PATHOGENIC VARIABILITY IN *PUCCINIA SORGHI* ON MAIZE IN SOUTH AFRICA

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CHAPTER 1

COMMON RUST OF MAIZE: AN OVERVIEW

ABSTRACT

This literature review addresses the principal aspects related to maize common rust caused by *Puccinia sorghi*. The biology of the pathogen, host range, host-pathogen interactions and economic importance of the disease are addressed. Furthermore, different methods of disease management are reviewed, with emphasis on genetic control. In general, more references were obtained from studies done on sweet corn in the United States of America where maize rust is an important disease. Because of lack of information, only a few references addressing this disease throughout the Southern African region were included.

INTRODUCTION

Maize (*Zea mays* L.) is one of the most important food crops worldwide. Among the world's major cereal crops, maize ranks second after wheat in global production and first in terms of yield per hectare (FAO, 1992; Dowswell, Paliwal and Cantrell, 1996; Kling and Edmeades, 1997).

Maize is used principally for human staple food, feed for livestock and as raw material for many industrial products (Purseglove, 1975). Maize is the major human nutrient source in the tropics. In America, Africa, and Asia, several hundred million people depend on maize for their daily food. For many, it is the primary source of dietary protein (National Research Council, 1988) and calories, especially for the majority of the population in the SADC countries (Anonymous, 1998). Africa produces about 6% of the total world production, the majority of which is for human consumption (Kling and Edmeades, 1997). Maize grown in the temperate and developed countries is primarily used for animal feed (Purseglove, 1975; Kling and Edmeades, 1997). Dowswell *et al.* (1996) reported that animal feed accounts for 70% or more of total maize utilisation in industrialised economies, including Eastern Europe and the former Soviet Union as well as certain middle-income and newly industrialised nations of the Third World. In industrial economies, maize is the formula feed ingredient of choice because of low cost and high degree of consistency. As a raw material, maize serves as a basis for production of starch, oil and protein, alcoholic beverages, food sweeteners and, more recently, fuel (FAO, 1992; Dowswell *et al.*, 1996).

Sixty-four percent of the world's maize production area is in the developing countries (Kling and Edmeades, 1997). In these countries the area planted to maize has increased by 41% between 1961 and 1993, more than that for rice or wheat (Hess, 1997). Nearly 40% of the total

world maize crop is produced in the United States, where the average yield is 7.5 t/ha. Marked differences in yields between industrialised and developing countries occur, with average yields for industrialised countries being 6.2 t/ha, compared with 2.5 t/ha for developing countries. For example, average yield in West and Central Africa is about 1 t/ha and 1.5-2 t/ha in East Africa, Asia, and Latin America (Kling and Edmeades, 1997).

Low yields in developing countries are due to environmental, technological, and organisational factors (Dowswell *et al.*, 1996). Environmental factors include incidence and severity of diseases. Maize diseases can be divided into parasitic and non-parasitic disorders (Jugenheimer, 1958). Common rust, caused by the fungus *Puccinia sorghi* Schwein., is a parasitic disease widely distributed throughout maize-growing regions of the world, and may cause economic losses (Ullstrup, 1966). In South Africa, yield losses of economic significance occur in areas where the disease is severe. Although the disease has not been studied intensively in South Africa, losses of up to 40% have been reported (Kaiser and Nowell, 1983). Within southern Africa, *P. sorghi* has been reported in Zimbabwe (Rothwell, 1979) and some areas of Mozambique (Segeren, 1995; M. Denic, personal communication).

Gains in yield of up to 100% are possible with the use of fungicide control (Onofeghara and Kapooria, 1975; Groth *et al.*, 1983; Dillard and Becker, 1985). However, resistance is considered more effective and economically viable. Vanderplank (1968) proposed two types of disease resistance, viz. vertical (pathotype-specific) and horizontal (pathotype non-specific) resistance. Vertical resistance is governed by single dominant (Pataky, 1986; Hu and Hulbert, 1996) or recessive (Malm and Hooker, 1962; Kim and Brewbaker, 1987) genes that can easily be manipulated in breeding programmes. This type of resistance is characterised by a differential interaction between host genotypes and pathotypes. Horizontal resistance is multigenic, where additive genes are usually responsible for

resistance. This resistance is effective against all pathogen isolates but is not complete (Vanderplank, 1968).

The aim of this review is to discuss the biology, epidemiology and geographical distribution of the pathogen, host range, host-pathogen interactions, yield losses, pathogen variability and different methods of control, particularly genetic control.

1.2. CAUSAL ORGANISM

Maize common rust caused by the obligate parasitic fungus *Puccinia sorghi* was first described by Schweinitz from plant tissue that he thought to be sorghum. Hooker (1985) indicated that while the species name would suggest sorghum as a host, *P. sorghi* does not infect *Sorghum* species.

Puccinia sorghi belongs to the Basidiomycetes, class Hemibasidiomycetes, order Uredinales and family Pucciniaceae (Agrios, 1988). Basidiomycetes produce basidia and basidiospores in the reproductive cycle. The morphology of spermagonia, aecia, uredia, and telia are used for classification of genera and species in rust fungi (Anonymous, 1977). *Puccinia sorghi* urediospores are cinnamon-brown, spherical to ellipsoid, moderately echinulate, with three or four equatorial pores, and measure 23-29 x 26-32 μm (Hooker, 1985). Each spore is binucleate, as is mycelium which develops on germination (Anonymous, 1977). Teliospores are chest nut-brown to black, oblong to ellipsoid, two-celled with a constriction at the septum, measuring 16–23 x 29–54 μm (Hooker, 1985), and are attached to a pedicel which is twice the length of the spore. Cells of teliospores are binucleate, but prior to germination, the two haploid nuclei fuse to form the diploid phase of the fungus (Smith and White, 1988). Basidiospores are produced in basidia developing from germinating teliospores, and are small, thin-walled, hyaline, and haploid

(Anonymous, 1977). Pycniospores are formed within the pycnia (spermagonia) and are exuded in a gelatinous mass. Pycniospores are uninucleate, haploid spores, which fuse with paraphyses of the opposite mating type, while aeciospores are plane-yellow, verrucose, globoid to ellipsoid, and measure 13-19 x 18-26 μm (Smith and White, 1988).

1.3. HOST RANGE

Uredial and telial stages of *P. sorghi* occur wherever maize or its close relatives are grown. *Euchlaena* spp., such as annual teosinte (*Euchlaena mexicana*. Schrad.) appear to be highly susceptible to this disease (Mahindapala, 1978c). Perennial teosinte (*Euchlaena perennis)*, *Tripsacum* spp. (*Tripsacum dactyloide* (L.) L. and *T. lancedatum* Rupr. Ex-Forrn.) and Jobs-tears (*Coix lacrymajobi* L.) were previously reported to be susceptible, however, Malm and Beckett (1962) and Mahindapala (1978c) questioned their host status for maize rust.

The pycnial and aecial stages of the fungus occur on *Oxalis* spp., the alternate host for the pathogen. Earlier work reporting *Oxalis* spp. (in particular *O. corniculata)* as an alternate host for *P. sorghi* was from India (Lele *et al.*, 1962 cited by Nowell, 1981a). However, Zogg (1949 cited by Nowell, 1981a) was the first to show an *Oxalis* species (*O. stricta*) to be responsible for the annual occurrence of maize rust in Switzerland. Other studies reported pycnial and aecial stages on *O. corniculata* in Nepal (Nowell, 1981a). Aecial infections occur in many other areas, including temperate regions of Europe, USA, former USSR, highlands of Mexico, and South Africa (Anonymous, 1977; Hooker, 1985). Should teliospore germination coincide with the occurrence of the alternate host in these areas, it is expected that aeciospores not only contribute to early spring infections of maize, but also in creating pathogenic variability through sexual recombination.

1.4. LIFE CYCLE

Puccinia sorghi is a macrocyclic, heteroecious, and heterothallic fungus, with all five spore stages, basidial, pycnial, aecial, uredial and telial, occurring in its life cycle (Littlefield and Heath, 1979). The basidial, pycnial, and aecial stages occur on *Oxalis spp.*, and uredial and telial stages develop on maize (Mains, 1934; Smith and White, 1988) or related hosts, such as annual teosinte (*Euchlaena mexicana* Schrad) (Mahindapala, 1978c). Hooker and Yarwood (1966) were the first to sequentially culture all stages of this pathogen on detached leaves of corn (*Zea mays* L.) and *Oxalis corniculata* L.

The general life cycle of *P. sorghi* is initiated by germination of teliospores in spring. Teliospores produce basidiospores or sporidia on an elongated basidium, also known as a promycelium. Sporidia are unable to infect maize but infect certain *Oxalis* spp. (Ullstrup, 1966). At maturity, basidiospores are expelled from sterigmata, and germinate rapidly via one or two slender germ tubes (Pavgi, 1975, cited by Nowell, 1981a). Once basidiospores are deposited on a compatible Oxalis plant, they germinate, penetrate leaves, and, produce pycnia. Initially, pycnia are undifferentiated masses of hyphae, which form under the upper or lower epidermis (Nowell, 1981a).

The hyphae nearest to the epidermis break out to form a bush of paraphyses. The internal body of the pycnium then develops rapidly and at maturity consists of a hollow hemispherical wall from which pycniospores are projected into the cavity (Allen, 1934). Pycniospores are formed within the pycnia and exuded in a gelatinous mass. Pycniospores are uninucleate, haploid spores, which fuse with paraphyses or receptive hyphae of the opposite mating type protruding from ostioles of pycnia (Allen, 1934; Nowell, 1981a).

The aecial stage develops on the lower epidermis of *Oxalis* spp. leaves (Allen, 1934; Anonymous, 1977). If fusion between pycniospores and paraphyses of an opposite mating type does not occur, the mycelium remains haploid and further development is restricted (Allen, 1934; Nowell, 1981a). After karyogamy, binucleate aeciospores are produced in "cluster-cups" on the lower surface of the *Oxalis* spp. leaves (Smith and White, 1988). About six days after spermatization the first aecia open and wind-borne aeciospores, which infect maize leaves, are discharged (Allen, 1934). These infections give rise to uredia, which produce urediospores in repeating cycles providing favourable conditions prevail. Towards the end of the growing season, teliospores are produced. The fungus overwinters as teliospores in areas where the aecial stage is needed for survival from one season to the next. In the highlands of Mexico, pycnial, aecial, uredial and telial stages of the fungus can be seen frequently in the same field (Hooker, 1985). In June 2000, telia on crop residues, as well as pycnia and aecia on *O. corniculata,* were discovered in maize fields near Greytown, South Africa (Dunhin, personal communication). In areas where maize is grown throughout the year, urediospores survive on the primary host, thus bypassing the necessity for the spore stages on the alternate host. In such areas, the uredial stage becomes the repeating stage of the fungus (Flangas and Dickson, 1961 cited by Nowell, 1981a; Ullstrup, 1966; Anonymous, 1977; Hooker, 1978).

