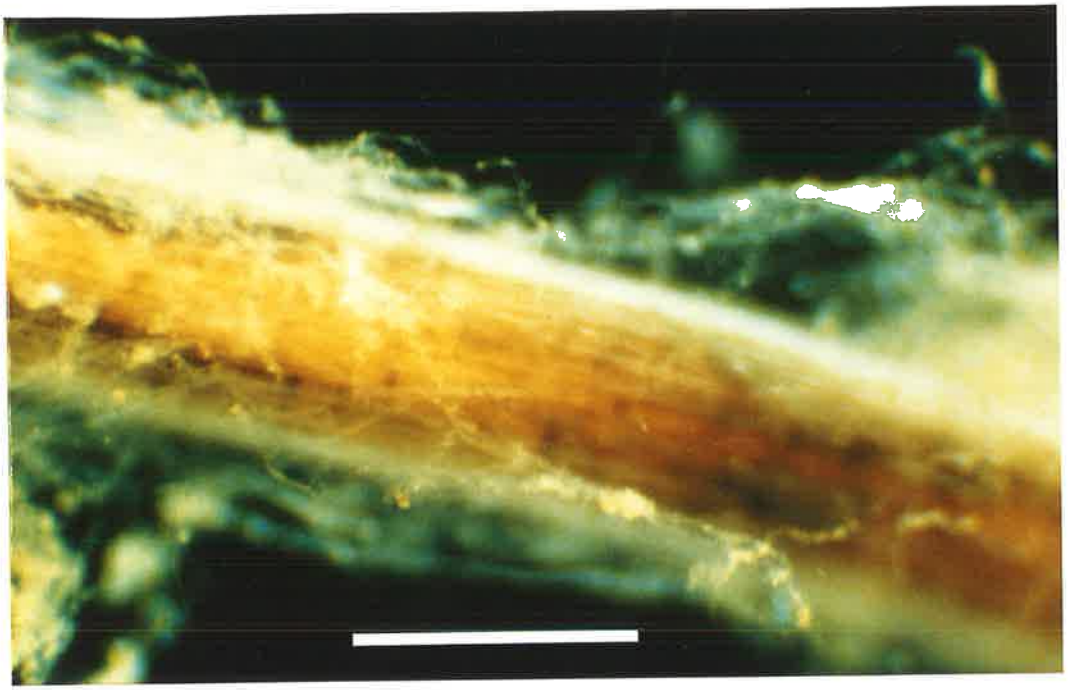
A. An exterior view at 10 days

(Scale bar = 2 mm)

B. A transverse section showing the endodermis (e) and thickened and discoloured walls of cortical cells (t) at 7 days

(Scale bar = 0.1 mm)

A



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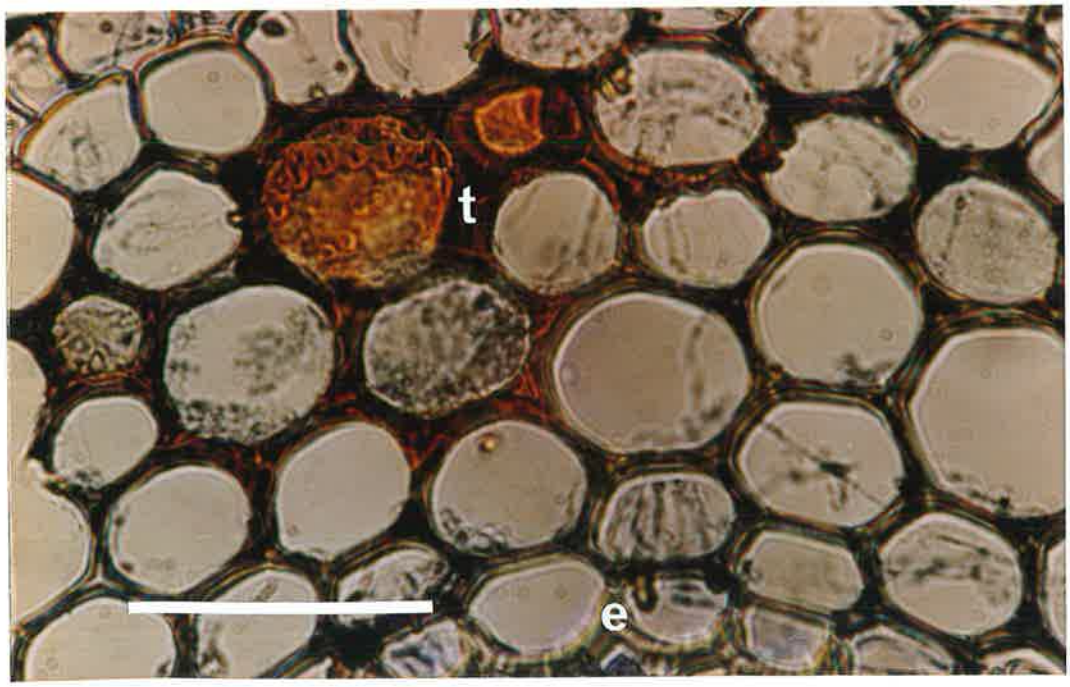


Conditions in which to infect T. aestivum with G. graminis var tritici

In previous studies, seedlings of wheat have been artificially infected with G.g.t. in many conditions (Nilsson, 1969). While infection of wheats in a controlled environment should resemble that in the field, several conditions in the present study were artefacts of the method with which seedlings were infected with G.g.t. These included the agar medium of the inoculum discs, the weight of graded seed for seedlings and the isolate with which they were infected. These conditions do not correspond to those in the field.

In preliminary studies, roots of wheat were more extensively colonised when inoculated with hyphae grown on an agar with the more concentrated of two levels of nutrients (Table 5). In further studies with isolates 19 and 201 of G.g.t., inoculum discs of one sixth strength potato dextrose agar gave a consistent level of stelar infection that was incomplete at ten days and allowed the blackening of steles of roots. Subsequently, all inoculum discs were of one sixth strength potato dextrose agar. Other preliminary experiments tested the influence of weight of grain and effect of isolate on infection of cultivars of wheat.

(1) Weight of seed

As nutrition of seedlings in cups relied solely on reserves in the grain, the effect of weight of seed on infection of seedlings with G.g.t. was examined in detail. While reserve of endosperm has not been reported to influence infection of seedlings with G.g.t., seed is frequently graded for size before experimentation. To test the influence of weight of seed on infection, seedlings of 'Kite' grown

TABLE 5: Effect as inoculum discs, of two dilutions of potato dextrose agar, on percentage colonisation of the first seminal root of Kite infected with G.g.t. isolates 19 and 42 for 8 days

Dilution	Isolate	Colonisation (as angles)	
		inner cortex	endodermis
1:12	19	29.0	15.6
1:24	19	25.4	6.7
t - test (P)		n.s.	n.s.
1:12	42	84.2	40.8
1:24	42	41.8	16.5
t - test (P)		0.01	0.01

data represent means of 8 replicates

from seeds from a single plant and selected for ten levels in weight were infected with isolate 19 of G.g.t. The extent of infection in seedlings was measured by recording the number and size of stelar lesions in the first three seminal roots at 13 days. The experiment employed five replicates and was analysed by least squares regression. Findings are presented in Figure 6 and show weight of grain to influence both the number of lesions in roots ($P<0.001$ for a quadratic association) and the size of the largest lesion in roots ($P<0.01$ for a linear association). While both scores provide evidence that endosperm reserves influence infection of roots with G.g.t., the relationships are dissimilar.

To clarify findings, colonisation by hyphae was measured directly in a further experiment. Seedlings of cultivar Aus1080 were grown from five levels in seed weight, infected with G.g.t. isolate 201 and harvested at ten days. Only the first seminal root was scored. Measurements of infection were restricted to direct observation of hyphae as few stelar lesions were formed. The experiment employed seven replicates, was analysed as previously, and the findings are depicted in Figure 7. The data confirm earlier evidence that weight of grain influenced infection of roots with hyphae of G.g.t. Moreover, a comparison of infection in the innermost layer of cortical cells and endodermal cells (Fig. 7) showed hyphae dissimilarly invaded the endodermis of roots as weight of grain was varied. Regression analyses showed growth of hyphae down roots was favoured by increasing weight of grain (at $P<0.1$), in contrast to growth of hyphae into

FIGURE 6: Effect of weight of seed on number and size (as ranks, page 17) of stelar lesions in the first three seminal roots of Kite infected with G.g.t. isolate 19 for 13 days

Quadratic regression ($P<0.001$)

Linear regression ($P<0.01$)

data represent means of five replicates

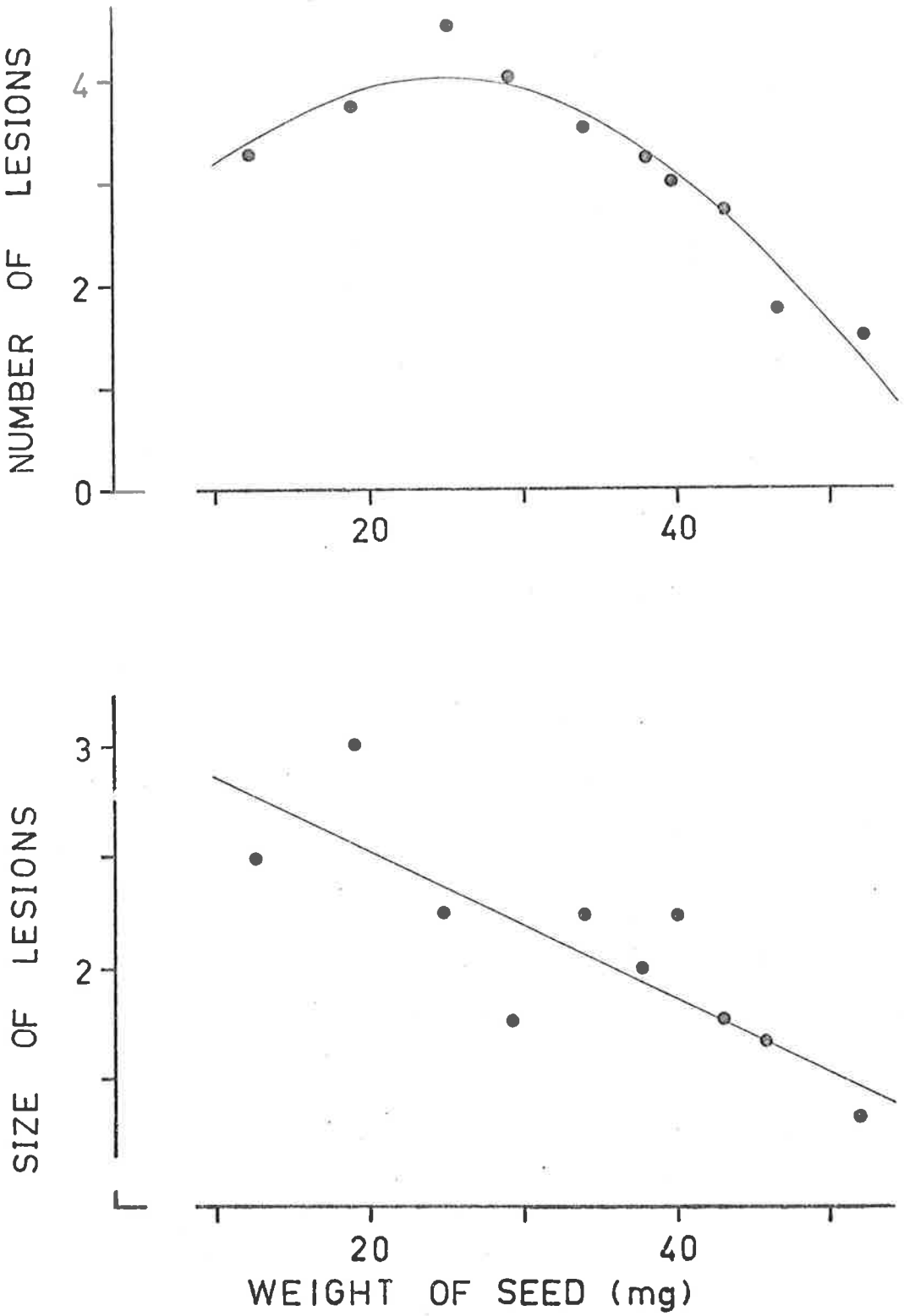
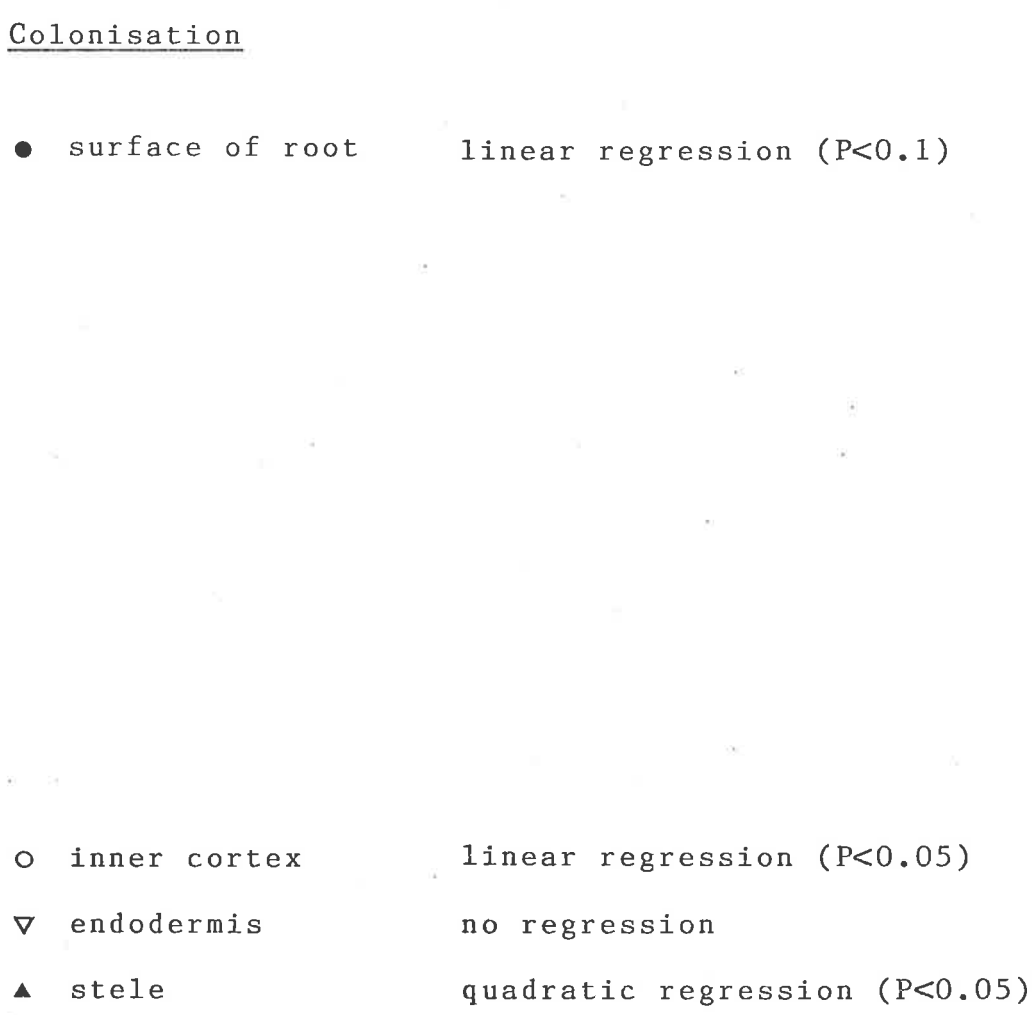
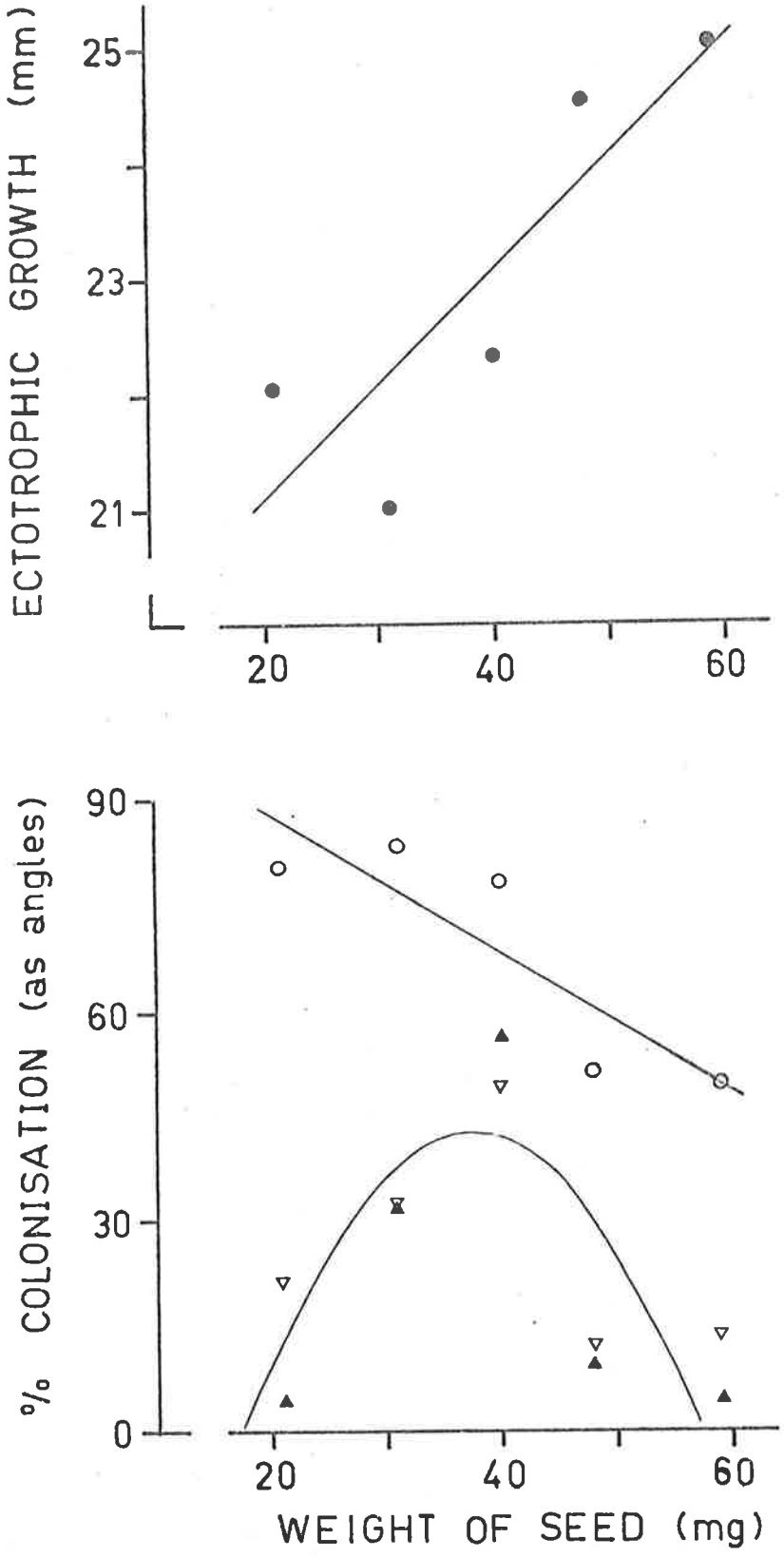

NUMBER OF LESIONS
4
2
0
20
40
SIZE OF LESIONS
3
2
20
40
WEIGHT OF SEED (mg)

FIGURE 7: Effect of weight of seed on colonisation of the first seminal root of Aus1080 infected with *G.g.t.* isolate 201 for 10 days



data represent means of 7 replicates


ECTOTROPHIC GROWTH (mm)
25
23
21
20
40
60
% COLONISATION (as angles)
90
60
30
0
20
40
60
WEIGHT OF SEED (mg)

the cortex of roots (Fig. 7),which decreased as weight of grain was increased. Therefore, these findings suggested that the number of stelar lesions in roots reflected colonisation of the stele with G.g.t. more accurately than did size of the largest lesion.