1.5. SYMPTOMATOLOGY AND DISEASE ASSESSMENT

Initial symptoms of common maize rust on a susceptible host are development of small, circular to elongate pustules that appear on aboveground plant parts, being most abundant on upper and lower leaf surfaces (Jugenheimer, 1958; Anonymous, 1977; De León, 1984). Disease

is most conspicuous at plant tasseling or maturity (De León, 1984; Fernandes, 1987), but seedling infections may occur, resulting in defoliation and stunting (Fernandes, 1992). In the early infection stages, pustules are golden to cinnamon-brown and associated with ruptured epidermal cell layers. Pustules become erumpent and powdery early in their development (Anonymous, 1977). The size and number of pustules depend on susceptibility of cultivars (Fernandes, 1987). Lesions become dark brown to black as plants mature and urediospores are replaced by teliospores (De León, 1984; Fernandes, 1987; Singh, 1987; Smith and White, 1988). Severe chlorosis and death of leaves and leaf sheaths may also occur.

Light orange-coloured pustules (De León, 1984) typify the aecial stage on the alternate host (*Oxalis spp.*). Aeciospores of *Puccinia andropogonis* var. *oxalidis,* also occurring on *Oxalis* spp., are nearly the same size as those of *P. sorghi*, and should not be confused with maize rust (Savile, 1984).

Common rust can be differentiated from the other two maize leaf rusts, southern rust (caused by *P. polysora* Underw.) and tropical rust (caused by *Physopella pallescens* (Arth.) Cummins & Ramachar). *Puccinia sorghi* is contrasted from *P. polysora* by larger, sparsely distributed, elongate uredia; darker urediospores; erumpent telia; teliospores with thicker walls, longer pedicels, and a bullet-shaped terminal cell. Uredia of *P. sorghi* are common on both leaf surfaces while those of *P. polysora* are more numerous on the upper leaf surface. Teliospores of *P. polysora* remain covered. *Physopella pallescens* is identified by hyaline urediospores; covered, black telia, and sessile teliospores in short chains (Anonymous, 1977; De León, 1984).

Macroscopic symptoms of common rust of maize in cultivars with pathotype-specific resistance are expressed as hypersensitive reactions with distinct qualitative infection types. Infection type ratings are usually

used in seedling tests according to a 0 to 4 scale (Table 1.1), where 0 to 2 represent low infection types (resistant host response), and 3 to 4 high infection types (moderately susceptible and susceptible host responses, respectively) (Hooker, 1985). Two mesothetic or "X" infection types have been reported. The Y infection type was added for wheat leaf rust by Johnston (1963, cited by Roelfs, 1984) and the Z infection type for common rust by Van Dyke and Hooker (1969) (Roelfs, 1984). On maize, mesothetic infection types appear as a mix of fleck-type response and small pustules (Van Dyke and Hooker, 1969). Hu *et al.* (1996) referred to such reactions as the formation of necrotic spots in addition to pustules that are not associated with necrosis. These were described for wheat stem rust as a reaction where different infection types are interspersed on a single leaf (Heyne and Johnston, 1954). The Z-infection type is where lesions at leaf tips are typically hypersensitive (resistant flecking), while those at leaf bases appear as normal pustules (Wilkinson and Hooker, 1968; Van Dyke and Hooker, 1969).

Experimental evidence suggests that age and maturation of leaf tissue determine Z-type reactions. Older, mature tissues are resistant and younger tissues, or tissue with delayed maturation, are susceptible (Van Dyke and Hooker, 1969). Z-reactions appear to be influenced more by light than temperature. Van Dyke and Hooker (1969) reported that temperatures did not change the Z-reaction, while lack of light before and after inoculation increased susceptibility.

Resistant host responses to common rust in cultivars with partial or pathotype non-specific resistance are expressed as a reduced receptivity. This is achieved by reduction in number and size of pustules, less sporulation per pustule, or an increased length of the latent period (Pataky, 1986). Slow rusting has been mentioned as an expression of resistance, but has not been clearly distinguished from mature plant resistance (Hooker, 1985).

Table 1.1. Description of seedling infection types produced by *Puccinia sorghi* in pathogenicity studies on maize (Hooker, 1985)

Type	Symptom description
0;	Small chlorotic flecks
1-	Small necrotic spots
1	Small pustules surrounded by necrotic tissue
2	Small pustules surrounded by chlorotic areas
3	Medium sized sporulating pustules without chlorosis
4	Large sporulating pustules
X	Mixture of resistant and susceptible-type pustules interspersed over the leaf
Z	Resistant-type pustules on the older leaf tissue inoculated and susceptible- type pustules on the younger leaf tissue such as that in the leaf whorl at the time of inoculation

A susceptible reaction for maize rust is typified by the formation of normal pustules, which may vary in size according to the genotype attacked by disease (Hooker and Le Roux, 1957; Kim and Brewbaker, 1976a; 1977). Distinct infection types are not recognised for horizontal resistance (McGee, 1988). Ratings on plant reaction expressed as pustule number are usually taken in the field. The conventional way of measuring it is to estimate the percentage of leaf area infected, using the modified Cobb scale. According to this scale, 37% of the actual leaf surface covered by pustules represent a value of 100% (Peterson, Campbell and Hannah, 1948).

The Horsfall-Barratt (1945) rating scale is widely used when precision in assessing disease is warranted (Dillard and Seem, 1990a). It has been useful in fungicide research and studies of varietal resistance. This scale was based on the Weber-Fechner Law, which holds that the human eye distinguishes according to the logarithm of the stimulus. Below 50% severity, the eye sees the amount of diseased tissue whereas above 50%, it sees the amount of disease-free tissue. The Horsfall-Barratt scoring system is therefore based on 50% as midpoint.

The popular 0 to 5 or 0 to 9 numerical rating scales are usually considered adequate for measuring maize rust severity in the field. However, Campbell and Madden (1990) stated that class values of numerical scales could not be averaged, as biased results would occur. Thus, to statistically compare maize entries in a replicated field trial, numerical values have to be converted to a percentage scale, for example as described by Davis, Randle and Groth (1988) (Table 1.2).

Table 1.2. Conversion of a 0 to 9 numerical scale to percentage leaf area diseased (Davis *et al.*, 1988)

Numerical Scale	Description	% Scale
0	Immune response, no visible disease	0
1	Immune response, hypersensitive flecks present	0.68
2	One to 10 single disease areas (pustules and group of pustules) present	2.10
	10- 20 single disease areas present plus evidence of banding pattern	5.70
4	Numerous single disease areas present plus a well defined banding pattern	11.50
5	Up to two well-defined banding patterns with numerous single disease areas present	20.00
6	Leaf margins becoming necrotic with numerous single disease areas present plus up to two banding patterns	36.00
7	Numerous single disease areas, banding patterns and necrotic margins well defined	55.00
8	All leaf tissue necrotic except for centre of leaf	80.00
9	All leaf tissue necrotic except midrib and some adjoining leaf tissue	96.00

1.6. GEOGRAPHICAL DISTRIBUTION

Common rust has been reported in most maize producing countries worldwide. This disease is prevalent in subtropical production areas (17 to 25^{0}C), and in tropical mid-altitudes and highlands where maize is often grown all year round (Nowell, 1981a; Dowswell *et al.*, 1996). Maize common rust is endemic in the western hemisphere. Common rust has been reported as a serious sweet corn disease in the United States of America, including Hawaii, Wisconsin, Minnesota, Illinois, New York, California and southern and central Florida (Nowell, 1981a; Pataky, 1987a; 1987b; Groth, Pataky and Gingera, 1992; Pataky and Eastburn, 1993a; 1993b; Davis *et al.*, 1995; Hu and Hulbert, 1996; Hu, Webb and Hulbert, 1997). Rust has also been reported to be a major disease in central-west, south-east and southern Brazil (Fernandes and Balmer, 1990).

In South Africa, common maize rust occurs throughout the maize production areas. Severity is influenced by climatic conditions, with severe infections occurring in the humid eastern parts of the country, where yield losses may be of economic significance (Kaiser and Nowell, 1983). *Puccinia sorghi* has also been reported in Zimbabwe (Rothwell, 1979) and in the Marracune, Angonia and Montepuez districts of Mozambique (Segeren, 1995). Denic (personal communication) suggested that common rust can occur in other areas of Mozambique but that yield losses caused by this pathogen are unknown. No information was available from other SADC countries.

1.7. ECONOMIC IMPORTANCE

Leaf diseases reduce yield quality and quantity, and predispose plants to attack by stem rot pathogens (Agrios, 1988; Fernandes and Balmer, 1990). *Puccinia sorghi* has historically been considered a disease of minor

importance in the United States, but caused leaf damage in other countries (Jugenheimer, 1958). Severe rust epidemics have occurred in the upper midwest of the USA (Pataky and Headrick, 1989; Pataky and Eastburn, 1993a; Pataky, 1995), and epidemic outbreaks of the disease occur frequently when environmental conditions during a growing season are favourable (Wegulo *et al.*, 1998). This increase in occurrence may have been due to expanded maize cropping in the southern USA during winter (Pataky and Eastburn, 1993a; Futrell, 1975 cited by Gingera, Davis and Groth, 1994), planting of high quality, susceptible sweet corn hybrids and prevalence of cool, wet weather in the midwest (Pataky, 1987b; Pataky, 1995). Epidemics of common leaf rust are more severe on sweet corn than field corn as the latter possesses greater levels of resistance (Hooker, 1969). Field corn is also planted earlier in the season, and may escape infection to some extent. As many popular sweet corn hybrids are highly susceptible to rust, late planted fields may develop serious epidemics (Pataky, 1987b, Pataky and Eastburn, 1993a).

Yield reductions caused by common rust are variable. Early reports stated that losses caused by common rust range from very little damage in some years to 25% or more in others (Jugenheimer, 1958). Pataky (1987a) estimated that a 6% yield loss was associated with each 10% increase in rust severity a week before harvest on sweet corn hybrids in Illinois. Kim and Brewbaker (1976b) reported yield reductions of 12 to 40% during summer and 6 to 75% for winter grown maize in tropical Hawaii. Pataky and Eastburn (1993b) reported yield reductions between 15 and 45% on susceptible hybrids. Grain and maize fodder yield reductions of up to nearly 50% have been observed on susceptible hybrids (Groth *et al.*, 1983). Kaiser and Nowell (1983) reported that yield reductions caused by *P. sorghi* in South Africa were of similar magnitude than those found by Kim and Brewbaker (1976b). Results of a preliminary study at Greytown revealed yield decreases of up to 15%, and suggested that rust could be of

economic significance in the moist production areas of South Africa (Kaiser and Nowell, 1983).