In order to place in perspective experiments testing differences between wheats in infection with G.g.t., the effect of weight of grain on infection in two cultivars was studied. Thus, seedlings of cultivars Aus1080 and Condor were grown from grain of two weights, infected with G.g.t. (isolate 201) and harvested at ten days. The first formed seminal root of seedlings was scored for stelar lesions and colonisation by hyphae. The experiment employed six replicates. Findings are presented in Table 6 and show cultivar x seed weight interaction terms to be significant for all measurements of hyphal infection in roots except colonisation of the innermost cortical cells. This shows weight of seed to have dissimilarly influenced the infection of roots of the two cultivars. As interaction terms were significant for colonisation of the endodermal cells but not the adjacent innermost cortical cells (Table 6), weight of seed is again shown (Fig. 7) to have dissimilarly influenced growth of hyphae across the endodermis. Thus seed was arbitarily selected from a narrow range in weight (32 to 40 mg) to minimise error variation in subsequent experiments. Though findings obtained from an experiment with one weight of seed may be unique, underlying resistance in cultivars to infection with G.g.t. may still be tested by association with field data.

TABLE 6: Effect of weight of seed on number and size (as ranks, page 17) of stelar lesions and extent of colonisation in the first seminal root of two wheats infected with G.g.t. isolate 201 for 10 days

	Weight of seed				ANOVA (P)		
	32-40 mg		45-55 mg				
	Cultivar						
	Aus1080	Condor	Aus1080	Condor	cultivar	weight	interaction
stelar lesions							
Number	1.00	1.67	0.50	0.80	n.s.	n.s.	n.s.
Size of largest	1.75	2.60	1.01	1.50	0.05	0.05	n.s.
extent of colonisation							
Ectotrophic growth (mm)	27.8	27.3	27.0	16.8	0.01	0.01	0.05
Inner cortex (%)	88.1	92.3	86.8	78.0	n.s.	n.s.	n.s.
Endodermis (%)	17.6	71.0	34.5	26.6	n.s.	n.s.	0.05
Stele (%)	7.5	61.7	28.3	8.3	n.s.	n.s.	0.01

data represent means of six replicates

(2) Source of isolate

Wheats were infected with a single isolate of G.g.t. when grown in cups but are infected with a diverse population of the pathogen in the field. Thus, the extent to which experiments could be related to those in the field, was investigated by observing the consistency with which cultivars of wheat were infected with isolates of G.g.t. While previous studies had not found isolates of G.g.t. to differentially infect cultivars of wheat (Nilsson, 1969 and Mattsson, 1973), the current study employed different methods to those of Nilsson and Mattsson with which to inoculate and maintain seedlings, and different methods for measuring infection. Thus the consistency with which some South Australian isolates of G.g.t. had first infected roots of wheat was tested by infecting the eight cultivars of set 1 with isolates 19, 108 and 201, which differ in geographic origin (Fig. 1). The cultivars of set 1 had been found to differ in severity of infection with isolate 19 in preliminary experiments. Seedlings were harvested after 13 days and the extent of infection recorded by measuring the number of stelar lesions and the rank (Fig. 2) of the largest stelar lesion in the first three seminal roots. The experiment was of randomised complete block (isolate) design with 14 replicates and was analysed with two factor analysis of variance. Findings are presented in Table 7, and show cultivars to be consistent in the number of stelar lesions in roots but not in the size of the largest stelar lesion ($P<0.01$), when infected with different isolates of G.g.t. While scoring the size of the largest stelar lesion

TABLE 7: Effect of infection with G.g.t. isolates 19, 108 and 201 on number and size (as ranks, page 17) of stelar lesions in the first three seminal roots of cultivars of set 1 at 13 days

Isolate	Cultivar								ANOVA (P)		
	Aus1080	Condor	Festiguay	Halberd	Kite	Kite4a/2r	Rac311	Warigal	cultivar	isolate	interaction
				Number of stelar lesions							
19	5.0	9.2	7.3	7.5	5.9	7.8	10.2	7.6			
108	2.6	5.4	4.1	3.2	3.1	2.3	6.1	4.8	0.001	0.001	n.s.
201	3.3	9.2	5.9	6.7	5.5	4.6	8.3	6.5			
				Size of the largest stelar lesion							
19	2.0	3.2	2.9	2.6	2.6	3.0	3.1	2.5			
108	1.8	2.8	2.1	1.9	1.5	1.3	2.4	2.1	0.001	0.001	0.01
201	1.6	3.4	2.6	2.7	2.5	2.5	3.2	3.0			

data represent means of 14 replicates

provides evidence that cultivars were dissimilarly infected with G.g.t., this measurement was previously less reliable in indicating the extent of invasion of the stele than the number of stelar lesions in roots. On balance these findings indicate cultivars were similarly infected with the three isolates of G.g.t., supporting earlier work. Evidence was not found for effects of isolates to cause wheats to be dissimilarly infected with G.g.t. in cups and in the field.

Infection of wheats with G. graminis var tritici

Experiments were conducted to test differences between wheats in infection with G.g.t. Studies were restricted to wheats that had previously differed most in extent of colonisation when infected with isolate 19 of G.g.t., i.e. with the cultivars of set 2. Cultivars of set 3 were also used in a study of inheritance of susceptibility to infection by the pathogen.

(1) Differences between cultivars in infection

Cultivars of wheat have not been directly shown to differ in infection with G.g.t. Nevertheless, resistance to infection by the pathogen is thought to be weakly expressed in wheat (Scott, 1981), particularly where the pathogen is not highly virulent. Thus, cultivars of set 2 were infected with two isolates (201 and 226) of G.g.t. differing in virulence, to test the hypothesis that resistance to infection is influenced by the virulence of the pathogen. Clean seed of cultivars was obtained from glasshouse grown plants.

Seedlings were harvested when hyphae began entering the stele of the first seminal root adjacent to the inoculum

(6 days for 226 and 10 days for 201), and scored for infection of roots and blackening of the stele. The experiment employed six replicates for infection with 226 and eight replicates for infection with 201 and the results are presented in Table 8. Statistical terms for differences between isolates have no meaningful interpretation, since seedlings infected with different isolates were harvested at different times. Nevertheless, hyphae of isolate 226 invaded radially into roots more rapidly than hyphae of isolate 201. Ectotrophic hyphae of both isolates appeared to grow equally rapidly down roots since lengths of roots colonised were proportional to times of harvest. Cultivars were found to differ significantly ($P<0.001$) in the percentage of endodermal and stelar cells that were colonised by hyphae of G.g.t. The innermost cortical cells were too extensively colonised to differ between cultivars (Table 8). The absence of significant interaction terms for the colonisation of roots by hyphae do not support the hypothesis that resistance to invasion by hyphae of G.g.t. is influenced by the virulence of the pathogen. Stelar lesions were not observed in roots infected with isolate 226, as seedlings were harvested before blackening could occur. When infected with isolate 201, cultivars differed significantly in the number of stelar lesions in roots ($P<0.001$), which also reflected differences in the colonisation of roots by hyphae (Table 8).

While the number of stelar lesions in roots generally reflected the extent of stelar colonisation in sections, exact correspondence was not noted. For example the cultivar 'Kite' had the least number of lesions in roots, but was the fourth most extensively colonised wheat of set 2

TABLE 8: Number and size (as ranks, page 17) of stelar lesions and extent of colonisation in the first seminal root of cultivars of set 2 infected with G.g.t. isolates 201 (at 10 days) and 226 (at 6 days)

	Isolate	Cultivar							ANOVA (P)		
		Aus1080	Chile909	Chile911	Condor	Kite	Nebraska86	Rac311	cultivar	isolate	interaction
Stelar lesions											
Number	201	0.75	0.87	0.50	0.87	0.25	1.12	2.37	0.001	-	-
Size of largest	201	1.40	1.00	1.00	1.40	1.50	1.80	1.88	n.s.	-	-
Extent of colonisation											
Ectotrophic growth (mm)	201	26.9	27.8	25.7	26.5	24.4	26.9	29.4	n.s.	0.001	n.s.
	226	16.1	14.0	16.8	15.3	14.4	14.1	16.7			
Inner cortex (%)	201	99	98	89	98	93	99	100	n.s.	n.s.	n.s.
	226	98	81	91	98	100	75	94			
Endodermis (%)	201	79	69	57	95	81	88	99	0.001	0.001	n.s.
	226	54	23	33	60	54	63	90			
Stele (%)	201	72	56	50	89	84	88	96	0.001	0.001	n.s.
	226	32	6	17	48	19	61	73			

data for isolate 201 are means of eight replicates and for isolate 226 are means of six replicates

(Table 8). Departures from a closer association may simply reflect the comparing of infection in differing segments of root, as sections were from a region 0-5 mm below the scutellar node, while lesions occurred distally up to 12 mm from this region. Indeed, Robertson (1932) had noted wheat roots to change in resistance to infection by G.g.t. over this region. However, departures from close association may also reflect differences in the time that elapses before steles can blacken when invaded by hyphae of G.g.t. For the example given earlier, findings could suggest stelar tissue of 'Kite' must be older than that of the other cultivars of set 2 before blackening can commence. Thus stelar lesions may most reliably reflect the rate of hyphal invasion into roots of wheats when scored later than was done in the present study, that is after ten days from inoculation with G.g.t.

The results of this experiment showed wheats to differ in infection with G.g.t., and provided evidence for resistance to the pathogen in cultivars of wheat.

(2) Genetic control of factors influencing infection in a wheat cross

Few studies have concerned the genetic control of characters that influence infection of wheats with G.g.t. Mattsson (1973) reported a low level of resistance equal to that of the donor parent (Moscow red seeded) in some lines repeatedly backcrossed to susceptible Swedish cultivars (Prins & Starke), but it was not shown that observations

were not due to chance alone. The genetic control of a character is generally established by observing segregation in the first selfed (F_2) or backcrossed (BC_1) generations of the hybrid of two parental genotypes, and observing the heritability of characters in segregate lines in succeeding generations. To detect evidence for the segregation of characters influencing infection in wheat, clean seed of families of set 3 was obtained from glasshouse grown plants (35 F_2 individuals), germinated, and the seedlings infected with isolate 201 of G.g.t. Seedlings were harvested after ten days and scored as previously. The experiment employed six replicates and included the parents (Aus1080 and Condor) as controls. Findings are presented in Table 9. Where families or parents differed data are also presented as frequency distributions in Figure 8. Parents only differed for colonisation of the endodermis and stele with hyphae of G.g.t., while families of set 3 did not differ for infection of roots with the pathogen. In addition, the distribution of families (Fig. 8) suggested factors that most influence infection of roots were not simply inherited. However, this experiment did not critically examine genetic control of factors that influence infection, since F_2 families contain significant heterogeneity yet the phenotype of each was estimated from only six individuals. In preliminary experiments the wheats Aus1080 and Condor had differed most in infection with G.g.t. and were employed in the present study though evidence in Table 8 shows other crosses may have been more suitable. Insufficient time precluded the use of more carefully selected parents or more advanced families from the cross Aus1080 X Condor in this study.

TABLE 9: Number and size (as ranks, page 17) of stelar lesions and extent of colonisation in the first seminal root of parents and F_2 families of set 3 infected with G.g.t. isolate 201 for 10 days

	Parents		Families			ANOVA (P)	
	Aus1080	Condor	minimum	mean	maximum	parents	families
Stelar lesions							
Number	1.00	1.67	0.33	1.39	2.83	n.s.	n.s.
Size of largest	1.75	2.60	1.00	1.76	2.80	n.s.	n.s.
Extent of colonisation							
Ectotrophic growth (mm)	28.3	27.8	19.6	24.2	27.3	n.s.	n.s.
Inner cortex (as angles)	76.3	81.0	53.8	74.6	88.1	n.s.	n.s.
Endodermis (as angles)	23.1	61.6	11.8	36.6	64.1	0.05	n.s.
Stele (as angles)	11.0	56.9	5.2	29.9	61.0	0.05	n.s.

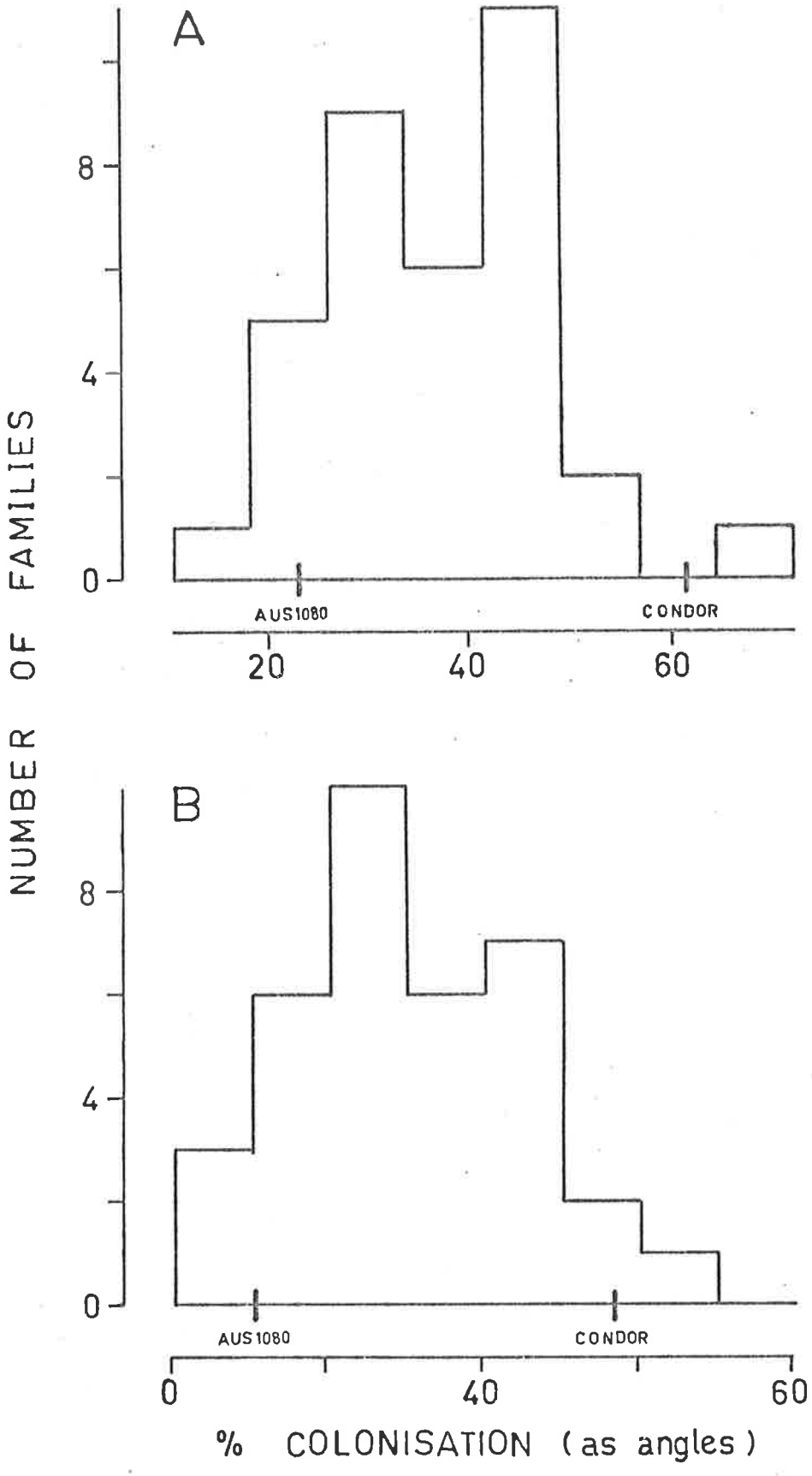
data represent means of 6 replicates

FIGURE 8: Frequency distributions of extent of colonisation in the first seminal root of families of set 3 infected with G.g.t. isolate 201 for 10 days

A. endodermis

B. stele

data obtained with means of 6 replicates


A
B
8
4
0
AUS1080
CONDOR
20
40
60
NUMBER OF FAMILIES
% COLONISATION (as angles)

Study of the brown discoloration of the cortex in roots infected with G. graminis var tritici

Additional aspects of the cortical browning of roots infected with G.g.t. were examined, as this character may condition the resistance of wheats to further infection. The data were gathered from previous experiments.

(1) Influence of weight of seed

The extent of general and intense cortical browning down roots was significantly and positively associated with weight of seed in previous experiments (Fig. 9). The extent of cortical browning down roots may be influenced by the extent of ectotrophic growth of hyphae on the surface of roots which is also positively associated with weight of seed. However the influence of weight of seed is far stronger on cortical browning than on ectotrophic growth of hyphae (Figs. 7 and 9), which suggests discoloration is a host response to infection by G.g.t. that consumes energy.

(2) Genetic control of discoloration in a wheat cross

Both parents and families of set 3 differed significantly in extent of the brown discoloration of the cortex (Table 10), providing evidence for the genetic control of this character. These data are also presented as frequency distributions in Figure 10. Distributions of F_2 families for cortical browning appear bimodal, and are consistent with two phenotypes having segregated. The phenotypes for the intense discoloration occur approximately with a 3:1 ratio that is indicative of single gene segregation with dominance. While more than 200 segregate families are

FIGURE 9: Effect of weight of seed on cortical browning (as ranks, page 17) in the first seminal root of Aus1080 (at 10 days) and Kite (at 13 days) infected with isolate 201 of G.g.t.

A. Aus1080

- ● discoloured — linear regression ($P<0.05$)
- o intensely discoloured — linear regression ($P<0.01$)

B. Kite

- ● discoloured — linear regression ($P<0.001$)

data represent means of 7 replicates for Aus1080 and 5 replicates for Kite

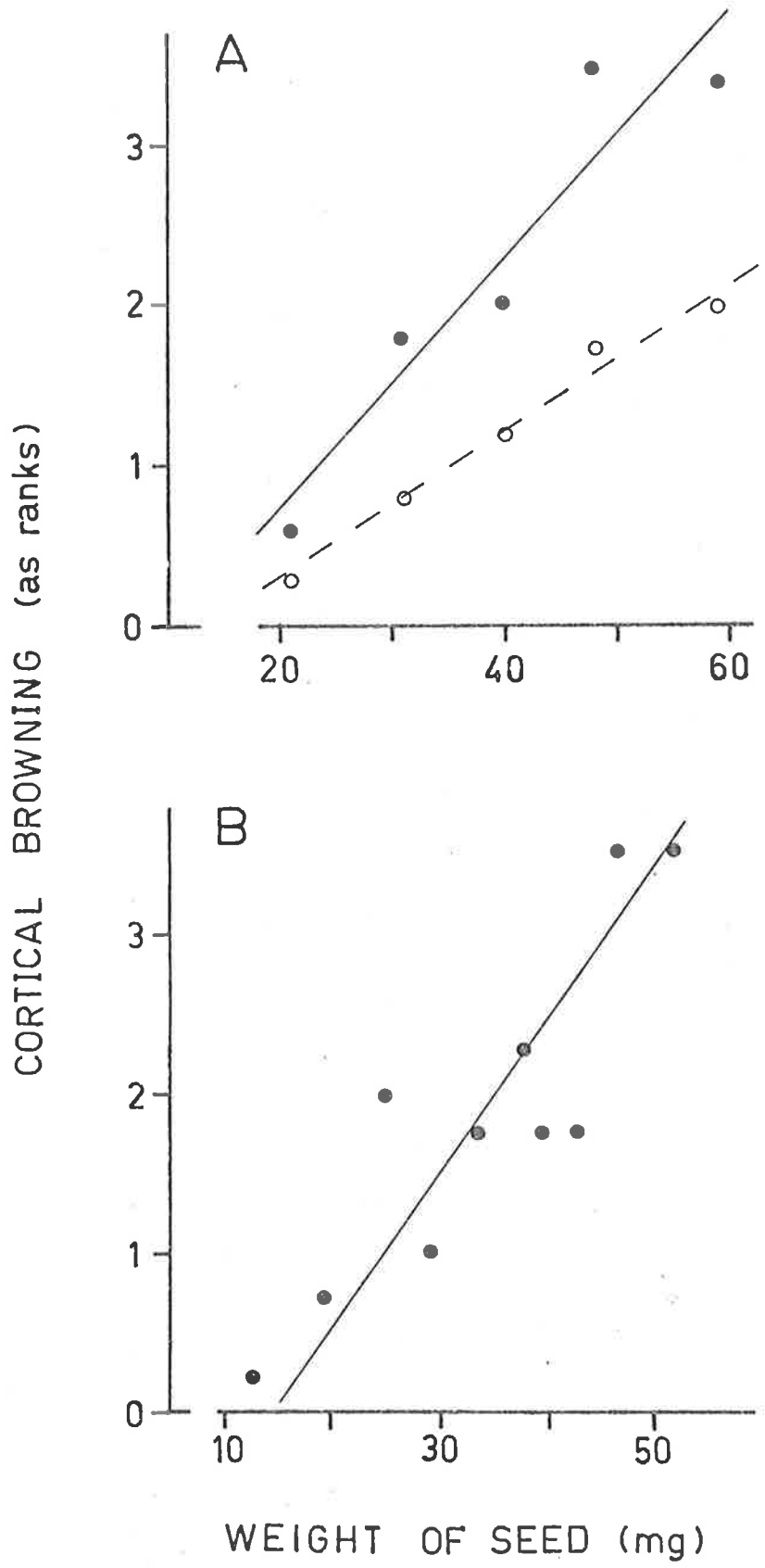

A
B
3
2
1
0
20
40
60
10
30
50
CORTICAL BROWNING (as ranks)
WEIGHT OF SEED (mg)

TABLE 10: Extent of cortical browning (as ranks, page 17) in the first seminal root of parents and F_2 families of set 3 infected with G.g.t. isolate 201 for 10 days

Cortical browning	Parents		Families			ANOVA (P)	
	Aus1080	Condor	minimum	mean	maximum	parents	families
discoloured	3.01	0.24	0.11	0.91	2.44	0.01	0.05
intensely discoloured	1.17	0.00	0.00	0.43	1.50	0.05	0.001

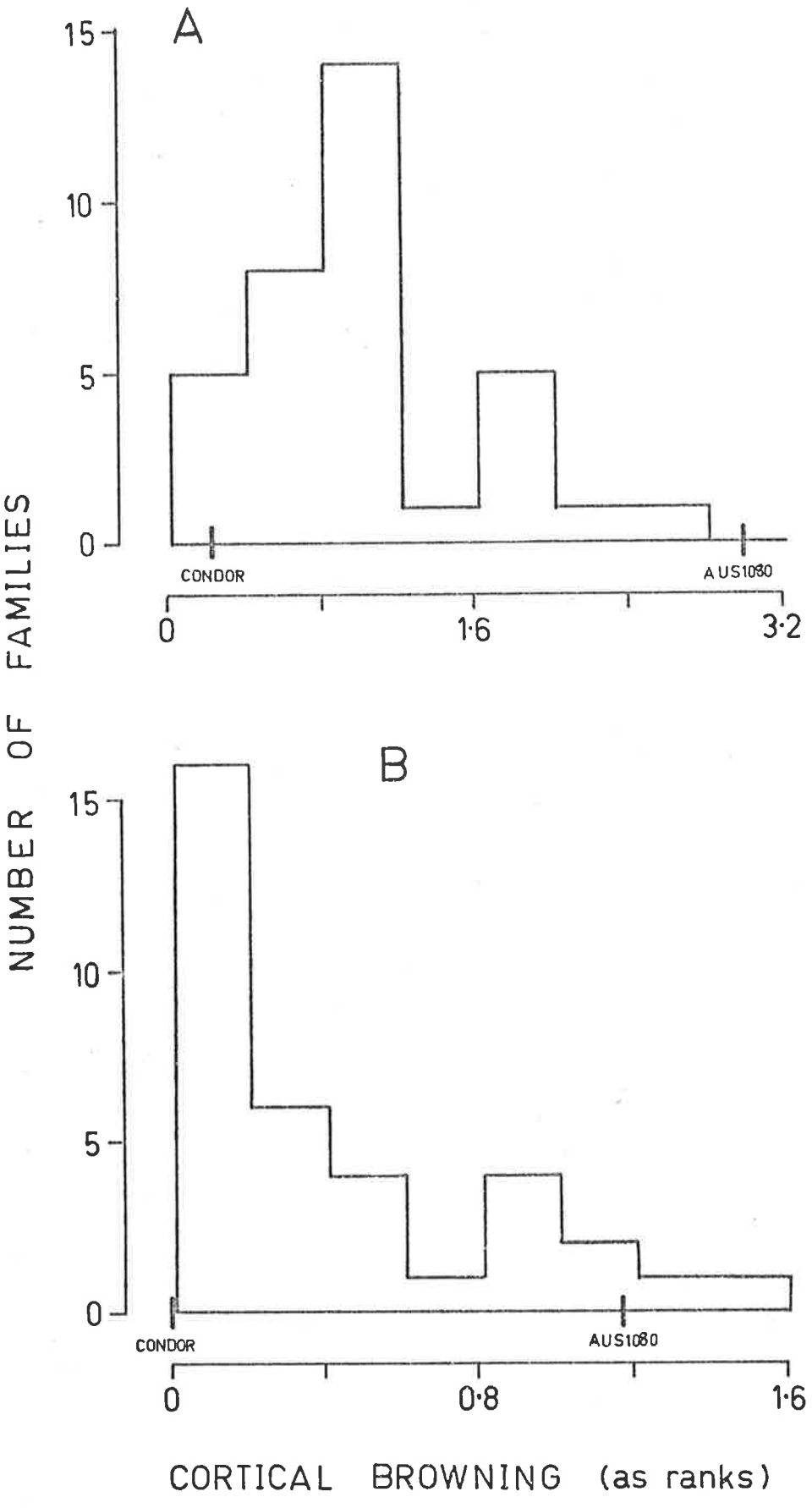
data represent means of 6 replicates

FIGURE 10: Frequency distributions of cortical browning (as ranks, page 17) in the first seminal root of families of set 3 infected with G.g.t. isolate 201 for 10 days

A. discoloured

B. intensely discoloured

data obtained with means of 6 replicates


A
15
10
5
0
CONDOR
AUS1080
0
1·6
3·2
B
15
10
5
0
CONDOR
AUS1080
0
0·8
1·6
NUMBER OF FAMILIES
CORTICAL BROWNING (as ranks)

typically necessary to test segregation ratios, the current data are consistent with single gene control of the brown discoloration of walls of cortical cells and for it to be a recessive character.

(3) The influence of cortical browning on radial invasion by hyphae into roots

As the thickening and discoloration of walls of cortical cells was not under experimental control, its effect in resisting hyphal invasion into roots could not be tested directly. However, hyphae were consistent in growing radially into seminal roots as observed by Fellows (1928), allowing the effect of cortical browning on invasion of underlying stelar tissue to be tested indirectly by association. The extent of cortical browning is scored down root axes while colonisations of steles of roots are scored in transverse sections at fixed points. Nevertheless, negative associations between these variates would provide evidence that cortical browning had retarded invasion of underlying stelar tissue. Associations were tested within treatments for data from previous experiments, excepting data of families of set 3 where associations were simply over individuals. Findings are presented in Table 11 and show associations to be negative and occasionally significant ($P<0.01$). Associations within cultivars differed ($P<0.001$) for data of wheats of set 2. This finding may have reflected differences between cultivars in the extent of colonisation of the endodermis in that experiment, as associations will

TABLE 11: Associations† between cortical browning and colonisation of endodermal cells in the first seminal root of wheat seedlings infected with G.g.t. isolate 201 in three experiments

Experiment			Cortical browning	
treatment	cultivars	time of harvest (days)	discoloured	intensely discoloured
weight of seed	Aus1080	10	-.22	-.26
			n.s.	n.s.
cultivar	set 2	10	-.55**	-.46*
			(0.001)	(0.001)
-	set 3	10	-.01	-.15

differences (P) in associations between treatments are bracketed

significant associations are depicted as * ($P<0.05$) or ** ($P<0.01$)

† associations are pooled from within treatments excepting experiment with set 3

decay to zero as roots become completely colonised. Overall, findings support the hypothesis that thickening and discoloration of walls of cortical cells inhibit invasion by hyphae into underlying stelar tissue of roots of wheat.

Discussion

Evidence is presented in this chapter for seminal roots of wheat to resist radial invasion by hyphae of G.g.t. Garrett (1970) had previously suggested the ectotrophic habit, which is exhibited by hyphae of G.g.t., to be imposed on many root pathogens by resistance in their hosts. This resistance allows hyphae to grow freely on the surface of roots but inhibits radial invasion into roots. Resistance of this nature was observed in the present study where hyphae of a virulent isolate of G.g.t. (226) grew 30x further down than radially into roots (Table 8).

Radial invasion by hyphae of G.g.t. appears to be retarded in the cortex and endodermis of seminal roots. In the present study, the brown discoloration of walls of cortical cells appeared to be associated with resistance to invasion by the pathogen. Cortical browning appears to form a mechanical barrier to hyphae, as it was found to follow premature lignification (Fellows, 1928) and thickening of walls of cells, and the filling of intercellular spaces (Holden, 1976). As previously reported by Skou (1981), resistance to infection by G.g.t. in the endodermis is observed in root sections with extensive colonisation of the innermost cortical cells but with no endodermal infection.

Additional evidence of endodermal resistance was seen in this study in the dissimilar influence of weight of seed on the colonisation of the innermost cortical cells and endodermal cells.

Having entered the stele, there is evidence of additional resistance to the growth of hyphae. As several stelar lesions can form in a root which is inoculated with G.g.t. at a single point (Deacon & Henry, 1978), and as stelar lesions then appear to indicate independant sites of stelar invasion, evidence is provided for the growth of hyphae to be retarded more in the stele than in the cortex of seminal roots. Resistance in stelar tissue can not be simply studied and was not pursued further in this study, but may in part be associated with stelar blackening as suggested by Robertson (1932). The extent to which resistance in each tissue retards the overall rate of hyphal entry into roots, as measured by colonisation of stelar tissue (ECOL & SC, page 16), could not be simply investigated.

Cortical browning and stelar blackening appear to be separate responses of the host to infection by G.g.t. These responses were similar in that cortical browning appeared to consume translocates (Fig. 9) as does stelar blackening (Gilligan, 1980a), and both have been implicated in resistance to the pathogen (Table 11 and Robertson, 1932, respectively). However, when sectioned, browning of walls of cells was only observed in cortical tissue and blackening only observed in stelar tissue. Moreover, only the walls of cortical cells are discoloured (Holden, 1976), while Robertson (1932) found the contents of stelar cells

to be blackened. Responses of the host to infection by G.g.t. were also seen to differ in development in this study. Walls of cortical cells appeared to be transformed when cells were alive but about to be colonised by hyphae of G.g.t. in roots that were less than six days old. In contrast, blackening did not occur in stelar tissue that was younger than about nine days.

In this study,wheats were found to differ in extent of cortical browning in roots infected with G.g.t. In one cross, cortical browning appeared to be under simple genetic control and recessive. Stelar blackening could not be so simply studied,as colonisation of the stele by G.g.t. was not under experimental control, being influenced by resistance to infection in overlying cortical and endodermal tissue. Nevertheless, evidence was found for wheats to differ in the time that must elapse before steles can blacken. Wheats also differed in colonisation of the stele, and thus the rates at which hyphae of G.g.t. invaded radially into roots. However, susceptibility to infection by G.g.t. that is measured as stelar colonisation was not simply inherited, and was most probably influenced by independant effects in overlying tissues or effects of complex inheritance, or both. Moreover, differences between cultivars in rates at which hyphae invaded radially into roots had varied with weight of seed. This effect was largely due to resistance to invasion by G.g.t. in the endodermis, of which further study was restricted by the inability to independantly control cortical infection.

The number and size of stelar lesions in roots were examined as measures of stelar colonisation in roots infected

with G.g.t. at a point. While the number of stelar lesions better indicated the infection of steles of roots than the size of lesions, this measure did not fully correspond with direct measures of infection (ECOL & SC, page 16). A closer association between the number of stelar lesions and stelar colonisation might be found if cultivars of wheat were inoculated with less virulent isolates of G.g.t. than used in this study, and infection measured later than ten days.

These studies were conducted in an artificial environment, a coarse and sterile sand, since field soil supports a diverse population of micro-organisms, some of which are antagonistic to G.g.t. (Rovira & Wildermuth, 1981). In addition, studies were restricted to seminal roots infected with G.g.t. soon after emergence. However, seminal roots grown in the field are usually infected with G.g.t. when older, as the pathogen usually grows a short distance through soil to reach its host (Gilligan, 1980b) and its hyphae undergo a resting phase before colonisation begins (Brown & Hornby, 1971). Moreover, infection was not studied in coronal roots, though tissues of seminal and coronal root systems are similarly invaded by G.g.t. (Fellows, 1928), and stelar blackening and cortical browning is exhibited in both (Fellows, 1928 and personal observation respectively). The extent to which findings of this chapter apply to coronal roots and to roots in the field, is investigated in Chapter 5.

CHAPTER 4

STUDIES IN THE FIELD

Consistent differences between wheats in severity of disease caused by G.g.t. have not been reported in Australian studies, and are infrequently reported elsewhere. The aim of the field experiments was to obtain evidence of resistance to infection by G.g.t. in wheats under the conditions in which cereals are normally grown in South Australia. Resistance was sought by testing differences between wheats in levels of infection of roots, and in the incidence of deadheads. The influence of characters other than resistance on disease caused by G.g.t. was also studied, to test whether observed differences between wheats uniquely indicated resistance to infection by the pathogen. As experiments were naturally infected with G.g.t. and grown similarly to commercial crops of wheat, there was no control of the distribution of the pathogen over the experimental areas, the virulence with which it infected roots, or the form of disease which it caused (Hornby, 1978). Similarly, there was no control over the incidence of other pathogens or their effect on wheats in the experiments. Thus, the effect of all root pathogens on the growth and yield of wheats was studied to determine the course and nature of disease at sites to allow accurate interpretation of findings.

Initially in 1981, trials of the Department of Agriculture at Nangari and Perponda were studied when found to be infected with the pathogen. Both sites are in

areas of the state where root diseases of wheat are endemic. In 1981, total rainfall at both sites was slightly below average, with most rain occurring in the middle of the growing season, and final development of plants occurring in slightly dry conditions (Appendix 2). Yield of grain was about average for each site. Early growth of the crops was not observed. There was a high incidence of deadheads in patches at Nangari, and while there were fewer deadheads at Perponda, they were spread more evenly throughout the plots. The experiments of 1982 were lost through severe drought.

More detailed experiments were conducted in 1983, the final year of field studies. Experiments were conducted at two sites as significant cultivar x environment interactions have been found in previous studies of resistance to infection by G.g.t. in wheat (Scott, 1981). Sites in regions where wheat is grown were selected for proximity to the Waite Institute. Total rainfall in 1983 was much higher than average at Palmer, particularly in the latter half of the growing season, while rainfall in the growing season was average at Strathalbyn after unseasonally heavy rain early in the year (Appendix 2). Crops were grown in favourable conditions at both sites, but for disease of roots. Yield of wheat was well above average at Palmer, but far below the average for the field studied at Strathalbyn (pers. comms. of farmers). Above ground symptoms of root disease in the plants differed between sites, and further, between soils at Palmer. At Strathalbyn

disease was severe, with retarded growth of shoots occurring ten weeks after sowing. By anthesis plants in many plots were stunted and had yellow leaves, with few shoots developing fertile heads. Disease was less severe at Palmer, with patches of retarded growth of shoots occurring 12 weeks after sowing in soil of type Dr4.63. Deadheads became more frequent in these patches upon ripening of the crop. Plants grown in soil of type Gc1.22 were not stunted and developed few deadheads.

Experiments with cultivars of T. aestivum

Cultivars of set 2 were tested for differences in disease caused by G.g.t. in the field, as these wheats were previously shown to differ in infection with the pathogen when grown in cups under controlled conditions. The range in infection in wheats of set 2 was compared to infection in three more resistant cereals, triticale (X Triticosecale Witt., cv. T701-2), rye (cv. South Australian Rye) and barley (cv. Clipper). In 1983 the wheats of set 2 were sown with these cereals as 'controls' at Palmer (in soil of type Dr4.63) and Strathalbyn. Rainfall at the two sites is recorded in Appendix 2. As disease caused by G.g.t. is influenced by the conditions under which wheat is grown, it probably is also influenced by the fitness of a wheat to its growing conditions. Since four of the cultivars of set 2 are poorly adapted to Australian conditions, wheats of sets 4 and 5 were also infected with G.g.t., to test differences in infection with the pathogen and in subsequent

expression of disease between wheats that are fitted to Australian conditions. Thus eight of the 29 cultivars of set 4 were sown at Palmer and all cultivars of set 5 sown at Strathalbyn in 1983.

Families of set 3 were sown at Palmer (in both types of soil) to investigate the genetic control of factors that influence infection with G.g.t. and disease caused by the pathogen in a natural environment. The experiment employed 40 families (as F_4 seed) with the parents as controls (cvs. Aus1080 and Condor).

Experiments with cultivars of sets 4 and 5 at Nangari and Perponda in 1981 employed two replicates. The incidence of pathogens on roots was established at these sites (Table 12) and the percentage of prematurely dead and unfilled heads was recorded. Experiments at Palmer and Strathalbyn in 1983 contained five replicates for cultivars of sets 2, 4 and 5, and ten replicates for families of set 3 (five in each type of soil). There was sufficient time at anthesis to score only two replicates of wheats of set 5 at Strathalbyn. Eight plants were sampled from plots for experiments with wheats of sets 2, 4 and 5. This was not possible with the number of plots (420) in the experiment with families of set 3 at Palmer where two plants were sampled from each plot at tillering and one at anthesis.

Incidence of G. graminis var tritici on roots

(1) Differences between cultivars in infection with G. graminis var tritici

Infection of roots of cultivars of sets 2 to 5 is summarised in Tables 13 to 16 respectively with analyses.