Rust infections in maize have also been reported to reduce plant height, fresh plant weight, ear length, ear diameter, oil content, and protein content, and increase stalk rot (Hooker, 1985).

1.8. ENVIRONMENTAL REQUIREMENTS

Sweet corn rust epidemics are generally attributed to optimum climatic conditions where temperatures are cool (16-23^{0}C) and relative humidity is high (100%) (Anonymous, 1977; Pataky and Headrick, 1989). Temperature appears to be extremely important for germination, penetration, establishment, and proliferation of the pathogen (Weber, 1922; Smith, 1926; Pavgi and Dickson, 1961; Kushalappa and Hegde, 1971, Mahindapala, 1978a; 1978b). Weber (1922) considered minimum, optimum and maximum temperatures for germination of urediospores, to be 4^{0}C, 17 to 25^{0}C, and 32^{0}C, respectively. The optimum temperature for infection was 18^{0}C. Pavgi and Dickson (1961) obtained similar results. Kushalappa and Hegde (1971) reported temperatures for urediospore germination on water agar to be 5^{0}C, 18-20^{0}C, and 35^{0}C.

Mahindapala (1978a) reported a temperature range of 5-25^{0}C for germination of urediospores on leaves and agar. The optimum for infection and appressorium and substomatal vesicle formation was 15^{0}C. A minimum period of 3-4 h was required for infection initiation. Mahindapala (1978b) found that the number of pustules on incubated maize seedlings at 100% relative humidity (RH) was directly related to temperature and duration of incubation period. The generation time was 16 days at 10^{0}C, 10 days at 15^{0}C, 7 days at 20^{0}C and 5 days at 25^{0}C.

Rust responses are influenced more by temperature than light, however, Syamananda and Dickson (1957) found that plants grown under supplemented light showed increased necrosis around uredia compared with plants grown under natural light. Headrick and Pataky (1986) found that 12 h light/12 h dark periods immediately after inoculation were more effective for disease development when a mist period of 48 h was employed. They found that a 6 h moisture period was the minimum for infection, with longer moisture periods increasing levels of infection. Night temperatures have also been shown important for uredium formation. Disease development was slow when night temperatures fell below 8^0C and little sporulation occurred at 32^0C (Headrick and Pataky, 1986).

Humidity is a major factor necessary for disease development. The highest and lowest germination of urediospores was reported at 100% and about 78% RH, respectively (Smith, 1926; Hooker, 1985). Mahindapala (1978a) found germination and germtube growth at RHs of 98.5%-100%.

Germination of urediospores decreased with approaching winter (Weber, 1922; Smith, 1926). Mederick and Sackston (1972), cited by Nowell (1981a) reported that urediospores survived only a few weeks on dry maize after which viability of spores declined markedly. A survey conducted in England showed that urediospores, at the end of summer, survived for up to 11 weeks (Mahindapala, 1978c). Furthermore, viability of urediospores depended upon minimum temperatures and relative humidity over the storage period (Mahindapala, 1978a). Temperatures below zero and higher humidity enhanced survival of urediospores (Von Meyer, 1963 cited by Nowell, 1981a).

Succulent growth of a susceptible host favours development of biotrophic fungi (Jugenheimer, 1958). Unlike other pathogens, which tend to attack weak or poorly growing plants, rust fungi parasitize tissue of vigorously growing plants. In general, this explains why rust fungi tend to increase under intensive, e.g. high levels of nitrogenous fertilisers and

adequate moisture, and extensive cultivation of many economically important crops (Anonymous, 1977; Agrios, 1988).

1.9. PATHOGENIC VARIABILITY

For plant pathologists and breeders, pathotype identification is most useful in organisms that have limited variability and stable pathotypes (Roelfs, 1984). In cases where many pathotypes are prevalent, or rapid changes in pathotype profiles occur, the less useful the system of studying variability become (Groth *et al.*, 1992). However, information on occurrence and distribution of pathotypes, as well as the availability of representative isolates, are useful in germplasm screening and breeding for resistance. The gene-for-gene system provides the genetic explanation for interactions between avirulence and resistance genes and plays a major role in pathotype identification for many cereal rusts.

Like most cereal rusts *P. sorghi* isolates differ in virulence to plants having major (*Rp*) alleles for resistance (Russell and Hooker, 1959; Hooker, 1963; Lee *et al.*, 1963; Kim and Brewbaker, 1976a; Hulbert, Lyons and Bennetzen, 1991). Russell and Hooker (1959, 1962) recognised that phenotypic expression (pustule type) of host-pathogen interaction in maize rust was the result of expression of host and pathogen genes in a given environment. Thus, pustule type was used to characterise virulence or avirulence in the pathogen and resistance or susceptibility in the host. The earlier work involving pathotype variability in *P. sorghi* was limited to the identification of host resistance. Mains (1931) reported genes, designated as *Rp* genes, in Golden Grow lines conditioning resistance to two pathotypes. In further studies various factors for resistance to common rust were identified and differentiated using a diverse collection of *P. sorghi* isolates (Hooker and Le Roux, 1957; Hagan and Hooker, 1965; Wilkinson

and Hooker, 1968; Groth *et al.*, 1983). Hooker and Le Roux (1957) tested over 300 maize lines from the United States, Mexico, Argentina, Australia, Guatemala, Ethiopia, Canada, Turkey, South Africa, and Peru. Wilkinson and Hooker (1968) reported different reactions in 15 inbred lines from Africa and Europe inoculated with seven North American cultures of *P. sorghi*.

The development of near-isogenic rust-resistant lines was initiated by incorporating resistance genes to common rust from several sources into the rust susceptible inbred lines B14 and R168 by backcrossing (Hooker and Russell, 1962). Hooker and Russell (1962) suggested that these lines were useful for genetic studies of the pathogen, studies of gene action in the host, and pathogenicity studies. Table 1.3 lists the current differential lines and their respective sources of *Rp* alleles, according to Pataky (1987b) and Hulbert *et al.* (1991). Development of these *Rp* differential lines resulted in more efficient studies of pathogenic variability in common maize rust. More recent reports of variation in *P. sorghi* were published by Bergquist and Pryor (1984), Hulbert *et al.* (1991), Hu and Hulbert (1996), Pataky and Tracy (1999 cited by Pataky, 2000), Pataky (2000) and Pate and Pataky (2000).

Hulbert *et al.* (1991) recognised considerable diversity in *P. sorghi* pathotypes in certain years, even within small geographical areas that did not have a previous record of extensive variability. None of the *Rp* genes have been used extensively over large areas to control common rust in North America (Hu and Hulbert, 1996). The occurrence of novel pathotypes virulent to certain *Rp* alleles previously reported as resistant suggested that changes occurred in the North American population of *P. sorghi* (Pataky, 1987b; Hulbert *et al.*, 1991; Groth *et al.*, 1992; Hu and Hulbert, 1996). In general, influx of exogenous inoculum and sexual recombination were suggested to be responsible for diversity in the North American population

Table 1.3. Maize lines with designated *Rp* alleles and the original source of resistance (Pataky, 1987b; Hulbert *et al.*, 1991)

Rp Genes	Source	[a] Line name
Rp1^{A}	GG208, G. King	R168 and Lines from Golden King
Rp 1^{B}	B38	B14, B216, B217
Rp 1^{C}	K148, Syn. A, B. Y. Dent	R168, B14
Rp 1^{D}	CuZCO, Kitale, Njoro	R168, B14
Rp1^{E}	B49	R168
Rp1^{F}	PI 172332	R168
Rp1^{G}	PI 163558	R168
Rp1^{H}	Guanajuato 29-157A	R168
Rp1^{I}	PI 163558	R168
Rp1^{J}	Queretaro VI 366	R168
Rp1^{K}	Queretaro VI 231-5	R168
Rp1^{L}	PI 163558	R168
Rp1^{M}	PI 163563	R168
Rp1^{N}	BZU-20	R168
Rp3^{A}	25	R168
Rp3^{B}	M16	R168
Rp3^{C}	Nn14	R168
Rp3^{D}	Leoni 27-4-1	R168
Rp3^{E}	Hidalgo 3-5-1	R168
Rp3^{F}	PI 251653	R168
Rp4^{A}	Queretaro V260-1	R168
Rp4^{B}	PI 193906	R168
Rp5	PI 186191	R168
Rp6	PI 172597	Cultivar (PI 172597)

[a]Isogenic series of differential lines created by Hooker and co-workers, available as R168 and R14 inbred backgrounds. Hulbert *et al.* (1991) used additional sources for *Rp1*^{A} (lines derived from the Cultivar Golden King), *Rp1*^{B} (inbred lines B216 and B217), and *Rp6* (cultivar PI 172597).

of *P. sorghi* (Hulbert *et al.*, 1991). Pataky (2000) reported a pattern of virulence in Mexican *P. sorghi* populations similar to those collected from the midwestern United States in 1999. This was considered the first widespread occurrence of *P. sorghi* virulent on maize with the $Rp1^D$ gene in North America and confirmed the influx of *P. sorghi* inoculum from Mexico to the United States.

Procedures used to differentiate rust pathotypes are similar to those employed for other cereal rust fungi. Collection of urediospores from rust-infected field samples can be done either by lightly tapping the leaves over clean paper or by using a cyclone spore collector. Spores collected from the field are inoculated onto susceptible maize cultivars, which are then incubated in a dew chamber before placement in an air-conditioned greenhouse. For inoculations, urediospores are suspended in light mineral oil and sprayed onto plants as described by Browder (1971). Single-pustule isolates are subsequently established, increased, and inoculated onto differential sets as described above. After approximately seven to 10 days infection types on each differential line are rated according to a scale (e.g. the 0 to 4 scale described in Table 1.1), and classified as either resistant or susceptible. Isolates can be stored by first drying the urediospores in a desiccator for three to four days, before placement in a refrigerator (5^0C) or regular freezer (-20^0C) for short-term storage, or at -70^0C for longer periods (Hulbert *et al.*, 1991).

Based on the resistance /susceptibility profile of the differential set a particular pathotype is identified. Thus, a pathotype is an isolate or a group of similar isolates that can be distinguished from other isolates by differences in their avirulence/virulence phenotype, as reflected by the infection type, and are designated by numbers, letters, or a combination of both. Codes become well known and useful when they are used for several years. It is also important that the coding system is flexible to incorporate new differential lines should they be included in the set (Roelfs, 1984).

1.10. DISEASE CONTROL

In general, fungicide applications, host resistance (Anonymous, 1977; Nowell, 1981b; Nowell and Rijkenberg, 1983; Pataky and Headrick, 1988; Pataky and Eastburn, 1993a), and disease avoidance by planting date adjustments (Pataky, 1987b; Groth, *et al.*, 1992), are measures that can contribute to the control of common maize rust.