TABLE 12: Incidence of coronal roots (at anthesis) infected with *G.g.t.* and *R. solani* for cultivars of sets 4 and 5 at Nangari and Perponda.

Cultivars	Sites	Incidence of infected roots (as % of plants)			
		G.g.t. > *R. solani*	*G.g.t.* = *R. solani*	*G.g.t.* < *R. solani*	no symptoms
set 4	Nangari rep. 1	94	2	4	0
set 4	Nangari rep. 4	84	0	7	9
set 5	Nangari rep. 4	98	2	0	0
set 5	Perponda rep. 4	50	6	38	6

data are of 80 observations for set 4
and 60 observations for set 5

TABLE 13: Percentages (as angles) of roots infected with G.g.t. at tillering (t) and anthesis (a) for wheat cultivars of set 2 and barley, rye and triticale in field experiments.

Site of Experiment	Sample time	Wheats								Barley	Rye	Triticale	ANOVA (P)		
		(Aus1080)	(Chile909)	(Chile911)	(Condor)	(Kite)	(Nebraska) 86	(Rac311)	(Means)				(Wheats)	(Cereals)	(site x wheats)
Seminal roots															
Palmer	t	20.0	22.5	20.1	12.5	19.5	15.9	22.1	18.9	9.2	5.1	16.7	n.s.	0.05	n.s.
Strathalbyn	t	22.7	33.0	33.5	31.9	31.5	37.4	29.8	31.4	26.0	5.0	22.0	n.s.	0.01	
Palmer	a	28.7	23.5	30.0	28.8	30.0	25.9	29.3	28.0	9.1	0.0	24.9	n.s.	0.01	n.s.
Strathalbyn	a	64.5	60.6	62.6	68.4	58.0	66.2	56.8	62.4	38.1	13.1	60.2	n.s.	0.001	
Coronal roots															
Palmer	t	6.2	5.2	0.0	0.0	0.0	5.3	0.0	2.4	5.2	0.0	7.0	n.s.	n.s.	-
Strathalbyn	t	0.0	1.6	0.0	1.6	0.0	0.0	0.0	0.5	0.0	0.0	0.0	n.s.	n.s.	
Palmer	a	22.1	18.2	29.1	25.3	24.8	24.5	26.9	24.4	9.4	5.1	19.5	n.s.	0.001	n.s.
Strathalbyn	a	38.1	32.3	38.6	43.5	34.7	45.3	46.9	39.9	19.7	6.3	40.7	0.05	0.001	

data represent means of 40 individuals from 5 plots

TABLE 14: Percentages (as angles) of roots infected with *G.g.t.* at tillering (t) and anthesis (a) for parents and F_2 families of set 3 at Palmer

Sample time	Parents		Families			ANOVA (P)	
	Aus1080	Condor	minimum	mean	maximum	parents	families
	Seminal roots						
t	5.8	1.8	2.3	7.6	16.0	n.s.	n.s.
a	17.8	22.8	0.0	19.2	37.5	n.s.	n.s.
	Coronal roots						
a	11.3	12.3	6.1	11.7	18.4	n.s.	n.s.

data of parents represent means of 20 replicates and data of families represent means of 10 replicates

TABLE 15: Percentages (as angles) of roots infected with *G.g.t.* for cultivars of set 4 at Palmer

Cultivar	Infection with *G.g.t.*	
	seminal roots at tillering	coronal roots at anthesis
Egret	21.4	12.9
M2335	22.9	14.7
M2424	15.3	20.5
N10/TG2248-8	21.8	11.8
PD36	18.7	11.5
RAC416	5.7	17.9
SD34	24.7	15.6
(WW-15*MH-49)/36/W3	16.1	10.9
mean	18.3	14.5
ANOVA (P)	n.s.	n.s.

data represent means of 40 individuals from 5 plots

TABLE 16: Percentages (as angles) of roots infected with *G.g.t.* for cultivars of set 5 at Strathalbyn

Cultivar	Infection with *G.g.t.*	
	seminal roots at tillering	coronal roots at anthesis
Aroona	40.2	71.1
Condor	44.7	73.6
Egret	43.1	68.9
Festiguay	40.6	64.9
Kite	41.2	77.8
Lance	41.8	63.8
Miling	39.1	66.4
Millewa	44.2	80.9
Oxley	41.9	79.2
PF/41/W1	37.1	72.5
Rac311	33.7	62.7
Warigal	42.3	66.4
Warimba	44.7	72.7
means	41.1	70.8
ANOVA (P)	n.s.	n.s.

data represent means of 40 individuals from 5 plots for seminal roots and 16 individuals from 2 plots for coronal roots

Seminal roots of wheats did not differ in infection at any site while coronal roots only differed in infection with G.g.t. when cultivars of set 2 were sampled at anthesis at Strathalbyn. The finding that wheats differed most in infection with G.g.t. when mature had been found earlier by Wachter and Mogling (1977). Frequency distributions of families of set 3 (Fig. 11) did not provide independent evidence for a simple genetic control of factors that influence infection of roots with G.g.t. The repeatability of measures of infection could only be tested for the seven wheats of set 2 that were grown at two sites. However, cultivar x site interactions were not significant, which was consistent with an earlier finding by Nilsson (1969) that wheats were consistently infected with G.g.t. in natural environments.

Wheats were found to differ from the more resistant cereals in infection with G.g.t. (Table 13). As has been found previously (Scott, 1981), roots of triticale were similarly infected to roots of wheats, while roots of barley were less infected and roots of rye less infected again.

(2) Factors influencing the infection of roots with G. graminis var tritici

Some of the characters that influence infection of roots with G.g.t. were identified, and the extent to which they influenced the infection of cultivars was investigated. Several characters have been previously associated with the infection of roots. Wachter and Mogling (1979) found growth of shoots to be associated with 'stronger vulnerability

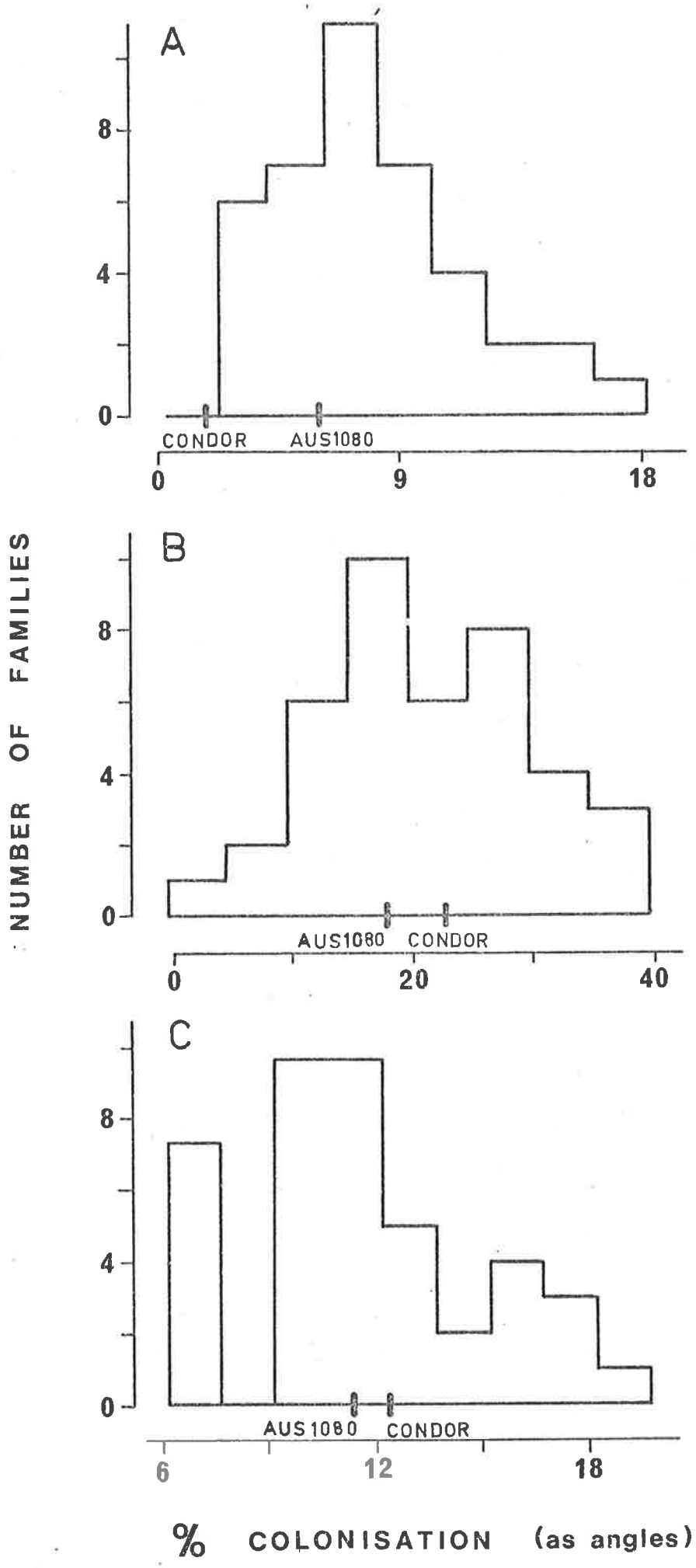
FIGURE 11: Frequency distributions of percentage (as angles) of roots infected with G.g.t. for families of set 3 at Palmer

A. Seminal roots at tillering

B. Seminal roots at anthesis

C. Coronal roots at anthesis

data obtained using means of ten plots


A
8
4
0
CONDOR
AUS1080
0
9
18
B
8
4
0
AUS1080
CONDOR
0
20
40
C
8
4
0
AUS1080
CONDOR
6
12
18
NUMBER OF FAMILIES
% COLONISATION (as angles)

of wheats' to infection by G.g.t. As growth of shoots and infection of roots can not be causally and positively associated, more direct associations between percentage infection of roots and number of roots, and length of the subcoronal internode were tested here. Patel (pers. comm.) found that the incidence of R. solani was negatively associated with the incidence of G.g.t. on roots of wheat at some field sites. Thus, associations between percentages of roots with symptoms of infection with G.g.t. and other pathogens were also examined. Associations were determined within cultivars for data of set 2, within replicates for data of families of set 3, but over all data for cultivars of sets 4 and 5 which were adapted to local conditions and less diverse than other wheats. Associations were determined for individual plants and are summarised in Tables 17 and 18 for infection of the seminal and coronal roots respectively.

The number of roots and their extent of infection tended to be positively associated where significant (Tables 17 and 18), reflecting the significance of spread of infection between adjacent roots in a plant. Significant and consistently negative associations were found between the length of the subcoronal internode and extent of infection of the seminal roots (Tables 17 and 18). This suggests inoculum of G.g.t. was most frequent or most infective when high in the soil profile. Data show this effect to persist to anthesis in some experiments but to decline in others. Evidence for strong antagonism between G.g.t. and R. solani was observed (Tables 17 and 18),

TABLE 17: Associations† between infections with G.g.t. of seminal roots at tillering (t) and anthesis (a) and characters that may influence infection, for individual plants from field experiments

Experiment		Characters§					
cultivars	site	SEM		SCI		RSEM	
		t	a	t	a	t	a
set 2	Palmer	.10*	.14*	-.05	-.14*	-.10*	-
set 2	Strathalbyn	-.06	.06	-.11	.08	-.28***	-.38***
set 3	Palmer (Dr4.63)	.03	.11	-.12*	-.04	-.07	-
set 3	Palmer (Gc1.22)	-.02	.17*	-.12**	-.24**	-.07	-
set 4	Palmer	-.14	-	-.33	-	.11	-
set 5	Strathalbyn	.11	.15	-.26*	-.05	-.36**	-

differences in associations between cultivars or replicates were not significant

significant associations are depicted as * ($P<0.05$), ** ($P<0.01$) or *** ($P<0.001$)

† associations are pooled from within cultivars for set 2 and from within replicates for set 3

§ abbreviations of characters were explained on page 21

TABLE 18: Associations[†] between infection with G.g.t. of coronal roots (at anthesis) and characters that may influence infection for individual plants from field experiments

Experiment		Characters[§]				
cultivar	site	COR	SCI	GSEM	RCOR	OCOR
set 2	Palmer	-.01	-.11	.58***	-	-.27**
set 2	Strathalbyn	.25***	.07	.83***	-.54***	-
set 3	Palmer (Dr4.63)	.04	-.05	.42***	-	-.23***
set 3	Palmer (Gc1.22)	-.05	-.17**	.20**	-	-.07
set 4	Palmer	.14	-.10	-	-	.02
set 5	Strathalbyn	-.46*	.19	.49**	-	-.02

differences in associations between cultivars or replicates were not significant

significant associations are depicted as * ($P<0.05$), ** ($P<0.01$) or *** ($P<0.001$)

[†] associations are pooled from within cultivars for set 2 and from within replicates for set 3

[§] abbreviations of characters were explained on page 21

supporting earlier observations by Patel (pers. comm.) made in both natural and controlled environments. Antagonism appeared to occur on seminal and coronal roots and appeared stronger in effect at anthesis than at tillering. Evidence of antagonism was also found between G.g.t. and the unknown pathogen on coronal roots at anthesis (Table 18). Strong positive associations between infection of seminal and coronal roots with G.g.t. at anthesis (Table 18) reflect the progressive spread of the pathogen within the root system of plants at both sites.

The influence of these characters on differences between cultivars in infection with G.g.t. were determined for the wheats in field experiments. These effects were tested both by association and analysis of variance. Associations between the infection of roots with G.g.t. and the number of roots, length of the subcoronal internode and infection of roots with other pathogens were determined for means of cultivars from experiments in 1983 and findings are summarised in Table 19. Differences between cultivars for these characters were tested and analyses presented in Table 20, while means of wheats are presented in Appendix 3. While the number of seminal roots influenced the infection of individual plants with G.g.t. (Table 17), and though cultivars differed in number of seminal roots (Table 20), the effect of root number was too weak to affect the extent to which field grown cultivars of wheat were infected with the pathogen. Wheats also differed in the number of coronal roots (Table 20), but the effect of this character on the infection of coronal roots of cultivars appeared to be obscured by the reduced vigour of diseased

TABLE 19: Associations between infection with G.g.t. of roots at tillering (t) and anthesis (a) and characters that may influence infection for means of cultivars from field experiments

Experiment		Characters[§]							
cultivar	site	SEM/COR[¶]		SCI		RSEM		RCOR	OCOR
		t	a	t	a	t	a	a	a
seminal roots (GSEM)									
set 2	Palmer	.36	-.33	-.68	.42	.08			
set 2	Strathalbyn	.20	-.14	-.47	-.03	-.44	-.55		
set 3	Palmer	.09	.03	-.47**	-.04	-.25			
set 4	Palmer	-.22		.25		-.07			
set 5	Strathalbyn	.24		-.60*		.14			
coronal roots (GCOR)									
set 2	Palmer		-.75		.13				.13
set 2	Strathalbyn		-.72		.60			.33	
set 3	Palmer		-.01		-.11				-.04
set 4	Palmer		.41		-.16				.25
set 5	Strathalbyn		-.65*		.10				.08

significant associations are depicted as * ($P<0.05$) or ** ($P<0.01$)

§ abbreviations of characters are explained on page 21

¶ SEM is only associated with GSEM and COR only with GCOR

TABLE 20: Significance of differences between cultivars in characters that may influence infection of roots with G.g.t. in field experiments

Experiment			Characters[§]					
cultivars		site	SEM	COR	SCI	RSEM	RCOR	OCOR
	at tillering							
set	2	Palmer	***		**	n.s.	n.s.	
set	2	Strathalbyn	*		***	n.s.	n.s.	n.s.
set	3	Palmer	***		***	n.s.		
set	4	Palmer	**		n.s.	n.s.	n.s.	
set	5	Strathalbyn	**		***	n.s.	n.s.	
	at anthesis							
set	2	Palmer	***	***	***			n.s.
set	2	Strathalbyn	*	**	***	n.s.	n.s.	n.s.
set	3	Palmer		**				n.s.
set	4	Palmer		**				*
set	5	Strathalbyn	n.s.	*	*			n.s.

significant differences are depicted as * ($P<0.05$), ** ($P<0.01$) or *** ($P<0.001$)

§ abbreviations of characters are explained on page 21

plants at some sites (Table 19). The length of the sub-coronal internode also differed strongly between wheats, and had consistently influenced the infection of seminal roots of individual plants with G.g.t. at tillering (Table 17) to the extent that infection in cultivars of wheat was also affected (Table 19). This effect was not found in older plants (Tables 17, 18 and 19). Cultivars of wheat did not differ in infection with R. solani (Table 20) which could not then influence the infection of cultivars of wheat with G.g.t. (Table 19) to which it appeared to be strongly antagonistic (Tables 17 and 18). While infection with the unknown pathogen appeared to retard infection of individual plants with G.g.t. in some experiments, it did not influence the extent of infection in cultivars of wheat (Table 19). Wheats did differ in infection with the unknown pathogen in an experiment at Palmer (cultivars of set 4 : Table 20), but the pathogen was then not antagonistic to G.g.t. (Table 18).

The course of disease caused by pathogens of roots at sites

The course of disease was indicated at sites by the effect of infection of roots on the growth of plants, deadheads and yield of grain. As disease was not under experimental control, effects could not be tested directly but were tested indirectly by associations, which were determined as previously.

(1) Damage to the vegetative growth of plants

Three measurements of growth of plants were adopted, the dry weight of shoots at tillering and at anthesis and

the number of fertile tillers in plots. Infections that damage plants would be expected to be negatively associated with these measures of growth and were tested with one tailed tests of significance.

Associations between the infection of roots with pathogens and weight of dried shoots at tillering and anthesis were determined for individual plants, and findings summarised in Table 21. Evidence was found for weight of dried shoots to be significantly reduced at tillering by infection of seminal roots with R. solani but not G.g.t. However, there was strong evidence that at all sites, shoots were most damaged at anthesis by infection of roots with G.g.t. Infection of coronal roots with the unknown pathogen at anthesis also appeared to have significantly reduced weight of dried shoots ($P<0.01$) of plants of set 3 grown in soil of type Dr4.63 at Palmer. Blackening of the subcoronal internode was not significantly associated with weight of dried shoots at anthesis except for wheats of set 2 at Strathalbyn where the infection of roots with G.g.t. was severe.

Associations between the infection of roots with pathogens and the number of fertile tillers in plots were determined and findings summarised in Table 22. Fertile tillers were only measured in experiments with cultivars of set 2. Findings show infection of roots with G.g.t. had reduced the number of fertile tillers in plots at Strathalbyn but not Palmer. Moreover, infection of roots with R. solani was positively associated with fertile tillers in plots ($P<0.01$, 2 tailed test), and most likely

TABLE 21: Associations[†] between infection of roots with pathogens and weight of dried shoots at tillering and anthesis for individual plants from field experiments

Experiment		Infection with pathogens[§]							
		tillering		anthesis					
cultivars	site	GSEM	RSEM	GSEM	GCOR	GSCI	RSEM	RCOR	OCOR
set 2	Palmer	-.01	-.14**	-.09	-.06	-.09			-.05
set 2	Strathalbyn	.01	-.20***	-.09	-.13*	-.22***	-.05	.00	
set 3	Palmer (Dr4.63)	.09	-.08*	-.26***	-.09	-.04			-.15**
set 3	Palmer (Gc1.22)	.09	-.04	-.33***	-.17**	-.01			.02
set 4	Palmer	.35			.04				-.19
set 5	Strathalbyn		.02	-.58**	-.71***	.36			-.11

differences in associations between cultivars or replicates were not significant

significant associations are depicted as * (P<0.05), ** (P<0.01) or *** (P<0.001)

† associations are pooled from within cultivars for set 2 and from within replicates for set 3

§ abbreviations of infections are explained on page 21

TABLE 22: Associations[†] between infection of roots with pathogens at tillering and anthesis and number of fertile tillers in plots in field experiments

Experiment		Infection with pathogens[§]							
		tillering		anthesis					
cultivars	site	GSEM	RSEM	GSEM	GCOR	GSCI	RSEM	RCOR	OCOR
set 2	Palmer	.37	-.39	.06	.46	-.12			-.29
set 2	Strathalbyn			-.71**	-.67**	-.33	.30	.74**	

differences in associations between cultivars were not significant

significant associations are depicted as ** ($P<0.01$)

[†] associations are pooled from within cultivars

[§] abbreviations of infections are explained on page 21

reflected the strong antagonism detected earlier between this pathogen and G.g.t. at Strathalbyn (Tables 17 and 18).