1.10.1 Chemical control

Smith and White (1988) suggested that fungicide control of common maize rust is justified for dent maize seed production fields and sweet corn hybrids when the disease is severe. However, no guidelines exist for the number and timing of fungicide applications to control rust economically (Pataky and Headrick, 1988).

In South Africa, limited studies on chemical control of *P. sorghi* have been carried out. Nowell (1981b) investigated the efficacy of systemic fungicides and the protectant fungicide mancozeb. Mancozeb controlled maize rust more effectively than bitertanol (L), bitertanol (H), propiconazole, and oxycarboxin. There were no significant differences in efficacy amongst the systemic fungicides, although they were all significantly different from the unsprayed control. Unfortunately, results from this study were influenced by unfavourable climatic conditions, which delayed disease progress. Nowell and Rijkenberg (1983) conducted trials to examine effects of four fungicides on *P. sorghi*. Similar to previous work, mancozeb controlled *P. sorghi* more effectively and economically than the other fungicides. Bitertanol fungicides showed better curative action than mancozeb but appeared to be phytotoxic at high concentrations. Nowell and Laing (1998) tested at least 20 fungicides over three seasons for their efficiency to control *Exserohilum turcicum* (Pass.) K.J. Leonard and E.G. Suggs and *P. sorghi* on maize, at Greytown in KwaZulu-Natal. From their

study, difenoconazole was consistently the most effective in controlling *P. sorghi*, followed by mancozeb. Timing and frequency of fungicide application are important to achieve maximum disease control and yield benefits in maize (Pataky, 1987a; Wegulo *et al.*, 1998). Fungicide treatments are effective in initial applications to protect plants at early growth stages, thus preventing cycles of autoinfection (Pataky and Headrick, 1988). Pataky (1987a) and Wegulo *et al.* (1998) reported that three to five sprays of chlorothalonil, mancozeb, or propiconazole on maize resulted in lower disease severity and higher yields than zero, one, or two applications of the same fungicides. Fungicide applications are also effective if environmental conditions are not favourable for rapid disease development, or rust inoculum levels are low (Pataky and Headrick, 1988). Pataky and Headrick (1988) and Dillard and Seem (1990b) determined incidence and severity relationships for common maize rust on sweet corn. When incidence was less than 80%, rust severity on leaves was 1-2%. Severity significantly increased when incidence exceeded 80%, leading them to propose this as a threshold level for the initiation of fungicide applications, prior to tasseling. This threshold was applicable to moderately susceptible to susceptible hybrids grown in rust-conducive environments.

Reuveni, Agapov and Reuveni (1994) and Reuveni, Reuveni and Agapov (1996) addressed the possibility of dual use of phosphate and NPK salts as foliar spray fertilisers and as inducing agents for disease protection. The protection induced by phosphate in maize was expressed by reduction in the number of pustules on the upper leaves of protected plants, and restriction of lesion expansion. Non-specific mechanisms were involved in this systemic protection, which was suspected to be either caused by chemical or enzymatic effects (Reuveni, *et al.*, 1994).

Although the use of fungicides in relation to host resistance is significant, and yield losses can be minimised between 4-100% (Onofeghara and Kapooria, 1975), breeding programmes showed that

maize rust can be easily and effectively controlled genetically (Nowell, 1981a; Pataky and Eastburn, 1993b). Chemical control of disease is, furthermore, expensive and raises environmental concerns (Pataky and Headrick, 1989). In general, protectant chemicals like mancozeb and related compounds were reported to be effective to control common rust, but because of economic reasons are not widely used for disease control (Hooker, 1985). At present, no fungicides are registered for maize rust control in South Africa (Nel *et al.*, 1999). However, many DMI compounds, specifically the triazoles, are known for their efficacy against several *Puccinia* spp. (Kuck *et al.*, 1995).

1.10. 2. Genetic resistance

Plants are resistant to pathogens either because of specific pathogenicity restricting the latter to a certain host or host range, or plants possess genes for resistance directed against genes of avirulence of the pathogen, or because plants escape or tolerate infection by such pathogens (Agrios, 1988).

Two types of resistance to common maize rust have been described. Vertical resistance is when a variety is resistant to some pathotypes and not to others. Horizontal resistance is when resistance is effective against all pathotypes (Vanderplank, 1968). Vertical resistance implies a differential interaction between host varieties and pathotypes, while horizontal resistance shows no differential interaction (Vanderplank, 1968).

Terminology for disease resistance is variable. The terms vertical and horizontal resistance are popular among breeders and pathologists, and will be used to indicate pathotype (race) -specific and pathotype non-specific resistance types. Parlevliet (1985), however, considered these terms obsolete and he defined race-specific as resistance, which is characterised by differential genetic interactions between host and

pathogen genotypes. Table 1.4 lists some of the terms used to describe resistance types in plants.

Studies of vertical resistance in maize to common rust were started by inoculating four inbreds, with high levels of resistance, with three pathotypes of *P. sorghi* (Mains, 1931). Two of the inbreds tested were resistant to pathotypes 1 and 3, and the other two inbreds were only resistant to pathotype 1 (Mains, 1931). Resistance appeared to be based on a single Mendelian factor. Vertical resistance has also been referred to as qualitative or differential resistance. As it is controlled by one or few genes, this kind of resistance is regarded as monogenic or oligogenic. In some cases, it is known as major gene resistance (Hagan and Hooker, 1965; Agrios, 1988). Genes for vertical resistance can be used to differentiate between pathotypes.

In maize vertical resistance is qualitatively expressed in seedling and adult plant stages (Pataky, 1986; Hu and Hulbert, 1996), and the level therefore increases with host maturity. Resistant reactions are expressed as chlorotic or necrotic hypersensitive flecks with little or no sporulation (Hulbert *et al.*, 1991). Genes controlling vertical resistance act in a gene-for-gene manner with rust isolates, conferring high levels of resistance to specific rust pathotypes (Hu and Hulbert, 1996). Inheritance of vertical rust resistance is commonly monogenic and dominant (Bergquist and Pryor, 1984).

Based on early genetic studies on vertical rust resistance a single *Rp* gene was designated (Mains, 1931). Rhoades (1935), and Rhoades and Rhoades (1939) described the *Rp* gene on the terminal position of the short arm of chromosome 10. It was later discovered that resistance genes in chromosome 10 were members of an allelic series (Russell and Hooker, 1959; Hooker and Russell, 1962).

Table 1.4. Characteristics of vertical and horizontal resistance to rust diseases

Type of resistance	Synonyms	Characteristics
Vertical resistance	Specific resistance Race-specific resistance Qualitative resistance Differential resistance Monogenic or oligogenic resistance Major gene resistance	Expressed qualitatively by seedling and older plants; Resistance reactions: chlorotic or necrotic hypersensitive flecks with little or no sporulation; Controlled by single genes, either recessive or dominant; Genes act in gene-for-gene manner with the rust fungus; Confers high level of resistance to specific rust pathotypes; Varieties carrying this type of resistance show resistance to a specific pathotype under most environments; The resistance can be easily overcome by new rust pathotypes; The most frequently studied resistance.
Horizontal resistance	Partial resistance Generalised resistance Quantitative resistance Adult-plant resistance Field resistance Durable resistance Non-specific resistance Polygenic or multigenic resistance	Expressed quantitatively at adult plant stage in the field; Resistance reactions are expressed phenotypically as low number of pustules on leaves, smaller uredia and reduced chlorosis surrounding uredia; Controlled by multiple genes; Effective to all pathotypes; Resistance is stable over environments; The least studied resistance.

At least six or more loci containing over 25 single, dominant resistant genes for common maize rust have been identified (Hooker and Russell, 1962; Hagan and Hooker, 1965; Saxena and Hooker, 1967; Wilkinson and Hooker, 1968; Hu and Hulbert, 1996). Fourteen genes (*Rp1*^{A} *to Rp1*^{N}) were designated *Rp1*, and genetic mapping experiments placed them on chromosome 10 (Hooker, 1978). In the same chromosome two additional genes, *Rp5*, and *Rp6* were mapped (Wilkinson and Hooker, 1968). Multiple resistance genes were also mapped to the *Rp3* and *Rp4* loci on chromosomes 3 and 4, respectively (Saxena and Hooker, 1974). Reports on the effectiveness of these *Rp* alleles in Hawaii and the USA have been provided by Bergquist and Pryor (1984), Pataky (1986), Hulbert *et al.* (1991), Groth *et al.* (1992), Gingera *et al.* (1994), Pataky and Tracy cited by Pataky (2000) and Pate and Pataky (2000).

Horizontal resistance has been referred to as partial resistance (Groth *et al.*, 1983; Pataky, 1986), general resistance (Hooker, 1969), polygenic or multigene resistance (Kim and Brewbaker, 1977; Hooker, 1985; Pataky, 1986), and adult or mature plant resistance (Hooker, 1967). In accordance with previous reports, Newburg (1992) concluded that horizontal resistance is not complete, but usually moderate, allowing limited pathogen invasion and spore production. It has also been referred to as non-race specific resistance, non-differential resistance, quantitative resistance, field resistance, or durable resistance (Agrios, 1988). Horizontal resistance is polygenic in nature, and is controlled by several minor genes. Each gene alone may be ineffective against the pathogen and play a minor role in the total resistance (Agrios, 1988).

Due to the number of genes involved, the genetic basis of horizontal resistance to *P. sorghi* is complex (Hooker, 1969; Kim and Brewbaker, 1977; Randle, Davis and Groth, 1984; Hooker, 1985), and is expressed primarily at the adult plant stage (Pataky, 1986; Hu and Hulbert, 1996). Inheritance of horizontal resistance to rust, studied in field and sweet corn,

appears to be heritable despite being poligenically inherited (Jugenheimer, 1958; Hooker, 1967, 1969; Kim and Brewbaker, 1977; Davis *et al.*, 1995). Hooker (1969) reported that heritability estimates averaged 84% for 64 crosses. Kim and Brewbaker (1977) estimated general and specific combining ability of heritability at 86 and 73%, respectively. Pataky (1986) reported that dominant and additive gene effects also play a role in inheritance of horizontal resistance. Resistance has been characterised by quantifying different components of the infection cycle. Although these studies would often refer to such partial resistance as horizontal, the influence of different pathotypes on resistance expression, as implied by definition, was not investigated. Partial resistance is expressed as fewer and smaller uredia, and reduced chlorosis surrounding uredia (Hooker, 1969, Kim and Brewbaker, 1977; Randle *et al.*, 1984; Hooker, 1985; Pataky, 1986). Horizontal resistance is extremely important in breeding for rust resistance. It should protect the crop from economic losses as well as decrease the selective advantage for new virulence genes in the pathogen (Gingera *et al.*, 1994).

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CHAPTER 2

PATHOGENIC VARIATION OF *PUCCINIA SORGHI* IN SOUTH AFRICA

ABSTRACT

To determine pathogenic variability in *Puccinia sorghi* in South Africa, rust infected maize leaves were collected during the 1999/2000 season. Isolates collected in the field were increased on susceptible plants and inoculated onto maize differential lines carrying different *Rp genes* for resistance to *P. sorghi*. Seven pathotypes, namely, A, B, C, D, E, F and G, were differentiated in the greenhouse. Pathotype B was the most virulent and occurred in Gauteng, KwaZulu-Natal, and Mpumalanga. Pathotype A, virulent only on $Rp3^A$ and $Rp3^B$, was widely distributed, occurring in six provinces. Pathotype E was collected from North West, F and G from KwaZulu-Natal, C from Mpumalanga and the Northern Province, and D from Mpumalanga. No virulence was detected for genes $Rp1^C$ (line V20495), $Rp1^G$, $Rp1^L$, $Rp3^D$, and $Rp3^F$. All isolates were virulent on $Rp3^B$ (line V20523). The occurrence of virulence for most *Rp* genes suggests that monogenic resistance is of little value for future protection of maize cultivars against common rust in South Africa.

INTRODUCTION

Common maize rust caused by *Puccinia sorghi* Schwein. has recently increased in incidence and severity in South Africa. Comprehensive maize leaf disease surveys carried out during 1994 – 1996 showed that common rust was endemic and widespread throughout the South African maize production area (Flett and Bensch, unpublished). Despite recognition of the importance of maize rust by Nowell (1981), and Nowell and Laing (1998), very few research efforts have been made to control this disease locally. Maize breeders thus need to take cognisance, introduce sources of resistance into their programmes, and select for rust resistance that has the potential to remain durable. However, the variability of the pathogen and its concomitant population biology is largely unknown in southern Africa, making directed breeding for resistance to *P. sorghi* difficult.

Various genes or alleles for pathotype-specific resistance to common rust in maize (*Zea mays* L.) lines have been identified using isolates of the pathogen (Hooker and Le Roux, 1957; Hooker and Russell, 1962; Lee *et al.*, 1963; Hagan and Hooker, 1965; Wilkinson and Hooker, 1968; Hooker, 1969; Hulbert, Lyons and Bennetzen, 1991). Many resistance genes were backcrossed into a common inbred background and maintained as differentials (near-isogenic or isogenic lines), or incorporated into commercial hybrids (Groth, Pataky and Gingera, 1992). The transfer of *Rp* alleles in susceptible sweet corn hybrids was reported to be easy and ensured effective control of rust in North America until 1980. A small number of *P. sorghi* pathotypes, which were avirulent to most of the *Rp* alleles being used, occurred (Pataky and Headrick, 1989). For instance, hybrids carrying the $Rp1^D$ gene were considered to confer resistance to all variants of *P. sorghi* that commonly occurred in the United States until virulence was reported in 1984 (Bergquist and Pryor, 1984; Pataky, 1986; Hu and Hulbert, 1996; Pataky and Tracy cited by Pataky, 2000; Pate and

Pataky, 2000). Hu and Hulbert (1996) recognised that promising cultivars with high levels of pathotype-specific resistance often failed to reach their potential due to the appearance of new pathotypes.

Differential genetic interactions between host and pathogen genotypes, based on a gene-for-gene system, have been used to differentiate pathotypes of *P. sorghi* isolates using a set of maize differential lines (Hulbert *et al.*, 1991). The term pathotype as applied in this study is a group of individuals in a parasitic population with pathogenicity characters in common, and is used as a synonym for race as defined by Browder, Lyon and Eversmeyer (1980). The aim of this study was to determine genetic variability in *P. sorghi* isolates collected from different localities in South Africa.

MATERIALS AND METHODS

Field isolates of *P. sorghi* were collected from rust infected maize leaves from several localities throughout South Africa during the 1999 and 2000 seasons. Infected leaves, collected from commercial fields or breeders' plots, were placed in envelopes marked with locality and date of collection. While travelling, samples were kept in a cooled container to avoid desiccation of spores. At night envelopes were placed at 4^0C in a refrigerator.

Field isolates were inoculated onto 3-week-old plants of the susceptible hybrid PHB3394, raised in a rust-free greenhouse cubicle. Inoculation was carried out by spraying plants with urediospores suspended in light mineral oil, using the technique described for cereal rust fungi by Browder (1971). The protocol involves the collection of spores from infected leaves into a 1-ml gelatine capsule by means of a cyclone collector connected to a vacuum pump. After adding carrier oil to the spores, the suspension can be sprayed onto plants by attaching the same capsule to an inoculation device connected to an air pressure source. All inoculations were carried out in an enclosed booth which was thoroughly

rinsed between the spraying of different isolates. Inoculated plants were allowed to dry for approximately 1 h before placement in the dark in dew chamber for 16 h. Temperature inside the chamber was kept at 19-22^{0}C. Upon removal from the chamber, plants were transferred to a greenhouse where the different rust isolates were separated in 40 x 40 x 40 cm isolation compartments constructed of a wooden frame and plastic sheeting. No additional lighting was provided and temperature inside the greenhouse varied between 15 and 25^{0}C.

From rust infections developing from the field isolates, pure cultures were established by collecting the spores of a single uredium after 10 to 14 days, using a cyclone spore collector attached to a vacuum pump. These cultures were then reinoculated onto PHB3394 seedlings planted in 10 cm diameter pots (five plants per pot). Reinoculation was carried out at the 2-3-leaf stage using procedures described above. To collect cultures, increased from single pustule isolates, rust-infected leaves were gently tapped to dislodge urediospores onto a clean sheet of paper (Weber, 1922). Care was taken to avoid contamination of isolates. Spores were placed in cryovials and dried in a desiccator (3-4 days) before being stored at –72^{0}C in a low-temperature freezer. Prior to use, spores retrieved from the freezer were heat-shocked at 45^{0}C for 6-7-min. to break their possible dormancy. Prior to inoculation onto differential lines, stored spores were increased on susceptible plants. In cases where isolates gave mixed reactions on differential lines, they were sub-cultured by increasing single pustules in a further cycle of purification.

Twenty-four inbred maize lines carrying previously characterised resistance genes for common rust were used as differentials in greenhouse studies (Table 2.1). Seed of the differential set was originally obtained from D. Nowell, PANNAR Seed Co Pty Ltd., and increased in a commercial greenhouse before use. For pathotype characterisation all differentials and the susceptible control hybrid PHB3394 were grown individually in a steam-

Table 2.1. Typical infection types produced by *Puccinia sorghi* on differentiating maize lines containing specific genes

Line no.			Pathotypes						
	Gene	Source	A	B	C	D	E	F	G
1	*Rp1^{A}*	V20487	;	;	3++	;	;	3+	33+
2	*Rp1^{A}*	V20489	;	;	3++	;	;	3+	;2++
3	*Rp1^{B}*	V20491	;	X+3	;	4	;	;	;
4	*Rp1^{C}*	V20493	;	;1	3	;	;	3+	33+
5	*Rp1^{C}*	V20495	;	;	;	;	;	;	;
6	*Rp1^{D}*	V20499	;	3++	;	3	;	;	;
7	*Rp1^{F}*	V20503	;	;1	3++	;	;	33+	33+
8	*Rp1^{G}*	V20505	;	;	;	;	;	;	;
9	*Rp1^{H}*	V20507	;	3+	;	3++	;	;	;
10	*Rp1^{I}*	V20509	;	3+	;	3	;	;	;
11	*Rp1^{J}*	V20511	;	;	;,3	;	;	3++	X-
12	*Rp1^{K}*	V20513	;	3	;	3++	;	;	;
13	*Rp1^{L}*	V20515	;	;	;	;	;	;	;
14	*Rp1^{M}*	V20517	;	3	;	3++	3++	;	;
15	*Rp1^{N}*	V20519	;	4	;	;,3+	;	;	;
16	*Rp3^{A}*	V20521	3C	;1, 2+3	;	;1+,2+3	3C	;CN	3++
17	*Rp3^{B}*	V20523	4	3++	3	3++	3+	3++	3+
18	*Rp3^{B}*	V20525	;	;	3++	;	;	3++	;
19	*Rp3^{C}*	V20527	;	;1	3	;	;	33+	X+, 33+
20	*Rp3^{D}*	V20529	;	;	;	;	;	;	;
21	*Rp3^{E}*	V20531	;	;	3++	;	;	3++	;
22	*Rp3^{F}*	V20533	;	;	;	;	;	;	;
23	*Rp4^{A}*	V20535	;	3++	;	;1	;	;	;
24	*Rp4^{B}*	V20537	;	3++	;	2	;	;	;
25	Check	PHB3394	3++	3++	3++	4	3++	3++	3++

Infection type was rated in a 0; - 4 scale (Table 1.1, Chapter 1), (;): fleck resistant, C: chlorotic, N: necrotic flecks; "X": intermediate mesothetic type.

Pathotype A isolates: 11-1, 11-1-1, 11-2, 22-1, 24-1, 24-2, 26-1, 28-2, 30-1, 31-1, 32-2, 33-1, 33-2, 34-1, 34-2, 34-2-1, 37b, G-1, Gt 1.

Pathotype B isolates: 23-1, 23-1-1, 23-1-1-1, 23-2, 23-2-1, 27-1, 27- 3, 38b, Gt 2, Gt 5.

Pathotype C isolates: 10-1, 10-2, 35b, 35-1, 35-2.

Pathotype D isolates: 4-2, 4-2-1, 4-2-2.

Pathotype E isolates: 34-2, 34-2-1.

Pathotypes F isolates: Gt 3.

Pathotypes G isolates: Gt 4.

sterilized soil-peat moss mixture in seedling cones (two seeds per cone). When plants reached the 2 to 3-leaf stage, complete differential sets were compiled and inoculated separately with each isolate. Inoculum was prepared by suspending a small quantity of freshly collected urediospores in mineral oil in 1-ml gelatine capsules. The spore suspension was then sprayed directly onto the leaves of rust differentiating plants. After incubation in the dew chamber, plants were placed in the greenhouse where temperatures of 18-23^{0}C, and supplemental lighting of 120 μE/m^2/s for 14 h per day, were maintained. Infection types were rated on a 0-4 scale (Table 1.1, Chapter 1) 10 days after inoculation, when symptoms on susceptible plants were maximally developed. Pathotypes were differentiated according to their reaction patterns (phenotypes) on the differential lines. An isolate or group of isolates giving the same reaction profile was considered one pathotype and given the same capital latter (Table 2.1). All pathotypes were confirmed in at least two replicated inoculation experiments.