(2) Incidence of deadheads

Deadheads are a visible form of loss of yield that is caused by disease of roots, and are frequently employed to measure the susceptibility of cultivars of wheat to infection by G.g.t. The pathogens responsible for deadheads in this study were investigated and differences between wheats in their incidence were tested. Characters that influence the incidence of deadheads were tested to more critically assess differences between wheats in susceptibility to infection by G.g.t.

The influence of the incidence of pathogens on the incidence of deadheads in plots was tested as previously by association. One tailed tests of significance were used as only positive associations implicate a pathogen as causing deadheads. Findings were summarised in Table 23. Evidence was found for G.g.t. alone to have caused deadheads in experiments, except for families of set 3 that were tested in soil of type Gc1.22 where the unknown pathogen was implicated.

Wheats were tested for differences in the incidence of deadheads that were most probably due to the infection of roots with G.g.t. and findings presented in Tables 24 to 27 for wheats of sets 2 to 5 respectively. Findings for cultivars of set 2 show cultivars to have differed significantly in the incidence of deadheads ($P<0.001$).

TABLE 23: Associations† between infection of roots with pathogens at tillering and anthesis and incidence of deadheads in plots in field experiments

Experiment		Infection with pathogens§								
cultivars	site	tillering			anthesis					
		GSEM	RSEM	RCOR	GSEM	GCOR	GSCI	RSEM	RCOR	OCOR
set 2	Palmer	.41	.10	.01	.50*	.41	.14			.28
set 2	Strathalbyn				.46*	.44*	.51*	-.27	-.44	
set 3	Palmer (Dr4.63)	.00	.06		.06	.21***	.00			.05
set 3	Palmer (Gc1.22)	.05	.05		-.01	-.07	.10			.13*
set 4	Palmer	.54**	.29	-.20		.35*				-.28

differences in associations between cultivars or replicates were not significant

significant associations are depicted as * (P<0.05), ** (P<0.01) or *** (P<0.001)

† associations are pooled from within cultivars for set 2 and from within replicates for set 3

§ abbreviations of infection are explained on page 21

TABLE 24: Percentages (as angles) of deadheads in plots of wheats of set 2 and barley, rye and triticale in field experiments

	Experiment	
	Palmer	Strathalbyn
Wheats		
Aus1080	6.6	1.5
Chile909	0.6	0.0
Chile911	20.5	11.3
Condor	17.0	26.2
Kite	11.8	4.7
Nebraska86	26.3	37.1
Rac311	20.7	18.4
means	14.8	14.8
Barley	0.0	0.0
Rye	0.0	0.0
Triticale	16.0	19.9
ANOVA (P)		
wheats	0.001	0.001
controls	0.001	0.001
site x wheats	0.001	

data represent means of 5 plots

TABLE 25: Number of deadheads in plots of parents and families of set 3 in two soil types at Palmer

Soil types	Parents		Families			ANOVA (P)		
	Aus1080	Condor	minimum	mean	maximum	parents	families	soil x family
Dr4.63	3.4	21.0	0.4	14.2	40.3	0.001	0.001	0.001
Gc1.22	2.5	1.8	0.2	3.1	9.9	n.s.	0.001	

data of parents represent means of ten plots and of five plots for families

TABLE 26: Percentages (as angles) of deadheads in plots of cultivars of set 4 at Nangari and number of deadheads in plots at Palmer

Cultivars of set 4	Nangari	Palmer
Eagle	27.5	-
Egret	19.7	15.7
Festiguay	27.5	-
M2335	20.9	17.3
M2424	24.9	32.6
Oxley	20.8	-
Sun39A	23.9	-
Sun41A	26.1	-
Sun43A	20.7	-
Cook	23.9	-
K-2003-12	27.6	-
LR/OXS2730-4	19.5	-
N10/TG2248-8	40.0	33.0
Halberd	20.1	-
(MKR*Kite)/57/S14	27.5	-
(MM*MMC)/59/W6	13.1	-
RAC357	18.3	-
RAC399	29.1	-
RAC415	31.3	-
RAC416	29.2	16.0
(WW-15*MH-49)/36/W3	17.7	20.3
Millewa	13.4	-
MQ6	22.6	-
PD36	17.5	23.3
SD34	17.7	31.0
Jacup	36.1	-
69W/237	24.7	-
69W/393	18.1	-
69Z/401	22.7	-
ANOVA (P)		
cultivar	n.s.	n.s.

Data from Nangari represent means of 2 plots and of Palmer represent means of 5 plots

TABLE 27: Percentages (as angles) of deadheads in plots of cultivars of set 5 in field experiments

Cultivars of set 5	Experiment	
	Nangari	Perponda
Aroona	21.4	15.3
Condor	23.3	17.5
Cook	-	24.3
Festiguay	19.0	24.3
Jacup	-	21.9
Kite	17.6	22.5
Lance	30.6	24.1
Miling	19.9	-
Millewa	20.0	29.0
Oxley	26.6	17.4
PF/41/W1	26.1	21.5
RAC311	32.4	17.1
Warigal	13.3	15.5
Warimba	23.1	38.3
ANOVA (P)		
cultivar	n.s.	

Data represent means of observations in one replicate

While a significant cultivar x site interaction was also found ($P<0.001$), wheats were similarly ranked for the incidence of deadheads at Palmer and Strathalbyn except for Condor (Table 24). Dead and empty heads were not observed in barley and rye but were similarly frequent in wheat and the triticale (Table 24). Families of set 3 also differed significantly in the incidence of deadheads in plots on soil of type Dr4.63 (Table 25). This provided evidence for the genetic control of the incidence of deadheads that were caused by G.g.t. as the parents also differed (Table 25). However, the frequency distribution of families (Fig. 12) did not indicate that genetic control was simple. Deadheads in families of set 3 grown on soil of type Gc1.22 did not appear to be caused by the infection of roots with G.g.t. This was reflected in the significant family x soil type interaction ($P<0.001$) detected when all data of deadheads in families of set 3 were analysed by a two factor ANOVA (Table 25). The distributions of pathogens were not measured at Nangari and Perponda in 1981, so the cause of deadheads at these sites could not be established. However G.g.t. was the most frequent pathogen on roots at both sites (Table 12) and the most likely cause of deadheads. Findings for cultivars of set 4 (Table 26) show wheats did not differ in the incidence of deadheads at either Nangari or Palmer. Moreover data for cultivars that were tested at both sites were not associated ($r = 0.36$ with $df=6$), which indicates deadheads were not similarly formed in wheats of set 4 at these sites. Cultivars of set 5 also did not differ in the

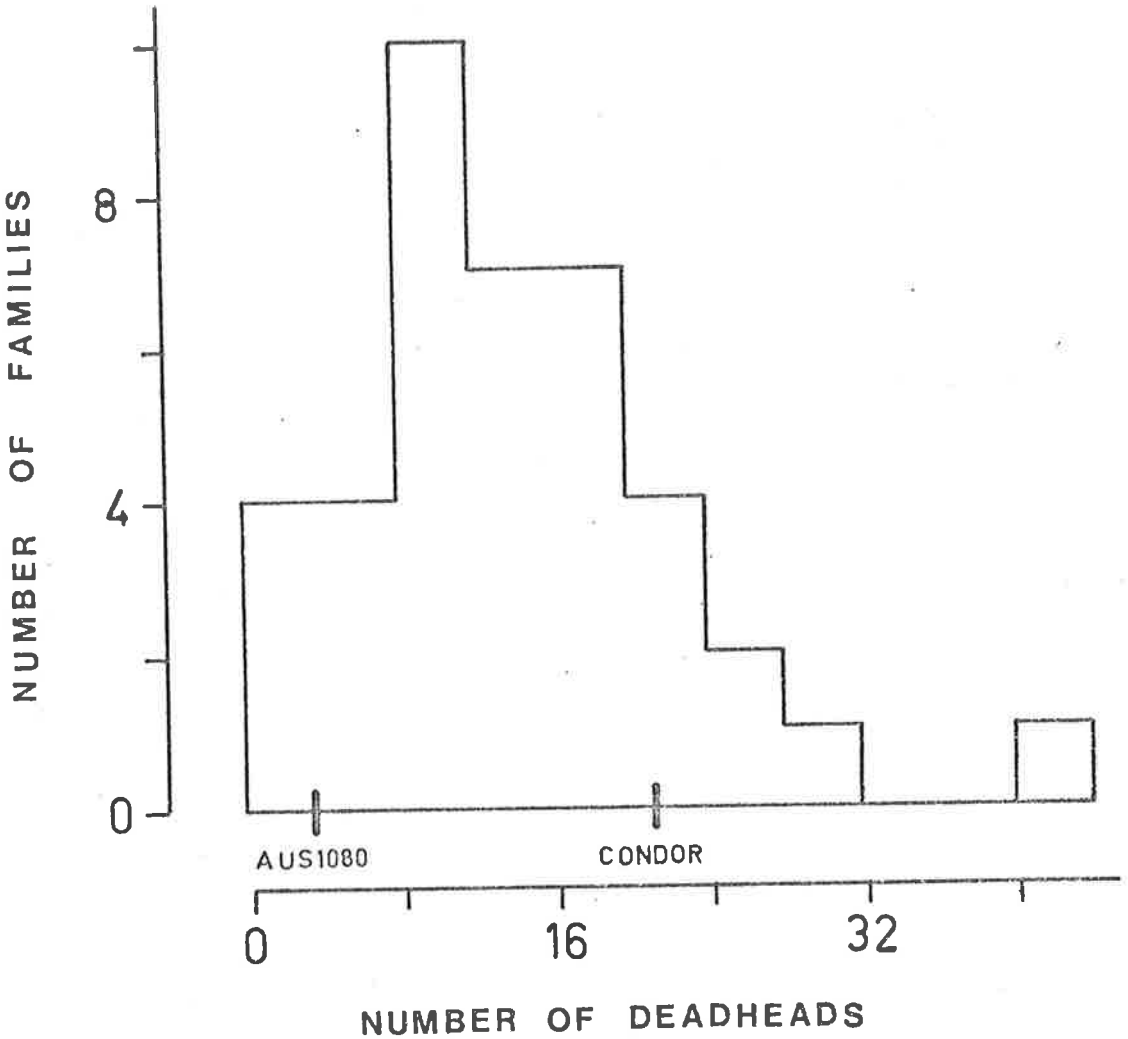


FIGURE 12: Frequency distribution of number of deadheads in plots of families of set 3 grown on soil of type Dr4.63 at Palmer

incidence of deadheads at Nangari and Perponda (Table 27).

Characters influencing the incidence of deadheads were studied in detail, to determine the cause of differences between cultivars in their incidence. Deadheads develop when plants that have sufficiently restricted root systems are stressed. Thus, Bockmann (1966) found cultivars tend to suffer less severe disease when new coronal roots are produced at a greater rate than established roots are destroyed by G.g.t. In another study, Wachter and Mogling (1979) found vigorous growth of shoots and early maturation of heads to be associated with 'susceptibility' of wheats to infection by G.g.t. The length of the subcoronal internode was also examined as this character had been associated with the infection of roots with G.g.t. earlier in this study. Associations between the incidence of deadheads and infection of the seminal and coronal roots had been presented earlier in Table 23. Deadheads tended to be more strongly associated with infection of seminal roots than coronal roots of wheats of set 2, but the reverse was true for wheats of set 3. Insufficient data were collected to similarly compare infections of roots of the remaining sets of wheats. Associations between the incidence of deadheads in plots and the number of coronal roots, weight of dried shoots, number of tillers and the length of the subcoronal internode at anthesis were determined as previously and findings are presented in Table 28. Maturity was not similarly tested as wheats were not all scored for this character in situ (Appendix 4). The number of coronal roots was not negatively associated

TABLE 28: Associations[†] between the incidence of deadheads in plots and characters of plants (at anthesis) that may influence deadheads in field experiments

Experiment		Characters[§]			
cultivars	site	COR	DW	TIL	SCI
set 2	Palmer	.25	.01	.05	.44
set 2	Strathalbyn	.48	.45	.63**	.03
set 3	Palmer (Dr4.63)	.05	.15*	-.06	.03
set 4	Palmer	-.30	.40*	-.03	

differences in associations between cultivars or replicates were not significant

significant associations are depicted as * ($P<0.05$) ** ($P<0.01$) or *** ($P<0.001$)

[†] associations are pooled from within cultivars for set 2 and from within replicates for set 3

[§] abbreviations of characters are explained on page 21

with the incidence of deadheads, and did not support the hypothesis that the growth of new coronal roots late in the season lessens the incidence of deadheads in wheat. However, infection with G.g.t. also stimulates the development of coronal roots (Manners & Myers, 1981) which may have obscured the effect under test, so that Bockmann's (1966) hypothesis was not critically tested in this study. Weight of dried shoots at anthesis tended to be positively associated with the incidence of deadheads, as was similarly found by Wachter and Mogling (1979). This finding is consistent with plants of large vegetative mass placing greater demand on diseased root systems at ripening than smaller plants, and are thus more prone to early death. Associations between the number of tillers and the incidence of deadheads were without trend, though highly significant in one experiment. As found earlier, the length of the subcoronal internode did not influence disease in older crops of wheat.

The influence of weight of dried shoots and number of tillers at anthesis on the incidence of deadheads in cultivars of wheat was further investigated. The influence of maturity was similarly tested by association of means of cultivars and analysis of differences between wheats. Data for maturity of cultivars of sets 2 to 5 were obtained from field plots at Waite, Palmer and Turretfield (Appendix 4). Associations are summarised in Table 29 and analyses of variance in Table 30. Findings suggest differences between wheats in dry weight of shoots at anthesis (Table 30) did influence the incidence of deadheads in cultivars of wheat (Table 29).

TABLE 29: Associations between incidence of deadheads and characters that may influence deadheads for means of cultivars from field experiments

Experiment		Characters[§]		
cultivars	site	DW (at anthesis)	TIL	MAT[¶]
set 2	Palmer	.68	-.52	.93**
set 2	Strathalbyn	.55	-.59	.71
set 3	Palmer (Dr4.63)	.46**	-.24	.80***
set 4	Palmer	.63	-.12	.59
set 4	Nangari, rep 1			-.14
set 4	Nangari, rep 4			.51**
set 5	Nangari, rep 4			.54
set 5	Perponda, rep 4			.05

significant associations are depicted as * ($P<0.05$), ** ($P<0.01$) or *** ($P<0.001$)

§ abbreviations of characters are explained on page 21

¶ data of maturity are presented in Appendix 4

TABLE 30: Significance of differences between cultivars in characters that may influence incidence of deadheads in field experiments

Experiment		Characters[§]		
cultivars	site	DW (anthesis)	TIL	MAT
set 2	Palmer	***	***	
set 2	Strathalbyn	***	***	
set 2	Waite			***
set 3	Palmer (Dr4.63)	*	*	***
set 4	Palmer	n.s.	**	
set 4	Turretfield			***

significant differences are depicted as * ($P<0.05$), ** ($P<0.01$) or *** ($P<0.001$)

§ abbreviations of characters are explained on pages 21 and 91

The number of tillers in wheats did not influence deadheads. Differences between wheats in earliness of maturity (Table 30) strongly promoted the incidence of deadheads in cultivars (Table 29), supporting the findings of Wachter and Mogling (1979). It could be argued that a positive association between the incidence of deadheads and earliness of maturity is expected when observation is at a single time, since heads must form before they can prematurely die. However, this argument is not valid since live and empty heads could be distinguished from live and partly filled heads, while only one cultivar (Chile909 at Strathalbyn) was of such delayed maturity that the future filling of grain was uncertain. In any event findings show deadheads do not accurately measure differences between cultivars in resistance to infection by G.g.t. but largely reflect differences in maturity and may therefore simply indicate differences in escape from disease. Data were not sufficient to establish differences between wheats in the incidence of deadheads that were not due to differences in maturity.

(3) Loss of yield of grain

Pathogens on roots were tested for effect on the filling and final yield of grain. After investigating the effect of pathogens on the growth of crops, the incidence of deadheads and weight of grain, an overview of loss of yield was sought by associating each character with final yield of grain.

The effect of pathogens on weight of grain was tested as previously, by associations within cultivars and

replicates. Weight of grain was only scored at Palmer and where infection of roots with G.g.t. clearly damaged plants. There was insufficient time to score this character at other locations. Findings are presented in Table 31 and provide evidence for infection of coronal roots to most reduce weight of grain. Infection with G.g.t. appeared to reduce weight of grain most in cultivars of set 2 while infection with the unknown pathogen appeared most damaging in families of set 3 (Table 31).

The effect of pathogens on final yield of grain was tested as previously by association and findings presented in Table 32. Data suggest infection by G.g.t. was most damaging in experiments at Strathalbyn and in families of set 3 when grown on soil of type Dr4.63 at Palmer. Other pathogens did not appear to significantly reduce yield of grain. Positive associations between yield of grain and infection of roots with R. solani at Strathalbyn most likely reflects antagonism on roots between this pathogen and G.g.t. which had been detected earlier (Tables 17 and 18). The significant positive association between yield of grain and infection with the unknown pathogen in cultivars of set 4 at Palmer could not be similarly explained.