RESULTS AND DISCUSSION

The phenotypic expression of the host-pathogen interaction, in the environment tested, allowed differentiation between resistance and susceptibility in the maize host. Based on whether these phenotypes were considered incompatible or compatible, avirulence and virulence for specific *Rp* genes could be determined in South African isolates of *P. sorghi*. Infection types expressed as small chlorotic or necrotic flecks (;C to ;N), or as small to intermediately sized pustules with or without chlorosis or necrosis (1 to 2 infection type range), were considered to indicate avirulence for a specific gene. Well-developed pustules (3 to 4 infection types) indicated a compatible interaction and thus virulence for the corresponding *Rp* gene.

Forty-one isolates, representing 32 field collections (Table 2.2), were differentiated by the 24 differential lines. Seven pathotypes were identified and designated as A, B, C, D, E, F and G (Table 2.1). All pathotypes were virulent (infection types 3 to 4) on the hybrid PHB3394. Pathotype A consisted of 19 isolates, pathotype B 10 isolates, pathotype C five isolates, pathotypes D and E three and two isolates respectively, and one isolate for pathotypes F and G (Table 2.1). Pathotype A was isolated from rust samples collected at 10 localities in the Free State, Gauteng, KwaZulu-Natal, Mpumalanga, Northern Province and North West (Table 2.2). Despite its widespread occurrence this pathotype was virulent only on $Rp3^A$ and one $Rp3^B$ allele (Table 2.1). Pathotype B occurred at four collection sites in Gauteng, KwaZulu-Natal and Mpumalanga. This pathotype was virulent on $Rp1^B$, 1^D, 1^H, 1^I, 1^K, 1^M, 1^N, 3^B, 4^A and 4^B. Gene $Rp1^B$ produced a mesothetic reaction (X+3) to this pathotype whereas the line containing $Rp3^A$ was heterogeneous. Compared to the other pathotypes, B was the most virulent, producing susceptible or moderately susceptible pustules on 11 differential lines (Fig. 2.1). Only two collections, restricted to Mpumalanga and the Northern Province, yielded pathotype C which was virulent to $Rp1^A$ (both alleles), 1^C (one allele), 1^F, 3^B (both alleles), 3^C and 3^E. The $Rp1^J$ line was mixed, displaying fleck and susceptible type reactions to pathotype C.

Pathotype D originated from Piet Retief in Mpumalanga and was characterised by virulence to $Rp1^B$, 1^D, 1^H, 1^I, 1^K, 1^M, and 3^B (one allele only). The differential lines containing $Rp1^N$ and $Rp3^A$ showed high and low reactions to these isolates. Pathotype E, collected only from Potchefstroom, North West, resembled pathotype A except that it had additional virulence for $Rp1^M$.

Table 2.2. The origin and pathotypes of common rust (*Puccinia sorghi*) isolates detected on maize in South Africa

Pathotype	Isolates[a]	Locality	Province
A	11-1, 11-1-1, 11-2	Lydenburg	Mpumalanga
A	22-1	Bloekomspruit	Gauteng
A	24-1, 24-2	Vaalharts	North west Province
A	26-1	Cedara	KwaZulu-Natal
A	28-2	Dargyle	KwaZulu-Natal
A	30-1	Stofberg	Mpumalanga
A	31-1	Bethlehem	Free State
A	32-2, 33-1, 33-2, 34-1, 34-2, 34-2-1	Potchefstroom	North west Province
A	37b	Letsitele	Northern Province
A	G-1, Gt 1	Greytown	KwaZulu-Natal
B	23-1, 23-1-1, 23-1-1-1, 23-2, 23-2-1	Petit	Gauteng
B	27-1, 27-3	Winterton	KwaZulu-Natal
B	38b	Hazyview	Mpumalanga
B	Gt 2, Gt 5	Greytown	KwaZulu-Natal
C	10-1, 10-2	Belfast	Mpumalanga
C	35b, 35-1, 35-2	Letsitele	Northern Province
D	4-2, 4-2-1, 4-2-2	Piet Retief	Mpumalanga
E	34-2, 34-2-1	Potchefstroom	North west Province
F	Gt 3	Greytown	KwaZulu-Natal
G	Gt 4	Greytown	KwaZulu-Natal

[a] Isolate number, e.g. 11-1-1, indicates that the particular collection was sub-cultured two times; whereas 37b indicates that the differentials were inoculated with a bulked collection.

Fig. 2.1. Virulence of *Puccinia sorghi* pathotypes to maize lines containing different *Rp* genes

Pathotype F was similar to C but did not elicit the low reactions on certain *Rp1*^{J} plants as was commonly observed with isolates of the latter. It is possible, however, that these two pathotypes are similar, and that the detection of mixed *Rp1*^{J} genotypes was incidental. In confirmation experiments using a larger number of test plants, isolate 35-1 (pathotype C) consistently produced fleck reactions on half of the *Rp1*^{J} plants and susceptible reactions on the other half, whereas isolate Gt3 (pathotype F) was virulent on all plants. Pathotype G also resembled C and F except that it was avirulent on one *Rp3*^{B} allele and *Rp3*^{E}. Furthermore, the infection types on *Rp1*^{A} (line V20489) did not indicate full susceptibility. Both pathotypes F and G occurred at Greytown only.

Five *Rp* genes, viz. *Rp1*^{C} (line V20495), *1*^{G}, *1*^{L}, *3*^{D} and *3*^{F} conditioned resistance to all isolates tested (Fig. 2.2). The opposite was true for *Rp3*^{B} (line V20523), which was susceptible to all 39 isolates. The designations of the respective *Rp1*^{C} and *Rp3*^{B} genes are questionable because specificity was observed between lines apparently carrying the same allele. The *Rp1* gene consists of multiple loci and Hulbert *et al.* (1991) recognised that resistance of certain *Rp1* alleles may in fact be controlled by different but closely linked genes.

Many sources of resistance to common maize rust were identified in the 1950s and 1960s (Hulbert *et al.*, 1991) and Hagan and Hooker (1965), Wilkinson and Hooker (1968) and Hooker (1969) provided the first published survey data of virulence diversity in *P. sorghi* in the USA. These early studies were followed by investigations of pathogenic variability by Pataky (1987), Pataky and Headrick (1989), Hulbert *et al.* (1991) and Groth *et al.* (1992). In Illinois the resistance genes *Rp1*^{D}, *Rp1*^{E}, *Rp1*^{F}, *Rp*^{G}, *Rp1*^{I} and *Rp3*^{C} were described as being effective during two seasons (Pataky, 1987). However, in more comprehensive field trials representing 21 localities, Groth *et al.* (1992) found that only *Rp1*^{D} and *Rp3*^{C} were

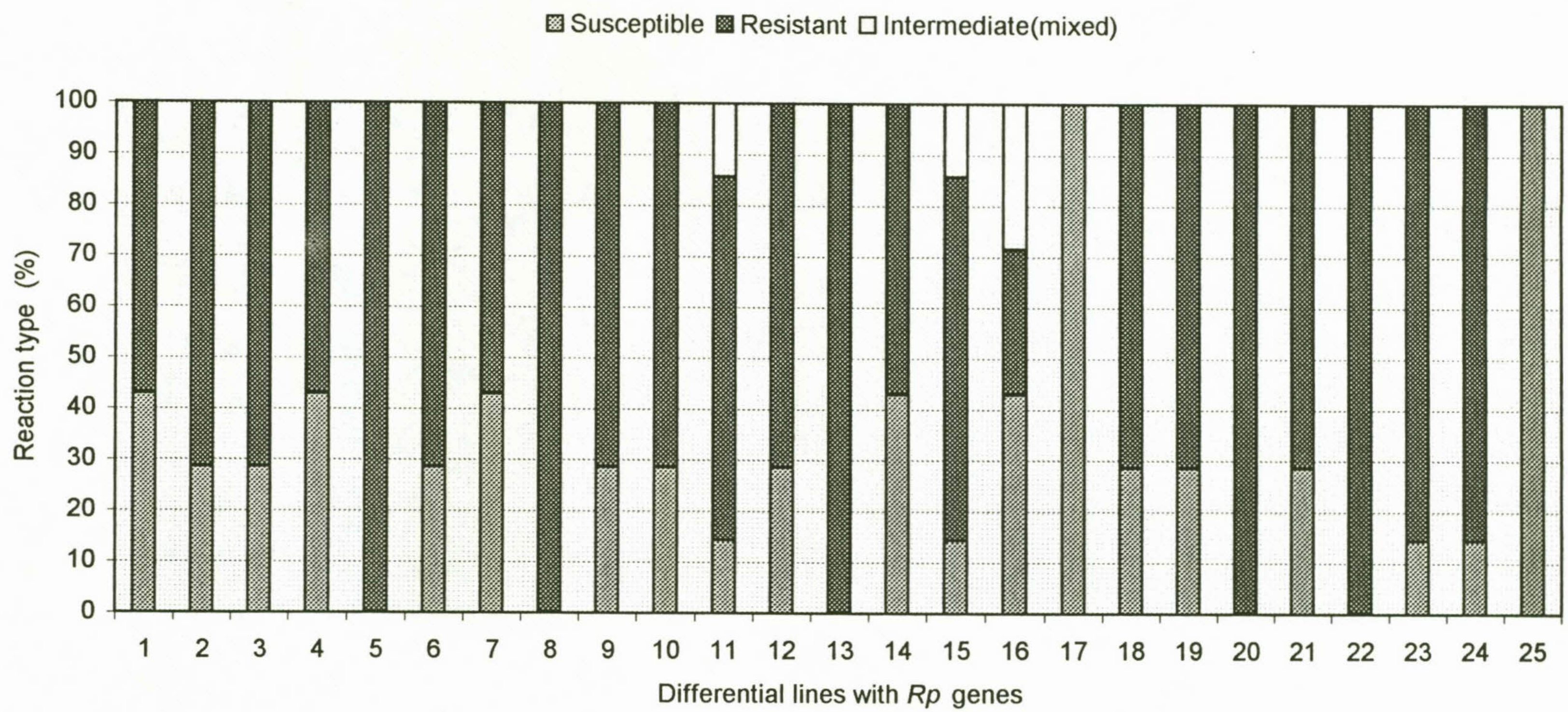

Fig. 2.2: Effectiveness of differential lines to *P. sorghi* pathotypes based on susceptible and resistant reaction types (see Table 2.1 for *Rp* gene designations).

totally effective, i.e. no large uredia developed on lines carrying these genes. Previously, Hulbert *et al.* (1991) also reported virulence in USA isolates of *P. sorghi* to all *Rp* genes tested except $Rp3^C$. Virulence to $Rp1^D$ occurred in isolates from Hawaii, Kansas and Kenya. The Kenyan isolate differed from the South African pathotypes described in the present study, most notably in virulence for $Rp1^G$ and $Rp1^L$.