An overview of the course of disease in experiments was sought by testing associations between final yield of grain and weight of dried shoots at anthesis, number of fertile tillers in plots, the incidence of deadheads and weight of grain, and findings are presented in Table 33. Loss of yield in plots infected with G.g.t. occurs

TABLE 31: Associations[†] between infection of roots with pathogens (at anthesis) and weight of grain from individual plots in field experiments

Experiment		Infection with pathogens[§]			
cultivars	site	GSEM	GCOR	GSCI	OCOR
set 2	Palmer	-.26 (n.s.)	-.53* (n.s.)	-.15 (n.s.)	.08 (n.s.)
set 3	Palmer	-.04 (n.s.)	-.14 (n.s.)	-.09 (0.05)	-.21* (n.s.)

significance (P) of differences between cultivars or replicates are bracketed

significant associations are depicted as * ($P<0.05$)

[†] associations are pooled from within cultivars for set 2 and within replicates 2 and 5 on soil of type Dr4.63 for set 3

[§] abbreviations of infections are explained on page 21

TABLE 32: Associations† between infection of roots with pathogens at tillering and anthesis and yield of grain in plots from field experiments

Experiment		Infection with pathogens§							
		tillering		anthesis					
cultivars	site	GSEM	RSEM	GSEM	GCOR	GSCI	RSEM	RCOR	OCOR
set 2	Palmer	-.18	-.19	-.24	-.37	-.25			-.16
set 2	Strathalbyn	-.81***	.24	-.87***	-.81***	-.49*	.37	.84***	
set 3	Palmer (Dr4.63)	-.13*	.02	-.20**	-.24***	-.04			-.10
set 3	Palmer (Gc1.22)	-.04	-.09	.10	-.06	.01			-.10
set 4	Palmer	-.14	-.17		-.02				.33*
set 5	Strathalbyn			-.55**	-.74***	-.44**			-.05

differences in associations between cultivars or replicates were not significant

significant associations are depicted as * (P<0.05), ** (P<0.01) or *** (P<0.001)

† associations are pooled from within cultivars for set 2 and from within replicates for set 3

§ abbreviations of infections are explained on page 21

TABLE 33: Associations† between yield of grain and mean weight of dried shoots (DW) at anthesis, number of fertile tillers (NFT) and deadheads (DH), and weight of grain (GRW) in plots from field experiments

Experiment					
cultivars	site	DW	NFT	DH	GRW
set 2	Palmer	-.27	.47	-.64**	.74**
		(n.s.)	(n.s.)	(n.s.)	(n.s.)
set 2	Strathalbyn	.27	.89***	-.27	
		(n.s.)	(n.s.)	(0.01)	
set 3	Palmer	.18**		-.16**	.65***
	(Dr4.63)	(0.01)		(n.s.)	(n.s.)
set 3	Palmer	-.00		-.08	
	(Gc1.22)	(n.s.)		(n.s.)	
set 4	Nangari (rep. 1)		.22	-.64***	
set 4	Nangari (rep. 4)		.49*	-.34	
set 4	Palmer	-.27		-.65	
set 5	Nangari		-.04	-.13	
set 5	Perponda		.14	.34	
set 5	Strathalbyn	.80**			

differences (P) in associations between cultivars or replicates are bracketed

significant associations are depicted as * (P<0.05), ** (P<0.01) or *** (P<0.001)

† associations are pooled from within cultivars for set 2 and from within replicates for set 3

cumulatively yet has been examined here with bivariate tests of associations. While this is limiting and though data are incomplete, associations allow comparisons between experiments in the course of disease.

The most complete data are for wheats of set 2, which shows disease to be more widespread and damaging at Strathalbyn than at Palmer (Tables 13 and 32). Loss of yield at Strathalbyn appeared largely due to a reduction in number of fertile heads in plots (Table 33) that followed the early and extensive infection of seminal roots by G.g.t. (Table 32). Data showed deadheads to be a poor indicator of yield of grain at Strathalbyn, since associations between these variables were inconsistent within cultivars (Table 33). This finding did implicate earlier and more extensive damage to plants than that expressed as deadheads. There was no direct evidence that infection with G.g.t. had reduced yield of grain at Palmer (Table 32), but yield had varied with the incidence of deadheads and weight of grain (Table 33) which were significantly associated with the infection of roots with G.g.t. (Tables 23 and 31). Data indirectly suggested loss of yield had occurred late in the development of the crop at Palmer and was perhaps due more to infection of coronal roots (Table 32) and shrivelling of grain than early death of heads (Table 33).

While less complete, data of families of set 3 allow a comparison of disease in soils of type Dr4.63 and Gc1.22. Weight of dried shoots at anthesis appeared to be

significantly reduced by infection of seminal roots with G.g.t. in both types of soil (Table 21). Plants grown in soil of type Dr4.63 did not recover vigour lost by anthesis, since final yield of grain was associated with infection of the coronal roots by G.g.t. In contrast, plants grown on soil of type Gc1.22 appeared to subsequently recover from damage at anthesis, since disease of roots did not influence final yield of grain. While infection with the unknown pathogen was associated with deadheads (Table 23) in this soil, they were infrequent (Table 25) and did not account for significant loss of yield of grain (Table 33). These findings suggest disease had developed on coronal roots in soil of type Dr4.63 but not in soil of type Gc1.22. A small loss of yield in soil of type Dr4.63 also appears due to incomplete filling of grain (Table 33) associated with infection of the coronal roots with the unknown pathogen (Table 31).

Data of wheats of sets 4 and 5 were incomplete and bivariate associations are not independent of differences between cultivars. Nevertheless disease in this material did not appear to be dissimilar to that in wheats similarly grown at Palmer and Strathalbyn. While loss of yield could not be associated with the incidence of G.g.t. at Nangari and Perponda, a high incidence of deadheads was observed at these sites, and the pathogen was most prevalent. Moreover replicates one and four of the experiment with cultivars of set 4 at Nangari appeared to differ in expression of disease as yields of plots were dissimilarly

associated with deadheads (Table 33) yet pathogens were equally prevalent (Table 12).

Discussion

Disease at the site of Palmer appeared to be typical of disease caused by G.g.t. in South Australia in that deadheads were frequent while severe stunting and premature death of tillers were not, in contrast to disease at Strathalbyn. However both sites contained a similar number of units of inoculum of G.g.t. in assays three months before sowing. The severe disease at Strathalbyn probably followed the buildup in inoculum over several months on roots of grassy weeds that had germinated after unseasonally heavy rain in March and April (Appendix 2). Similar weeds were controlled at Palmer. Data show G.g.t. can not be considered in isolation as a pathogen in the field, as two other pathogens which were frequently observed at sites were found to significantly influence the growth of wheat in one experiment, and appeared to have been antagonistic to infection of roots by G.g.t.

In this study, findings suggested infections of roots were more significant in contributing to loss of yield in wheats grown in the field than infection of other components of the root system. Infection of the subcoronal internode was less strongly associated with growth of plants and yield of grain in plots than infection of roots. Where infection of the subcoronal internode was closely associated with damage to plants (as at Strathalbyn), roots of crops were already extensively diseased, a condition which may have led to most loss of vigour of the

crop. These findings reflect greater resistance in the subcoronal internode to infection with G.g.t. than exists in roots (Fellows, 1928). While not studied, the coronal tissues are even more resistant to infection than the subcoronal internode, and their infection can be expected to be less significant to loss of yield in wheats grown in the field than infection of roots.

Provided there is moisture throughout the soil profile, as there was at Palmer and Strathalbyn in 1983 (Appendix 2), both seminal and coronal roots contribute to yields of wheats. However, seminal roots develop first and are therefore significant in contributing to earlier growth of plants than coronal roots which develop later and influence late growth of plants. Thus weight of dried shoots at anthesis was influenced more at Palmer by infection with G.g.t. of seminal roots than coronal roots (Table 21) (most seminal roots being infected at Strathalbyn), while weight and yield of grain were influenced more by infection of coronal roots than seminal roots at both sites (Tables 31 and 32).

Deadheads were not consistently associated with infection of either seminal or coronal roots in this study, suggesting their formation to be inconsistent as concluded earlier in this chapter. Indeed, two conditions have been described by White (1947) in which deadheads form. Both conditions require the seminal roots to be extensively infected with G.g.t. and unable to function. In the first condition, the coronal roots are poorly developed and are unable to supply maturing plants with moisture as the upper profile of the soil dries at the end of the growing season.

The second condition was also described by Bockmann (1966), in which the coronal roots are also extensively infected with G.g.t. and unable to supply plants with moisture that may be freely available throughout the soil. Deadheads formed at Strathalbyn in conditions described by White (1947) and Bockmann (1966), where moisture was abundant in the soil when the crop matured, and where seminal and coronal roots were extensively infected with G.g.t. However, these conditions had also led to earlier and severe damage to young plants, and therefore may not be representative of disease that is of common occurrence in South Australia. Deadheads were also frequent at Palmer where coronal roots were not extensively infected with G.g.t. (Tables 13 to 15) yet where moisture was abundant throughout the soil profile (Appendix 2). This condition had not been described by White (1947) to account for the development of deadheads. Conditions described by White (1947) also fail to account for the positive association between earliness of maturity and incidence of deadheads found in this study and previously by Wachter and Mogling (1979). However, findings at Palmer may be explained if deadheads follow earlier stress to crops than is assumed by White (1947), and at a specific stage of development of plants. If this stage of development were to occur before the coronal roots of wheats have developed extensively, deadheads may follow stress to plants when sufficient seminal roots are infected with G.g.t., despite an abundance of soil moisture. Moreover, fewer deadheads may develop in later maturing wheats whose seminal roots

are similarly infected with G.g.t., if the critical stage of development occurs when coronal roots are extensively developed. Early stress of this nature may widely cause deadheads in wheats as at Palmer in 1983, as positive associations between earliness of maturity and incidence of deadheads were also observed at Nangari in 1981, Strathalbyn in 1983, and elsewhere by Wachter and Mogling (1979). Stress that occurs later again may simply restrict filling of grain as at Palmer in 1983 (Table 31). Differences in the incidence of deadheads have previously encouraged belief of strong resistance to infection by G.g.t. in wheat (Rathjen, pers. comm.), but this has been shown to be unwarranted. Moreover, earliness of maturity can not be freely altered in breeding wheats for tolerance to infection by G.g.t. as it strongly determines yields of healthy wheats in South Australia (Rathjen, pers. comm.).

Wheats infrequently differed in infection of roots with G.g.t., while differences were not influenced by any of the characters studied (Table 19). In addition, wheats were not dissimilarly infected with G.g.t. at the sites of study (Table 13), unlike wheats infected with introduced inoculum (Scott, 1981). These findings may simply reflect study at only two sites, but may also suggest cultivars of wheat are more consistently infected with G.g.t. when sown in fields containing inoculum of natural occurrence, as was found by Nilsson (1969). While a cultivar x site interaction was detected for the incidence of deadheads in wheats of set 2 at sites in 1983, disease as at Strathalbyn does not occur widely in South Australia.

Previous studies in Australia have not demonstrated cultivar x site interactions for the incidence in wheats of deadheads caused by *G.g.t.*

CHAPTER 5

A COMPARISON OF STUDIES IN CONTROLLED AND FIELD ENVIRONMENTS

The object of this project is to investigate resistance to infection by G.g.t. in wheat, but until now wheats have not been shown to clearly differ in resisting this pathogen. While wheats grown in cups differed in infection with G.g.t. (Chapter 3), differences varied with weight of seed. In addition, wheats differed little in infection with G.g.t. in the field. However, effects that influence infection of wheats with G.g.t. pertain to the host when shown to be independent of environment in which roots are grown and infected. Such effects are in turn revealed by significant associations between measures of infection in cultivars of wheat grown in cups and in the field. Effects pertaining to the host and revealed by the significance of associations, include resistance to invasion by G.g.t. Where measures of infection from seminal and coronal roots are also found to be associated, evidence is also provided of resistance that operates in both root systems of the wheat plant. In addition, associations between histological responses of the host to infection that are measured in controlled conditions, and infection of wheats in the field provide evidence for mechanisms of resistance. Associations between infection of wheats with G.g.t. in a controlled environment and growth and yields of wheats in the field, test the potential use of resistance in future breeding programs.

Thus associations were determined between data of cultivars of four sets of genotypes (sets 2 to 5). Data of infection with G.g.t. and of cortical browning for wheats grown in cups were from earlier experiments (Chapter 3) and

an experiment summarised in Appendix 1. Measures of infection with pathogens and of growth and yield of cultivars grown at sites in the field were from experiments described in Chapter 4. Where wheats were infected with G.g.t. in cups in separate experiments, duplicate associations were pooled when found to be homologous. In addition, associations from Palmer and Strathalbyn were pooled for wheats of set 2 to test effects that are independent of field sites. To obtain measures of association that were independent of differences between sets of wheats in adaption to field conditions in South Australia, associations within sets were tested for homology and then pooled. Data were contributed to the pooled associations according to the number of wheats in each set.

Factors influencing infections of T. aestivum in the field

Associations were determined between measures of infection with G.g.t. from roots grown in cups and symptoms of infection with pathogens from roots from field sites. Results are presented in Tables 34 to 38. Sets of associations were not complete but were limited by incomplete data from cups and by variation in the occurrence of pathogens at sites.

(1) Infection of seminal roots with G. graminis var tritici at tillering

The early infection of seminal roots of wheat in field environments appeared to be negatively and significantly

TABLE 34: Associations between scores of infection with G.g.t. for cultivars grown in cups and infection of seminal roots with the pathogen at tillering in field experiments

Experiment		Scores of infection from cups §									Number of cultivars
cultivars	site	ECTG	CCOL	ECOL	SC	LN	MLS	LL	CL	CI	
set 2	Palmer	-.22	-.32	-.93***	-.92***	-.80**	-.16	-.90**	.41	.54	7
	Strathalbyn	-.55	-.49	-.11	.10	-.04	.15	-.08	-.33	-.50	7
mean of set 2		-.40	-.41	-.70**	-.63**	-.52*	-.01	-.65*	.04	.03	
set 3	Palmer	-.11	-.34*	-.11	-.22	.01			-.17	-.15	35
set 4	Palmer					-.70	-.30		.46	.15	8
set 5	Strathalbyn					-.35	-.12		.01	.05	14
overall mean		-.17	-.35*	-.26	-.32*	-.22	-.12	-.65*	-.05	-.08	
differences in associations (P)		n.s.	n.s.	0.05	0.05	n.s.	n.s.	n.s.	n.s.	n.s.	

significant associations are depicted as * (P<0.05), ** (P<0.01) or *** (P<0.001)

§ abbreviations of scores are explained on page 16

associated with measurements of the radial invasion by hyphae into roots grown in cups (Table 34). Associations were most significant at Palmer and may have been disrupted by antagonism with R. solani at Strathalbyn. The ectotrophic growth of hyphae down roots in the controlled environment was not associated with the early infection of seminal roots in the field. Associations with the discoloration of walls of cortical cells in the controlled environment were not significant and were without general trend. The inverse relationship between infection of seminal roots with G.g.t. in controlled and field environments can not be readily explained, but in being significant, provides evidence for resistance to radial invasion by the pathogen into roots of wheat. A significant association was found with colonisation of the innermost cortical cells with G.g.t. in roots of families of set 3 when grown in cups. This provides evidence of genetic control in the cross Aus1080 x Condor for both characters (CCOL & GSEM at tillering) as families of set 3 must differ in both respects for a significant association to have been detected.

(2) Infection of seminal roots with G. graminis var tritici at anthesis

Significant and positive associations were detected between infection of seminal roots with G.g.t. at anthesis in field environments and the extent of ectotrophic growth by hyphae down roots grown in cups (Table 35). While associations between infection of seminal roots with G.g.t.

TABLE 35: Associations between scores of infection with G.g.t. for cultivars grown in cups and infection of seminal roots with the pathogen at anthesis in field experiments

Experiment		Scores of infection from cups[§]									Number of cultivars
cultivars	site	ECTG	CCOL	ECOL	SC	LN	MLS	LL	CL	CI	
set 2	Palmer	.30	-.19	-.23	-.24	-.11	.51	-.19	-.23	.10	7
set 3	Palmer	.36*	-.22	-.21	-.17	-.04			.17	.24	35
set 5	Strathalbyn					-.57*	-.40		.01	-.25	14
overall mean		.35*	-.22	-.21	-.18	-.19	-.16	-.19	.10	.12	
differences in associations (P)		none significant									

significant associations are depicted as * ($P<0.05$)

§ abbreviations of scores are explained on page 16

in the field at anthesis and measurements of radial invasion by hyphae into roots grown in cups tended to be negative, they were not significant (except GSEM x LN for cultivars of set 5 at Strathalbyn). While not broadly based, these findings suggest infection of older previously uninfected seminal roots (at anthesis) was determined more by the spread of hyphae along adjacent infected roots, than by the radial entry of hyphae into roots from natural inoculum on plant residues from previous growing seasons. In turn, this may reflect greater resistance to infection by G.g.t. in seminal roots at anthesis than at tillering, which concurs with the findings of Robertson (1932). Associations between the infection of seminal roots with G.g.t. at anthesis and cortical browning were again not significant.

(3) Infection of coronal roots with G. graminis var tritici at anthesis

Associations between the infection of coronal roots with G.g.t. at anthesis and growth of hyphae down roots grown in cups were not significant (Table 36). However, associations with measures of radial invasion by hyphae into roots grown in cups were significant and positive for cultivars of set 2 but not for families of set 3 (Table 36). This may partly follow the detection of significant differences in infection of coronal roots with G.g.t. at anthesis between cultivars of set 2 (Table 13) but not between families of set 3. Moreover, the finding (Table 36) provides evidence for a common factor to influence invasion

TABLE 36: Associations between scores of infection with *G.g.t.* for cultivars grown in cups and infection of coronal roots with the pathogen at anthesis in field experiments

Experiment		Scores of infection from cups[§]									number of cultivars
cultivar	site	ECTG	CCOL	ECOL	SC	LN	MLS	LL	CL	CI	
set 2	Palmer	.13	-.06	.34	.37	.35	.65	.33	-.58	-.43	7
set 2	Strathalbyn	.10	.17	.80**	.88***	.73*	.52	.82	-.57	-.60	7
mean of set 2		.12	.06	.62**	.71**	.57*	.59	.64	-.57*	-.52	
set 3	Palmer	.05	-.11	.01	.06	-.06			-.11	-.09	35
set 4	Palmer					.19	.19		-.54	-.69	8
set 5	Strathalbyn					-.05	.16		.14	.10	14
overall mean		.06	-.08	.15	.22	.07	.33	.64	-.18	-.19	
differences in associations (P)		n.s.	n.s.	n.s.	0.05	n.s.	n.s.	n.s.	n.s.	n.s.	

significant associations are depicted as * ($P<0.05$), ** ($P<0.01$) or *** ($P<0.001$)

§ abbreviations of scores are expalined on page 16

by G.g.t. in both seminal and coronal roots. Data of coronal roots from the field show a tendency for the innermost layer of cortical cells to differ from underlying tissue in infection with G.g.t. at anthesis (Table 36). Similar differences between tissues in infection with G.g.t. were not observed in seminal roots at anthesis Table 35), and suggest seminal and coronal roots of wheat differ in resisting invasion by G.g.t. across the endodermis. This hypothesis may be related to observations by Robertson (1932) that hyphae in the cortex grow parallel to the vascular axis of coronal roots near the crown, while growing radially into the cortex of seminal roots near the scutellar node. Associations between the infection of coronal roots with G.g.t. at anthesis and brown discolorations of the cortex in roots grown in cups tended to be negative, while the association CL x GCOR was significant over sites for wheats of set 2. Thus, coronal roots tended to be least infected with G.g.t. in the field for cultivars whose seminal roots had most cortical browning when infected with the pathogen in cups. This finding provides evidence that cortical browning retarded invasion by G.g.t. into coronal roots in the field, as it appeared to have in seminal roots grown in cups (Table 11). Evidence was not similarly found for cortical browning to have retarded invasion by G.g.t. into seminal roots grown in the field (Tables 34 & 35). This anomaly may reflect the greater incidence of cortical browning in coronal roots than in seminal roots of field grown wheats (personal observation).