Differences in reactions among partially resistant sweet corn hybrids were reported from the North American Corn Belt, Hawaii and Mexico (Kim and Brewbaker, 1976; Pataky and Headrick, 1989), suggesting differences between the respective *P. sorghi* populations. However, isolates of *P. sorghi* collected from rust-resistant sweet corn in Illinois, Wisconsin, Minnesota, Michigan and New York in the USA in 1999, and from Florida and Texas in 2000, were similar to those obtained from Mexico (Pataky, 2000; Pate and Pataky, 2000). Susceptible reactions were observed in all sweet corn hybrids and inbred lines with $Rp1^D$, except on $Rp1^E$, $Rp1^G$, $Rp1^I$, $Rp1^K$ and $Rp1^L$. These virulence profiles were different to that reported from previous studies, where the $Rp1^D$ gene had been mostly resistant. This gene was widely used as a resistance source in South America and Mexico, consequently, selection pressure favoured the spread of these virulent pathotypes (Pataky, 2000). Susceptibility of the $Rp1^D$ gene has also been reported from Argentina (Gonzalez, 2000).

The relatively few genes that remain effective to the South African population of *P. sorghi* raise several issues. It is unlikely that many *Rp* genes have been transferred to South African maize cultivars specifically for protection against maize rust. Such directed breeding efforts would have needed pathogenicity information, which has now been obtained for the first time in the region. Virulence to the majority of the *Rp* genes could thus have arisen through mutation as forced by the occurrence of *Rp* genes in local germplasm, migration of exotic pathotypes, or sexual and parasexual recombination of virulence factors. All of these probably contribute to pathogenic variability in *P. sorghi* in South Africa. Maize

breeders often rely on international germ plasm and the inadvertent incorporation of *Rp* genes is not unexpected. However, the widespread occurrence of the relatively avirulent pathotype A suggested that many maize cultivars lack *Rp* genes. Similar to the distribution of pathotype A, Hulbert *et al.* (1991) reported that single pathotypes of *P. sorghi* can occupy large geographical areas and diverse environments.

The rediscovery of aecial infections of *P. sorghi* on *Oxalis corniculata* near Greytown in KwaZulu-Natal in 2000 suggests that sexual recombination contributes to the creation of new virulence combinations. To confirm this, the germination of teliospores, availability and distribution of the alternate host, environmental conditions, and the synchronisation of these with maize growing seasons, have to be investigated in an epidemiological study. Since urediospores are adapted to wind dissemination, it can be anticipated that migration of pathotypes occurs in southern Africa. However, the impact of this mechanism of variation will only be known once maize rust surveys are conducted on a regular basis in South Africa and neighbouring countries.

The recovery of seven pathotypes from a relatively small number of field collections suggests a high degree of variation in this pathogen. More detailed surveys, specifically aimed at the detection of variants below the normal sampling threshold, will provide information useful for resistance breeding and/or screening. Annual nurseries containing *Rp* lines could also be planted at different localities to detect new or unusual virulences. Furthermore, regular surveys will show whether pathotypes of *P. sorghi* are stable and thus useful in resistance breeding programmes, a concern raised by Groth *et al.* (1992). Efforts should be made to purify those differentiating maize lines which were mixed and to select the appropriate $Rp1^{C}$ and $Rp3^{B}$ lines. Similar to other cereal rust breeding programmes it is apparent that monogenic resistance will not be durable. Emphasis in breeding should therefore be placed on horizontal resistance, with particular attention to sources of resistance, screening procedures and

heritability. If a representative collection of *P. sorghi* pathotypes is available, breeders could select efficiently for pathotype non-specific resistance.

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CHAPTER 3

ASSESSMENT OF RESISTANCE TO COMMON RUST IN MAIZE GENOTYPES

ABSTRACT

One hundred maize (*Zea mays* L.) inbred lines from Mozambique, South Africa and Zimbabwe, as well as 58 South African hybrids, were evaluated for resistance to common rust (*Puccinia sorghi)* under field conditions over different localities in South Africa during the 1999/2000 season. Inbred lines were planted at Ermelo and Greytown and inoculated with a mixture of spores from previously identified *P. sorghi* pathotypes. Cultivars were tested under conditions of natural common rust infection at Greytown and Petit. Disease severity was assessed prior to anthesis, at anthesis and after anthesis at all localities except the Greytown cultivar trial, which was assessed only once after anthesis. Disease was scored on a 0-9 scale, converted to percentage leaf diseased area (0-96% scale). The percentage values were log-transformed using the natural logarithm function. Analyses of variance were carried out for each severity parameter (individual rating, mean, sum and AUDPC) within and (mean, sum and AUDPC) across localities. Significant genetic variation exists between genotypes for resistance to *P. sorghi* at each and across localities. Some inbred lines showed significant levels of partial resistance to the pathogen and may be used in resistance breeding programmes. Most cultivars showed susceptible reaction types to common rust and are thus potentially vulnerable to yield losses under conditions of epidemic occurrence of *P. sorghi.*

INTRODUCTION

Common rust, caused by *Puccinia sorghi* Schwein., is a major disease in maize producing regions throughout the world and was reported a serious sweet corn disease in the United States (Pataky, 1987a; Groth, Pataky and Gingera, 1992; Davis *et al.*, 1995; Hu and Hulbert, 1996). Environments favourable for epidemic development result in high yield losses on susceptible commercial hybrids (Groth *et al.*, 1983; Pataky, 1987b), and there is potential for significant yield losses when susceptible inbred lines are used in maize seed production (Wegulo *et al.*, 1998). Yield losses of up to 25% have been reported in temperate regions such as Argentina and the United States (Hooker, 1985). In the tropical climate of Hawaii and Illinois, yield reductions as high as 75% have been reported (Kim and Brewbaker, 1976; Pataky and Eastburn, 1993b). The majority of sweet corn hybrids planted in South Africa originate from the USA and are highly susceptible to *P. sorghi*. Consequently, epidemics are often reported from commercial production fields throughout South Africa (Nowell and Laing, 1998). In certain South African production areas yield losses of up to 40% have been reported (Kaiser and Nowell, 1983).

Pataky and Eastburn (1993b) urged for increased levels of partial resistance for environments, such as northern regions of the USA, where disease pressure is excessive. Levels of resistance of South African commercial cultivars are unknown. The use of susceptible germplasm, along with other factors, such as, late planting and occurrence of new pathotypes virulent to existing resistance genes, have been identified as major factors for increased incidence and severity of maize common rust in Southern Africa. Identification of maize germplasm resistant to common rust would improve resistance breeding (Pataky, 1987b). The objective of this study was to evaluate maize inbred lines and commercial hybrids for rust resistance under field conditions, across localities within South Africa, during the 1999/2000 growing season.

MATERIALS AND METHODS

Evaluation of maize inbred lines

One hundred inbred lines [74 from ARC-Grain Crops Institute, South Africa, 15 from INIA (National Agriculture Research Institute) in Mozambique, 11 from CIMMYT (International Maize and Wheat Improvement Center) in Zimbabwe], and a susceptible control hybrid, PHB3394, were evaluated for rust resistance in the field during the 1999/2000 growing season (Table 3.1). Experiments were planted at Ermelo and Greytown, both sites being situated in the maize production area of South Africa where regular common maize rust epidemics are experienced. Ermelo is situated 1698 m above sea level and Greytown, in the KwaZulu-Natal midlands, at an altitude of 1101 m. Climatic variables (rainfall, daily mean temperature) for both localities during the 1999/2000 season are shown in Fig. 3.1.

The trials were arranged according to a randomised block design with single-row plots of 4.2 m long. All treatments were replicated three times. Row spacing was 0.3 m within and 0.9 m between rows giving a total population of about 37,037 plants/hectare. Thirty seeds were hand-planted in single rows (two seeds per hill) and thinned to 14-15 plants per row (one plant per hill) three weeks after emergence. Two seeds per hill (four to five hills per row) of the PHB3394 hybrid were planted as rust spreader rows in 0.9 m miniplots at the head and foot of each 4.2 m single-row experimental plot. To ensure disease development and high inoculum levels, plants in spreader rows were not thinned. Three to four border rows, of the same hybrid were planted at either end of the blocks.

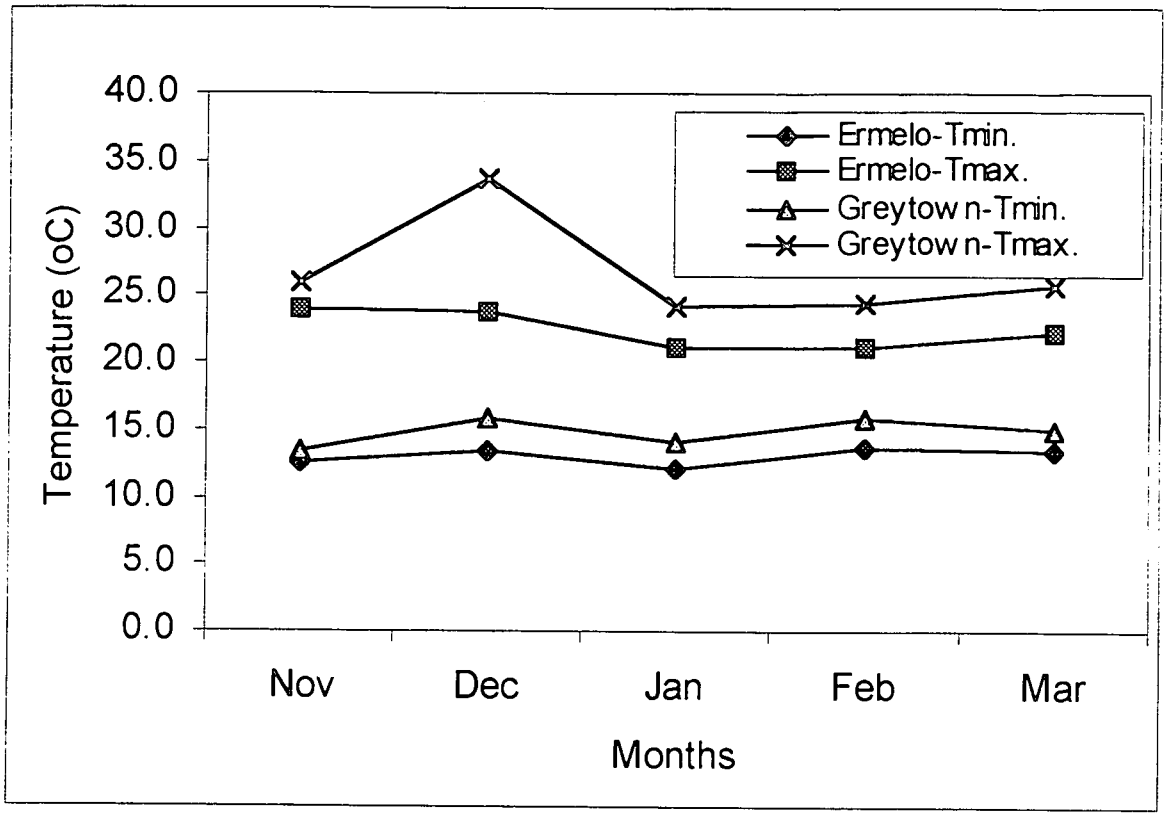
Table 3.1. List of maize inbred lines used in the field evaluation for rust resistance at Ermelo and Greytown (1999/2000 growing season)