(4) Infection of seminal roots with R. solani at tillering

Associations between infection of seminal roots with R. solani at tillering and measures of radial invasion by hyphae into seminal roots grown in cups were not consistent but were significant and negative with colonisation of the

TABLE 37: Associations between scores of infection with G.g.t. for cultivars grown in cups and infection of seminal roots with R. solani at tillering in field experiments

Experiment		Scores of infection in cups§									Number of cultivars
cultivars	site	ECTG	CCOL	ECOL	SC	LN	MLS	LL	CL	CI	
set 2	Palmer	.37	.49	.15	.01	.11	-.58	.06	.40	.31	7
set 2	Strathalbyn	.36	.21	.48	.42	.37	.36	.52	-.13	-.01	7
mean of set 2		.36	.36	.33	.22	.24	-.14	.31	.15	.15	
set 3	Palmer	.12	-.20	-.34*	-.20	.15			.24	.38*	35
set 4	Palmer					-.38	-.44		.36	.24	8
set 5	Strathalbyn					.18	.18		-.13	-.22	14
overall mean		.17	-.09	-.21	-.12	.12	-.06	.31	.17	.23	
diferences in associations (P)		none significant									

significant associations are depicted as * ($P<0.05$)

§ abbreviations of scores are explained on page 16

endodermis for families of set 3 (Table 37). The significance of this finding is not clear, despite associations between infection with G.g.t. of seminal roots grown in the field and in cups being similarly negative. Associations between brown discolorations of the cortex for roots grown in cups and the infection of seminal roots with R. solani at tillering were positive at Palmer (and significant for CI x RSEM for families of set 3), but not at Strathalbyn where there was strong antagonism with G.g.t. This finding was not expected as earlier evidence suggested cortical browning was associated with resistance to infection by G.g.t. in roots (Table 11), so that a similar finding could be expected for infection with other pathogens and negative associations observed.

(5) Infection of roots with R. solani and the unidentified pathogen at anthesis.

Associations were not significant and were without trend (Table 38).

Factors influencing yields of T. aestivum in the field

Associations were determined between infection and symptoms of infection with G.g.t. in roots of wheats grown in cups and yields of cultivars in the field, and the

TABLE 38: Associations between scores of infection with G.g.t. for cultivars grown in cups and infection of roots with pathogens other than G.g.t. at anthesis in field experiments

Experiments		Scores of infection in cups[§]									Number of cultivars
cultivars	site	ECTG	CCOL	ECOL	SC	LN	MLS	LL	CL	CI	
infection of seminal roots with R. solani											
set 2	Strathalbyn	-.24	-.17	-.54	-.60	-.62	-.68	-.70	.32	.28	7
infection of coronal roots with R. solani											
set 2	Strathalbyn	-.32	-.05	.42	.49	.23	-.22	.38	-.14	-.39	7
infection of coronal roots with the unknown pathogen											
set 2	Palmer	.17	.39	.54	.49	.40	.34	.42	-.34	-.22	7
set 3	Palmer	-.00	.21	.08	.02	-.14			.02	-.04	35
set 4	Palmer					.65	.44		-.51	-.35	8
set 5	Strathalbyn					-.18	.10		.33	.42	14

associations were not significant

§ abbreviations of scores are explained on page 16

findings are presented in Tables 39 to 43. Sets of associations were not complete being limited by incomplete data from cups and by the absence of observations of yields from some field sites. Data of field experiments were excluded where infection with G.g.t. was not damaging to wheats. Thus data of families of set 3 were only of wheats grown in soil of type Dr4.63.

(1) Weight of dried shoots at anthesis

Associations were not consistent over all data though occasionally significant (Table 39).

(2) Number of fertile tillers in plots

As before associations were not consistent over the data, though occasionally significant (Table 40).

(3) The incidence of dead and empty heads

Associations between the incidence of dead and empty heads and the extent of ectotrophic growth down roots by hyphae of G.g.t., tended to be negative and on one occasion significant (Table 41). This finding was not expected, and may have occurred through chance. Associations between the incidence of dead and empty heads and measures of the rate of radial invasion by hyphae into seminal roots were positive and significant, and associations with the extent of cortical browning in roots were negative and significant for cultivars of set 2. However, deadheads and measures of resistance to G.g.t. may not be causally associated in these wheats.

TABLE 39: Associations between scores of infection with G.g.t. for cultivars grown in cups and weight of dried shoots at anthesis in field experiments

Experiment		Scores of infection in cups[§]									number of cultivars
cultivars	site	ECTG	CCOL	ECOL	SC	LN	MLS	LL	CL	CI	
set 2	Palmer	-.13	-.32	.01	.14	.11	.88*	.35	-.07	.06	7
set 2	Strathalbyn	-.56	-.52	-.27	-.12	-.29	.57	-.22	-.37	-.25	7
set 3	Palmer	-.27	-.23	-.00	-.03	.17			.01	-.07	35
set 4	Palmer					-.71*	-.70		.59	.52	8
set 5	Strathalbyn					.47	.31		-.69*	-.62	14
overall mean		-.29	-.27	-.03	-.05	.11	.29	.07	-.13	-.14	
differences in associations (P)		n.s.	n.s.	n.s.	n.s.	n.s.	0.01	n.s.	0.05	n.s.	

significant associations are depicted as * ($P<0.05$)

§ abbreviations of scores are explained on page 16

TABLE 40: Associations between scores of infection with G.g.t. for cultivars grown in cups and number of fertile tillers per plot in field experiments

Experiments		Scores of infection in cups§									Number of cultivars
cultivars	site	ECTG	CCOL	ECOL	SC	LN	MLS	LL	CL	CI	
set 2	Palmer	.18	.68*	.28	.05	-.46	-.69	.03	.02	.25	7
set 2	Strathalbyn	-.21	-.05	.30	.31	.10	-.09	-.02	-.65*	-.67	7
set 4	Nangari (Rep. 1)					.34	.18		-.07	.05	28
set 4	Nangari (Rep. 4)					-.05	-.01		-.21	-.21	28
set 5	Nangari					.45	.17		.15	.10	12
set 5	Perponda					.32	.16		.29	.47	11
overall mean		-.03	.59	.29	.18	.20	.03	0.01	-.11	-.02	
differences in associations (P)		n.a.	0.05	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	

significant associations are depicted as * ($P<0.05$)

§ abbreviations of scores are explained on page 16

TABLE 41: Associations between scores of infection with G.g.t. for cultivars grown in cups and incidence of dead and empty heads in plots in field experiments

Experiments		Scores of infection in cups[§]									Number of cultivars
cultivars	site	ECTG	CCOL	ECOL	SC	LN	MLS	LL	CL	CI	
set 2	Palmer	-.05	-.10	.55	.66	.56	.60	.62	-.58	-.62	7
set 2	Strathalbyn	-.42	-.10	.47	.58	.34	.32	.39	-.56	-.66	7
mean of set 2		-.24	-.10	.50*	.61*	.44	.47	.51	-.57*	-.64*	
set 3	Palmer	-.38*	-.27	-.02	-.02	-.07			-.05	-.05	35
set 4	Palmer					.42	-.29		.57	.35	8
set 4	Nangari (Rep. 1)					-.02	-.21		-.20	-.17	28
set 4	Nangari (Rep. 4)					.09	-.16		-.01	.06	28
set 5	Nangari					.73*	.06		.26	.33	12
set 5	Perponda					-.27	-.52		.27	.26	11
overall mean		-.35*	-.24	.10	.13	.11	-.14	.51	-.02	-.02	
differences in associations (P)		none significant									

significant associations are depicted as * ($P<0.05$)

§ abbreviations of scores are explained on page 16

As the more resistant wheats in set 2 tended to be exotic and late in maturity, their infrequent incidence of dead-heads was more probably a product of their lateness than of their resistance to infection by G.g.t. that had been observed in cups (Table 8). This argument also applies to associations with discolorations of the cortex.

(4) Weight of grain

Associations between weight of grain of wheats grown at Palmer and the extent of distinct cortical browning down seminal roots grown in cups were positive (Table 42) and significant ($P<0.01$). A direct relationship between these variables is not obvious. However, an indirect relationship may have followed the influence of cortical browning in retarding invasion by G.g.t. into coronal roots of field grown wheats (Table 36), which in turn appear to have to most significantly influenced filling of grain at Palmer (Table 31). At first this hypothesis appears to be weak, as associations between cortical browning and filling of grain (Table 42) were stronger than associations with infection of coronal roots (Table 36). However, more significant associations between cortical browning and extent of infection with G.g.t. in coronal roots may have been obscured by the necessity for infection to initiate this form of resistance to the pathogen. Thus, coronal roots that were infected with G.g.t., frequently had discoloured cortices but not blackened steles, indicating cortical but not stelar invasion by the pathogen, and in turn suggesting partial function of infected roots (Figure 13). This is unlike seminal roots, where symptoms of infection with G.g.t. usually included stelar lesions, which indicate points of dysfunction of vascular tissue.

TABLE 42: Associations between scores of infection with G.g.t. for cultivars grown in cups and weight of grain in field experiments

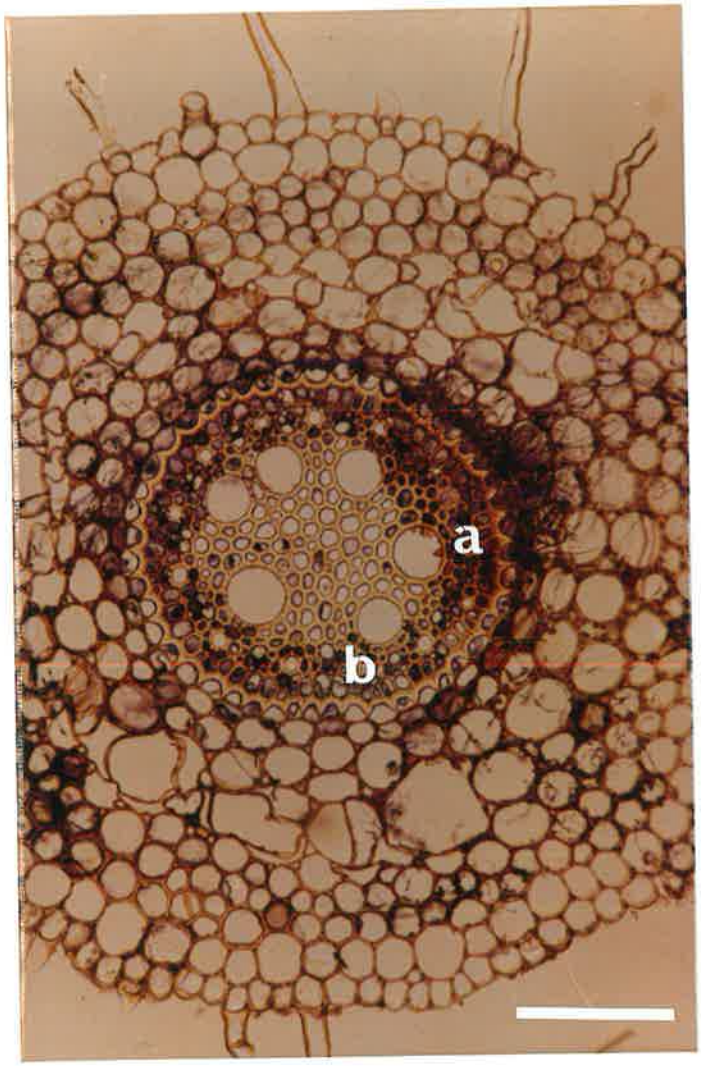
Experiments		Scores of infection in cups[§]									Number of cultivars
cultivars	site	ECTG	CCOL	ECOL	SC	LN	MLS	LL	CL	CI	
set 2	Palmer	.06	.13	-.04	-.07	-.10	-.70	.07	.63	.33	7
set 3	Palmer	-.14	.20	.20	.20	.23			.39*	.29	35
overall mean		-.12	.19	.17	.17	.20	-.70	.07	.43**	.29	
differences in associations (P)		none significant									

significant associations are depicted as * (P<0.05) or ** (P<0.01)

§ abbreviations of scores are explained on page 16

FIGURE 13: Section of a coronal root of wheat with a discoloured cortex but without a blackened stele at anthesis at Palmer, showing vascular tissue that is colonised (a) and not colonised (b) by hyphae

(Scale bar = 0.25 mm)


a
b

(5) Yield of grain

When grown in cups, exotic wheats of set 2 appeared more resistant than local cultivars to infection with G.g.t., though yielding poorly in the field. However, associations between yield of grain and measures of radial invasion of roots by hyphae of G.g.t. tended to be positive and significant over all the data (Table 43). Resistance to radial invasion by hyphae of G.g.t. is unlikely to have influenced yield of grain by influencing infection of roots with G.g.t. If this had been so, measurements of infection of stelar tissue of roots grown in cups should be negatively associated with yield, since these measures of infection were positively associated with the infection of coronal roots in the field (Table 36) which in turn most influenced yield of grain in plots (Table 32). Thus there is some evidence that resistance to infection by G.g.t. is associated with an effect that is detrimental to yield of grain in wheat. Associations between yield of grain and cortical browning tended to be negative but were not significant (Table 43).

Discussion

Evidence was found in this chapter, of differences between wheats in resistance to infection by G.g.t. Two forms of resistance were studied in roots, as were their influence on growth of wheats in the field. In Chapter 3, hyphae that invaded radially into roots were found to be resisted by factors in the cortex and endodermis. Such resistance appeared to be general in root tissues as infection of roots in the field was variously associated

TABLE 43: Associations between scores of infection with G.g.t. for cultivars grown in cups and yield of grain in field experiments

Experiments		Scores of infection in cups[§]									Number of cultivars
cultivars	site	ECTG	CCOL	ECOL	SC	LN	MLS	LL	CL	CI	
set 2	Palmer	.13	.23	.41	.37	.34	-.38	.11	-.47	-.61	7
set 2	Strathalbyn	-.32	-.01	.26	.27	-.01	-.42	-.16	-.56	-.66	7
set 3	Palmer	-.23	.14	.31	.32	.33*			.11	.06	35
set 4	Palmer					.66	.12		-.39	-.27	8
set 4	Nangari (Rep. 1)					.29	.34		.02	.16	28
set 4	Nangari (Rep. 4)					-.08	.24		-.18	-.20	28
set 5	Strathalbyn					.36	.43		-.28	-.11	14
set 5	Nangari					-.07	.30		-.55	-.59	12
set 5	Perponda					.34	.16		-.00	-.12	11
mean of all data		-.21	.13	.32*	.32*	.23*	.23*	-.03	-.13	-.12	
differences in associations		none significant									

significant associations are depicted as * ($P<0.05$)

§ abbreviations of scores are explained on page 16

with colonisation of the cortex, endodermis and stele of roots grown in cups. Wheats were also found to differ in resistance to radial invasion by hyphae when only six days old (Table 8), when cell walls are thin and not yet lignified (Chapter 3). This suggests a biochemical rather than a mechanical basis for this form of resistance, an hypothesis which can account for observations by Nilsson (1969), that extracts of freshly ground roots of wheats differentially influenced growth of G.g.t. in vitro. However, this form of resistance did not appear to uniformly affect seminal roots, since cultivars of wheat were inversely ranked for infection when grown in cups, to when infected with G.g.t. at tillering in the field. While influencing infection of seminal roots in the field at tillering, this form of resistance had little effect at anthesis, when infection of seminal roots appeared to be most influenced by ectotrophic growth of hyphae. In contrast, wheats were similarly ranked for infection with G.g.t. of coronal roots grown in the field at anthesis, and seminal roots grown in cups. Overall, the dissimilar effects of this form of resistance on infection of roots can not be readily explained, but may reflect differences in the morphology of seminal and coronal roots, and differences in the microflora of roots grown in cups and in the field. Further, in dissimilarly influencing infection of seminal and coronal roots grown in the field, this form of resistance may not consistently influence loss of yield that is caused by the pathogen. In this study, resistance to radial invasion by hyphae was

negatively associated with yield of grain, but these variables are unlikely to be causally linked (Chapter 4).

The other form of resistance to infection by G.g.t. was associated with a brown discoloration and thickening of walls of cortical cells that occurred as cells were invaded by hyphae of the pathogen (Chapter 3). As cortical browning was a response to infection by G.g.t., and was restricted to cortical tissue, it appears to be a separate form of resistance to that discussed above. Evidence was found for cortical browning to influence infection with G.g.t. of coronal but not seminal roots grown in the field. Thus, cortical browning appeared to only influence loss of yield due to disease that occurred late in the growth of the wheats. Nevertheless, cortical browning was not significantly associated with overall yield of wheat in the current data, and further work appears necessary to establish its value in reducing loss of yield caused by infection with G.g.t. Neither of the forms of resistance that were studied in this chapter were strongly associated with the colonisation of wheat roots with other pathogens, suggesting both to be specific to infection by G.g.t.

As measures of bivariate association test the degree to which variation in two variables is related, they also test the existence of variation in both. Thus, significant associations between mean infection scores for cultivars grown in cups and mean infection of seminal roots with G.g.t. at tillering (Table 34) and anthesis (Table 35), and with R. solani at tillering (Table 37), provide evidence that seminal roots of wheats grown in the field had differed in susceptibility to these pathogens. As these differences were not detected earlier by analysis of variance, they are therefore smaller than the differences between wheats in susceptibility of coronal roots to infection with G.g.t. (Table 13).

than differences in infection with G.g.t. of coronal roots that were detected earlier by analysis of variance (Table 13).

Associations between findings from cups and the field detected effects that were independent of both environments. Similarly, consistent infection with G.g.t. in both environments provides evidence of resistance to the pathogen in individual cultivars of wheat. Thus, findings from cups (Appendix 1 and Table 8) and the field (Tables 13-16) suggest wheats currently grown in Australia (sets 4 and 5) differ little in resistance to infection by G.g.t., while larger differences are found in wheats of set 2. Chile909 appeared to be the most resistant wheat of set 2 and Rac311 the least resistant to infection by G.g.t., while the other wheats could not be clearly ranked. In particular, Chile911 was as little infected with G.g.t. as Chile909 when grown in cups, but was as extensively infected as Rac311 when grown in the field. Thus, the method that was used in this study to infect roots with G.g.t. in cups, did not accurately indicate infection of individual wheats in the field. Improvement of the cup test may be possible if seedlings are grown in less coarse sand and from seed of another weight to that used in this study, or if infection were to be measured in coronal rather than seminal roots.

CHAPTER 6

GENERAL DISCUSSION

The current study is precursory to further work as it has largely relied on tests of association which do not demonstrate causal effects. However, its strength was to detect effects that were not specific to laboratory environments or to field sites, and could thus provide evidence of resistance to infection by G.g.t. in wheat. However, the current work provided a restricted study of resistance to infection by G.g.t. in wheat, as findings were limited to resistance that was expressed in tissues that were infected with the pathogen in the controlled environment, that is young seminal roots. Thus, resistance that is principally expressed in more mature tissues and in coronal roots was excluded from study, though coronal roots were found to differ from seminal roots in mode of infection with G.g.t. (Chapter 5). Moreover, infection with G.g.t. was measured as the extent of invasion by hyphae into roots grown in cups, so that resistance was largely detected in tissues that retard such invasion, which are the cortex and endodermis. Nevertheless, findings show resistance in the cortex of roots to significantly influence the infection of cultivars of wheat with G.g.t., though this tissue is the least resistant of the tissues of the root system of the wheat plant (Fellows, 1928). However, the cortex is the first tissue to be colonised in soil, and is important in influencing the rate at which the more resistant vascular tissues are exposed to infection.

Evidence was found for two forms of resistance in wheat roots :- a general form of resistance that appeared early in many of the tissues of roots and was not of mechanical origin, and cortical browning that appeared due to the thickening and lignification of cell walls in the cortex of roots infected with G.g.t. It can be expected that the general form of resistance is associated with living cells (Deacon & Henry, 1980), as is cortical browning. Thus it is significant that cortical cells progressively die as the role of the root changes from one of absorption to one of conduction of nutrients and water (Holden, 1976). This may account for the general form of resistance being associated with infection in seminal roots at tillering, but not at anthesis when cortical cells have largely died (Holden, 1976), and another form of resistance of greater strength has developed with aging of stelar tissue (Fellows, 1928). Similarly, cortical browning is infrequent in seminal roots in the field as cortical cells die relatively rapidly. The greater incidence of cortical browning in coronal roots from the field and evidence of the general form of resistance to infection by G.g.t. persisting in their tissues to anthesis, suggests the progressive death of cortical cells was less rapid in coronal than in seminal roots. This deduction can not be presently tested as rates of natural cell death in seminal and coronal roots have not been compared in studies to date. Nevertheless, further evidence that cortical cell death influences resistance to root pathogens in wheat is provided in an earlier study by Deacon and Lewis (1982), who found an association between

the rate at which cortical cells in seminal roots progressively died and resistance to infection by Cochliobolus sativus (Ito & Kurib.) Drechsler ex Dastur.

In the current study, resistance was assessed for use in improving field resistance of wheats to infection by G.g.t. This was attempted by observing the influence of resistance on yield of grain for wheats with disease caused by G.g.t., and by observing inheritance of resistance in a wheat cross. Previous field studies of loss of yield in wheats infected with G.g.t. have used introduced inoculum and have not differentiated between tolerance and resistance to the pathogen. Such studies have not consistently induced disease, leading to strong environment x genotype interactions in findings. By infecting wheats with virulent isolates of G.g.t. that are grown on exceptionally large food bases, seedlings are prone to be more severely diseased than when naturally infected in the field (Chapter 4). Damage to seedlings then tends to mask loss of yield that occurs late in the growth of crops, which is more representative of disease of natural occurrence in South Australia.

This study was unique in investigating the effect of resistance that exists in wheat on yields of wheats that were naturally infected with G.g.t. However, these investigations were restricted by the wheats studied, since wheats grown in Australia (sets 4 and 5) varied little in infection with G.g.t. (Chapter 5). Thus, resistance was largely seen in the seven wheats of set 2, of which many were exotic and yielded poorly, and the F_2 families of

set 3 which had exotic characters and had not been selected for yield of grain in any environment. Thus, the effect of resistance on yield is poorly tested in this study, as wheats that yield well tend to impose greater demand on their root system when grain is filling than wheats that yield poorly, and thus are likely to be more sensitive to disease of roots than were some wheats of set 2. Resistance to radial invasion by hyphae of G.g.t. that was general in root tissues was seen most clearly in wheats of set 2 where it appeared to promote more severe infection of seminal roots but less severe infection of coronal roots. However, this form of resistance was not simply inherited and tended overall to be negatively associated with yield of grain, and therefore seems of little future use in the breeding of wheats. Nevertheless, resistance to infection by G.g.t. that is expressed as cortical browning appeared to promote filling of grain in diseased wheats. Furthermore, cortical browning appeared to be simply inherited when expressed in seminal roots, and if similarly expressed in coronal roots, may be readily incorporated into Australian wheats and may improve their field resistance to G.g.t. However, further work is needed with wheats that have been selected for high yield in local conditions to determine the extent to which improvement by this means may be effected.

While resistance that is expressed in seminal and coronal roots was detected in this study, additional forms of resistance that are only expressed in coronal roots may exist. Such resistance should be investigated, as infection of coronal roots was most strongly associated with yield of grain in wheats that expressed disease of frequent occurrence in South Australia, and as field grown wheats differed more in infection of coronal roots than seminal roots (Chapter 4). Thus, infection of coronal roots with G.g.t. should be studied in

detail in a controlled environment as were seminal roots in the current project. However, infection of coronal roots will be more difficult to study than was infection of seminal roots, whose timing and rate of emergence can be controlled to allow highly uniform infection. When forms of resistance to infection by G.g.t. that exist in wheat have been more extensively investigated than in this study, sources of resistance can be better sought and forms of resistance better evaluated than at present.

Higher levels of resistance to G.g.t. have been found in species related to wheat, in particular oats and rye. While resistance in oats appears to be due to avenacin (Turner, 1961), a substance that inhibits the growth of G.g.t., little is known of the mechanisms of resistance in rye. However, resistance from related species may not be durable in cultivars of wheat that are widely grown, as some isolates of G.g.t. are pathogenic on oats (as is G. graminis var avenae) while others are equally pathogenic on wheat and rye (Scott, Hollins & Gregory, 1985). Moreover, these characters are not expressed in wheat-rye (Scott, 1981) and wheat-oat (Kruse, 1969) hybrids and may be more difficult to exploit in improving the field resistance of wheats to G.g.t. Nevertheless, these levels of resistance will be easier to detect and study than the weaker levels of resistance already found in wheat.

On balance, this study provides some evidence of useful resistance to infection by G.g.t. in wheat. However, considerable work remains before it can be established that resistance will be of use to plant breeders.

CHAPTER 7

APPENDICES

APPENDIX 1: Number and size of stelar lesions and extent of cortical browning in the first seminal root of cultivars of wheat infected with isolate 19 of *G.g.t.* for 13 days

	cultivar	stelar lesions		cortical browning (as ranks)	
		number	mean size (as ranks)	discoloured	intensely discoloured
	cultivars of set 2				
	Aus1080	0.60	1.56	2.07	1.16
	Chile909	0.53	1.09	2.60	1.04
	Chile911	0.28	1.98	1.87	0.84
	Condor	2.58	1.39	0.42	0.06
	Kite	0.41	1.00	1.02	0.23
	Nebraska86	1.22	1.68	0.69	0.19
	Rac311	2.71	1.58	0.83	0.36
	cultivars of set 4				
	Eagle	1.6	1.2	0.5	0.1
(P)	Egret	1.9	2.0	0.4	0.2
	Festiguay	1.3	1.3	1.7	1.1
(P)	M2335	1.5	1.3	0.4	0.0
(P)	M2424	2.4	1.6	0.4	0.0
	Oxley	1.6	1.6	0.6	0.2
	Sun39A	2.6	1.4	0.6	0.4
	Sun41A	2.4	1.4	0.8	0.3
	Sun43A	1.2	1.3	1.8	0.3
	Cook	2.4	1.8	0.6	0.1
	K-2003-12	1.8	1.4	0.8	0.1
	LR/OXS2730-4	1.7	1.5	1.2	0.7
(P)	N10/TG2248-8	1.4	1.4	2.0	1.3
	Halberd	1.2	1.6	0.9	0.3
	(MKR*Kite)/57/S14	1.5	1.5	1.1	0.5
	(MM*MMC)/59/W6	1.9	1.7	0.3	0.2
	Rac357	1.5	1.5	1.3	0.7
	Rac399	2.3	1.3	0.8	0.4
	Rac415	1.6	1.7	0.8	0.4
(P)	Rac416	2.4	1.5	0.2	0.1

cont....

APPENDIX 1: continued

	cultivar	stelar lesions		cortical browning (as ranks)	
		number	mean size (as ranks)	discoloured	intensely discoloured
	cultivars of set 4, continued				
(P)	(WW-15*MH49)/36/W3	2.6	1.6	1.3	0.9
	Millewa	1.7	1.6	2.2	1.3
	MQ6	2.4	1.5	1.7	1.3
(P)	PD36	1.6	1.1	1.8	1.1
(P)	SD34	2.6	1.5	1.9	0.7
	Jacup	2.2	1.6	0.5	0.2
	69W/393	1.0	2.1	1.0	0.3
	69Z/401	2.0	1.8	0.7	0.3
	cultivars of set 5				
	Aroona	1.52	1.60	0.97	0.28
	Condor	2.58	1.39	0.42	0.06
	Cook	2.40	1.80	0.60	0.10
	Festiguay	0.99	1.22	1.45	0.88
	Jacup	2.20	1.60	0.50	0.20
	Kite	0.41	1.00	1.02	0.23
	Lance	2.08	1.36	1.26	0.76
	MC/29/S5	-	-	-	-
	Miling	2.19	1.74	0.52	0.16
	Millewa	1.39	1.48	1.77	0.93
	Oxley	1.39	1.50	0.52	0.12
	PF/41/W1	1.52	1.24	0.53	0.19
	Rac311	2.71	1.58	0.83	0.36
	Warigal	1.41	1.74	0.32	0.06
	Warimba	1.47	1.31	0.76	0.26

(P) cultivars of set 4 selected for study at Palmer in 1983

data represent means of ten replicates

APPENDIX 2: Monthly rainfall at field sites in years of study and long term averages of nearest meteorological stations

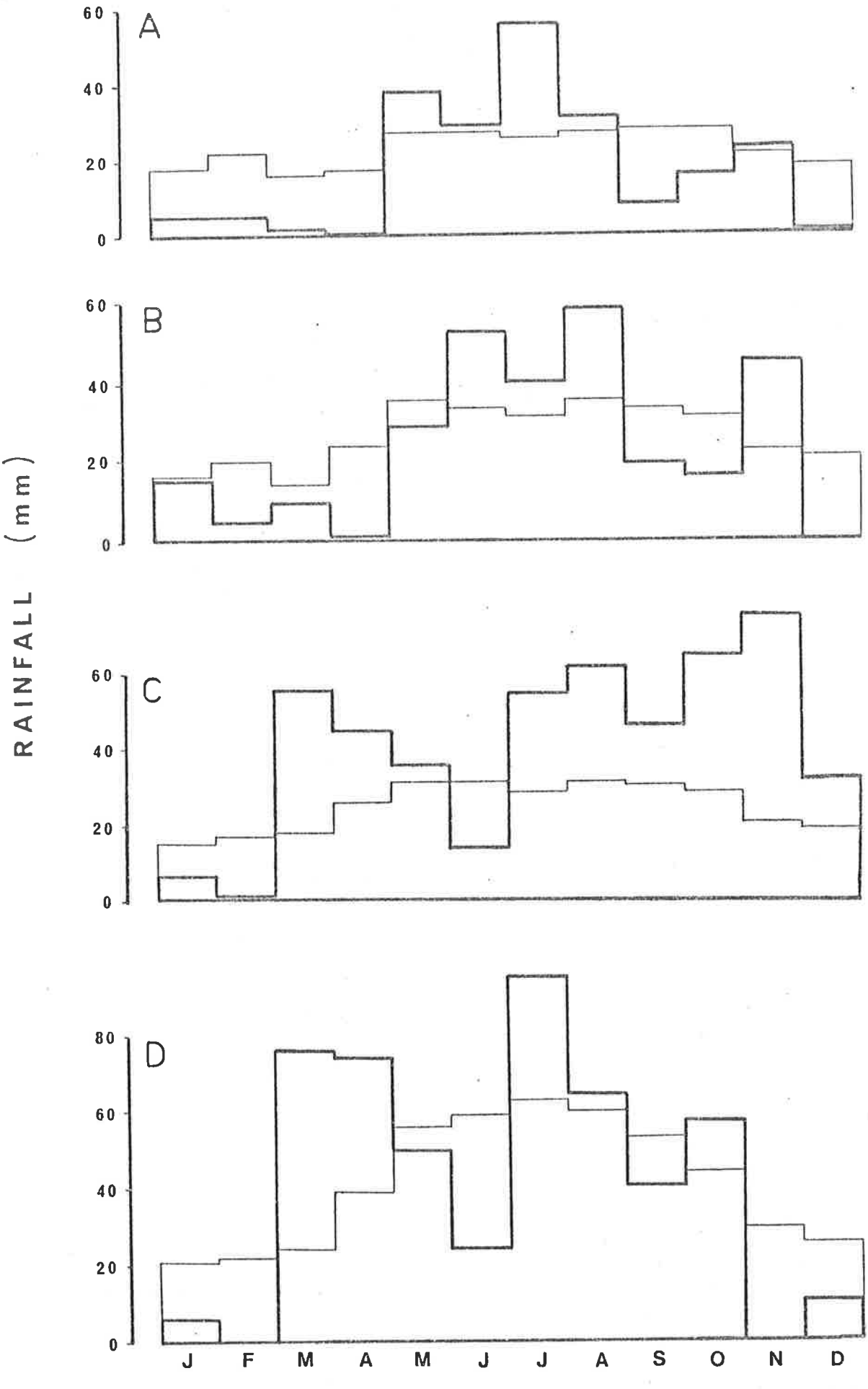
A. Nangari, 1981
Loxton, 1914-1983

B. Perponda, 1981
Karoonda, 1914-1983

C. Palmer, 1983
Mannum, 1876-1983

D. Strathalbyn, 1983
Strathalbyn, 1953-1983

long term averages are in faint line


A
B
C
D
60
40
20
0
80
RAINFALL (mm)
J
F
M
A
M
J
J
A
S
O
N
D

APPENDIX 3: Mean number of roots, length of the subcoronal internode, and percentage of roots infected with pathogens other than G.g.t. for cultivars grown in field experiments

Cultivars	at tillering §			at anthesis §			
	SEM	SCI (cms)	RSEM (angles)	COR	RSEM (angles	RCOR	OCOR)
SET 2 (Palmer)							
Aus1080	3.96	3.92	10.8	25.0	-	-	27.9
Chile909	4.48	2.36	8.1	37.6	-	-	15.9
Chile911	4.14	2.44	1.6	22.8	-	-	21.9
Condor	3.98	3.31	7.1	21.6	-	-	32.9
Kite	4.65	3.30	7.0	20.8	-	-	17.2
Nebraska86	4.30	3.26	4.3	23.2	-	-	28.7
Rac311	3.98	3.68	4.4	24.0	-	-	26.9
SET 2 (Strathalbyn)							
Aus1080	4.43	2.75	22.9	25.3	11.2	10.2	-
Chile909	5.03	0.80	18.3	40.1	12.1	11.5	-
Chile911	4.55	0.90	22.0	23.4	9.6	9.6	-
Condor	4.45	1.38	20.2	24.9	3.7	1.9	-
Kite	4.58	1.57	22.7	24.6	16.8	14.3	-
Nebraska86	4.35	2.28	21.1	22.8	4.4	14.4	-
Rac311	4.25	2.63	24.9	22.5	6.3	14.1	-
SET 3 (Palmer)							
Aus1080	4.45	1.60	5.9	25.4	-	-	24.3
Condor	4.43	1.65	7.0	24.9	-	-	10.8
mean of families	4.44	1.60	4.3	23.9	-	-	16.8
SET 4 (Palmer)							
Egret	4.70	2.03	10.8	24.9	-	-	20.2
M2335	4.06	2.56	10.0	29.3	-	-	13.9
M2424	4.10	1.50	16.8	26.3	-	-	19.1
N10/TG2248-8	3.60	2.22	14.9	19.7	-	-	10.0
PD36	4.13	2.33	18.1	26.3	-	-	20.3
Rac416	4.52	2.54	13.2	28.3	-	-	21.6
SD34	4.30	2.43	13.9	21.1	-	-	13.6
(WW15*MH49)/36/W3	4.03	1.60	5.7	22.6	-	-	18.4
SET 5 (Strathalbyn)							
Aroona	4.74	2.34	23.0	23.0	-	-	4.1
Condor	4.12	2.20	18.1	27.5	-	-	4.1
Egret	4.70	1.90	18.2	33.0	-	-	7.0
Festiguay	4.44	2.36	16.6	20.5	-	-	12.9
Kite	4.74	1.66	17.9	31.0	-	-	11.5
Lance	4.68	2.26	16.4	31.0	-	-	4.1
Milang	4.10	2.40	18.8	28.5	-	-	8.1
Millewa	4.32	0.00	18.8	20.5	-	-	10.0
Oxley	4.80	1.36	21.8	27.5	-	-	4.1
PF/41/W1	4.64	2,36	21.3	32.0	-	-	5.7
Rac311	4.04	3.00	20.6	28.5	-	-	10.0
Warigal	4.70	1.60	16.9	31.0	-	-	11.5
Warimba	4.38	1.56	19.7	26.5	-	-	0.0

§ abbreviations of means are explained on page 21

APPENDIX 4: Mean weight of dried shoots and number of tillers at anthesis for cultivars grown at Palmer and Strathalbyn, and percentage of ripened heads near maturation measured at Palmer, Turretfield or Waite

cultivar	shoot weight (gms)	number of tillers	ripened heads (%)
Set 2 (Palmer)			(a)
Aus1080	4.56	2.9	14
Chile909	3.68	3.8	0
Chile911	5.70	2.4	88
Condor	3.70	2.2	73
Kite	3.04	2.1	69
Nebraska86	6.30	2.6	86
Rac311	4.82	2.6	74
Set 2 (Strathalbyn)			
Aus1080	1.90	3.0	
Chile909	2.12	3.4	
Chile911	3.39	2.6	
Condor	2.26	2.5	
Kite	2.42	2.6	
Nebraska86	3.49	2.5	
Rac311	1.99	2.6	
Set 3 (Palmer)			(b)
Aus1080	3.35	3.7	25.5
Condor	3.11	2.6	95.0
mean of families	3.07	3.1	55.1
Set 4 (Palmer)			(c)
Eagle	-	-	4
Egret	4.13	3.75	15
Festiguay	-	-	38
M2335	4.50	3.38	18
M2424	4.52	3.60	33
Oxley	-	-	28

cont...

APPENDIX 4 continued

cultivar	shoot weight (gms)	number of tillers	ripened heads (%)
Set 4 (Palmer) continued			
Sun39A	-	-	2
Sun41A	-	-	30
Sun43A	-	-	18
Cook	-	-	25
K-2003-12	-	-	18
LR/OXS2730-4	-	-	48
N10/TG2248-8	4.97	3.84	71
Halberd	-	-	5
(MKR*Kite)/57/514	-	-	18
(MM*MMC)/59/W6	-	-	28
Rac357	-	-	2
Rac399	-1	-	25
Rac415	-	-	6
Rac416	4.23	4.44	3
(WW-15*MH49)/36/W3	3.94	2.93	40
Millewa	-	-	13
MQ6	-	-	30
PD36	4.84	4.20	2
SD34	4.53	3.40	8
Jacup	-	-	25
69W/393	-	-	28
69Z/401	-	-	85
Set 5 (Strathalbyn)			(c)
Aroona	1.8	2.6	99
Condor	2.5	2.8	95
Cook	-	-	-
Festiguay	1.2	2.1	99
Jacup	-	-	-
Kite	2.1	3.0	60

cont...

APPENDIX 4 continued

cultivar	shoot weight (gms)	number of tillers	ripened heads (%)
Set 5 (Strathalbyn) continued			
Lance	2.5	3.2	95
MC/29/S5	-	-	-
Miling	2.2	2.8	95
Millewa	1.5	2.1	95
Oxley	2.2	3.1	99
PF/41/W1	2.2	3.4	-
Rac311	2.8	3.0	85
Warigal	3.0	2.9	50
Warimba	2.7	2.5	80

Ripening measured in plots grown at:-

(a) Waite in 1983

(b) Palmer in 1983

(c) Turretfield in 1981

APPENDIX 5: Publication

Penrose, L. (1985). Evidence for resistance in wheat cultivars grown in sand culture to the take-all pathogen, *Gaeumannomyces graminis* var *tritici*.
Ann. appl. Biol. *107*, 105-108

Penrose, L. (1985). Evidence for resistance in wheat cultivars grown in sand culture to the take-all pathogen, Gaeumannomyces graminis var. tritici. *Annals of Applied Biology, 107*(1), 105-108.

CHAPTER 8

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