Entry	ID/ Name	Source	Entry	ID/ Name	Source	Entry	ID/Name	Source
1	L9798-714 [a]	ARC, South Africa	35	L9798-788	ARC, South Africa	69	L9798-859	ARC, South Africa
2	L9798-715	ARC, South Africa	36	L9798-790	ARC, South Africa	70	L9798-862	ARC, South Africa
3	L9798-724	ARC, South Africa	37	L9798-791	ARC, South Africa	71	L9798-863	ARC, South Africa
4	L9798-720	ARC, South Africa	38	L9798-794	ARC, South Africa	72	L9798-865	ARC, South Africa
5	L9798-725	ARC, South Africa	39	L9798-796	ARC, South Africa	73	L9798-734	ARC, South Africa
6	L9798-728	ARC, South Africa	40	L9798-798	ARC, South Africa	74	L9798-870	ARC, South Africa
7	L9798-731	ARC, South Africa	41	L9798-801	ARC, South Africa	75	MZLP-02 [b]	INIA, Mozambique
8	L9798-733	ARC, South Africa	42	L9798-805	ARC, South Africa	76	MZLP-05	INIA, Mozambique
9	L9798-736	ARC, South Africa	43	L9798-807	ARC, South Africa	77	MZLP-11	INIA, Mozambique
10	L9798-737	ARC, South Africa	44	L9798-808	ARC, South Africa	78	MZLP-18	INIA, Mozambique
11	L9798-738	ARC, South Africa	45	L9798-810	ARC, South Africa	79	MZLP-19	INIA, Mozambique
12	L9798-741	ARC, South Africa	46	L9798-812	ARC, South Africa	80	MZLP-21	INIA, Mozambique
13	L9798-745	ARC, South Africa	47	L9798-813	ARC, South Africa	81	MZLP-22	INIA, Mozambique
14	L9798-747	ARC, South Africa	48	L9798-815	ARC, South Africa	82	MZLP-23	INIA, Mozambique
15	L9798-748	ARC, South Africa	49	L9798-816	ARC, South Africa	83	MZLP-25	INIA, Mozambique
16	L9798-750	ARC, South Africa	50	L9798-817	ARC, South Africa	84	MZLP-26	INIA, Mozambique
17	L9798-752	ARC, South Africa	51	L9798-819	ARC, South Africa	85	MZLP-28	INIA, Mozambique
18	L9798-756	ARC, South Africa	52	L9798-824	ARC, South Africa	86	MZLP-31	INIA, Mozambique
19	L9798-758	ARC, South Africa	53	L9798-825	ARC, South Africa	87	MZLP-34	INIA, Mozambique
20	L9798-759	ARC, South Africa	54	L9798-827	ARC, South Africa	88	MZLP-52	INIA, Mozambique
21	L9798-760	ARC, South Africa	55	L9798-829	ARC, South Africa	89	MZLP-67	INIA, Mozambique
22	L9798-762	ARC, South Africa	56	L9798-830	ARC, South Africa	90	ZCML-386 [c]	CIMMYT, Zimbabwe
23	L9798-764	ARC, South Africa	57	L9798-832	ARC, South Africa	91	ZCML-387	CIMMYT, Zimbabwe
24	L9798-765	ARC, South Africa	58	L9798-833	ARC, South Africa	92	ZCML-389	CIMMYT, Zimbabwe
25	L9798-768	ARC, South Africa	59	L9798-730	ARC, South Africa	93	ZCML-390	CIMMYT, Zimbabwe
26	L9798-769	ARC, South Africa	60	L9798-837	ARC, South Africa	94	ZCML-392	CIMMYT, Zimbabwe
27	L9798-773	ARC, South Africa	61	L9798-842	ARC, South Africa	95	ZCML-393	CIMMYT, Zimbabwe
28	L9798-774	ARC, South Africa	62	L9798-846	ARC, South Africa	96	ZCML-204	CIMMYT, Zimbabwe
29	L9798-777	ARC, South Africa	63	L9798-849	ARC, South Africa	97	ZCML-202	CIMMYT, Zimbabwe
30	L9798-779	ARC, South Africa	64	L9798-850	ARC, South Africa	98	ZCML-205	CIMMYT, Zimbabwe
31	L9798-780	ARC, South Africa	65	L9798-852	ARC, South Africa	99	ZCML-216	CIMMYT, Zimbabwe
32	L9798-781	ARC, South Africa	66	L9798-854	ARC, South Africa	100	ZCML-312	CIMMYT, Zimbabwe
33	L9798-782	ARC, South Africa	67	L9798-855	ARC, South Africa	101	PHB3394	PIONEER
34	L9798-784	ARC, South Africa	68	L9798-856	ARC, South Africa			

[a] ARC (Agriculture Research Council-Summer Grain Crops) lines were coded according to the plot number in the list of 1997/1998 evaluation at KwaZulu-Natal.

[b] Material from INIA (National Agriculture Research Institute Mozambique) coded according to the original names. used by maize programme in Mozambique LP (Pool Line).

[c] CIMMYT (International Maize and Wheat Improvement Center) material was coded as CYMMYT Maize Lines, Zimbabwe.



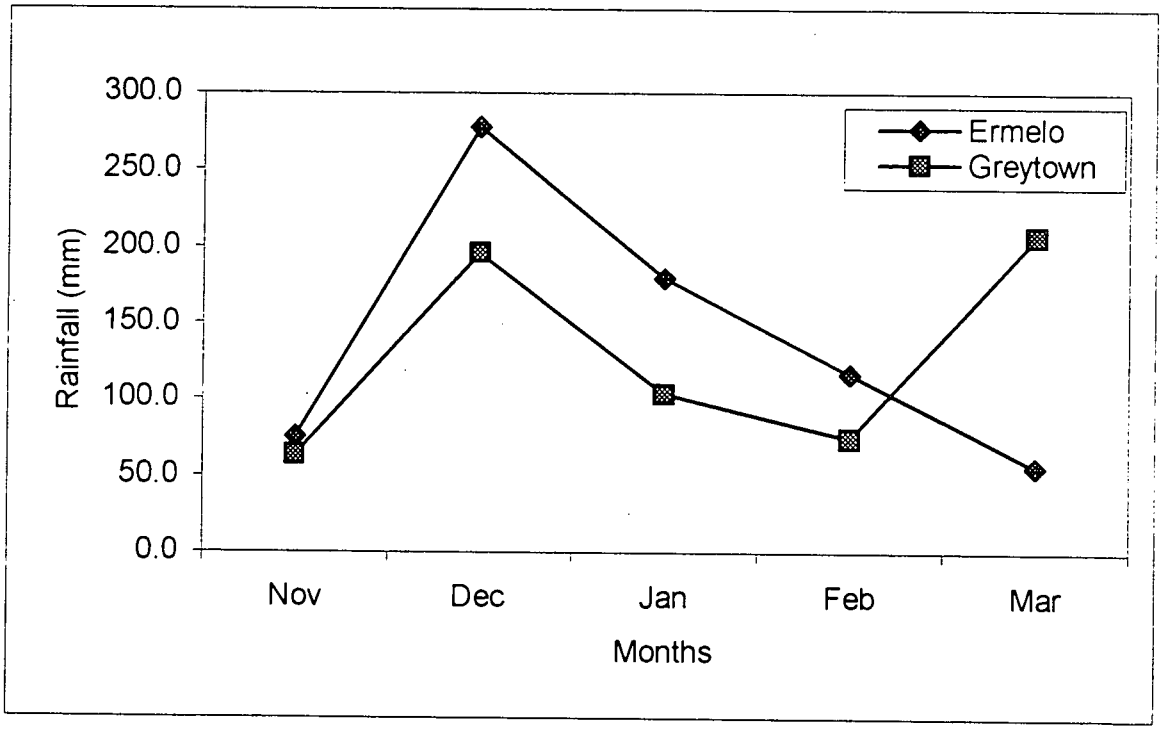


Fig. 3.1. Minimum and maximum temperature (top) and mean monthly rainfall (bottom) for Ermelo and Greytown during the 1999/2000 season.

Fertilisation was applied at 150 kg/ha 4: 3: 4 (N:P:K) (33) at Ermelo and 250 kg/ha of 2: 3: 4 (38) at Greytown. For top dressing, 143 kg/ha LAN (28) and 300 kg/ha urea were applied at Ermelo and Greytown, respectively. The pre-emergence herbicides acetochlor and atrazine/sulcotrione were applied at Ermelo at 1.2 ℓ/ha and 0.8 ℓ/ha, respectively. At Greytown 5 ℓ/ha of EPTC was used. At Ermelo the post-emergence herbicides acetochlor, atrazine/terbuthylazine, and 2,4-D were applied six weeks after planting, at rates of 1.6 ℓ/ha for acetochlor, and 0.3 ℓ/ha for atrazine/terbuthylazine and 2,4-D. At Greytown, the post-emergence herbicide bromoxynil/terbuthylazine was applied five weeks after planting at 1.5 ℓ/ha. At Ermelo 0.07 ℓ/ha and 0.1 ℓ/ha of the insecticide, lambda-cyhalothrin was applied at planting and six weeks after planting, respectively. No insecticide was used at the Greytown trial.

Disease epidemics were created by inoculation of spreader rows (Gingera, Davis and Groth, 1994), at the five to six leaf stage. Inoculum consisted of an urediospore mixture of four previously identified pathotypes. Urediospores were suspended in distilled water with the aid of Tween 20. The inoculum suspension was applied directly into plant whorls with a hand-held sprayer (Pataky, 1987a).

Ratings for rust severity were carried out three times, starting three weeks before anthesis, during anthesis, and two weeks after anthesis. The 0 to 9 scale (Davis, Randle and Groth, 1988) was used for scoring rust severity because it is efficient for assessing disease in the field, especially when large number of genotypes are tested. The intervals used were as follows: 0= immune response, no visible disease; 1= immune response, hypersensitive flecks present; 2= 1-10 single disease areas (pustules and group of pustules) present; 3= 10-20 single disease areas present plus evidence of banding pattern; 4= numerous single disease areas present plus a well defined banding pattern; 5= up to two well-defined banding patterns with numerous single disease areas present; 6= leaf margins becoming necrotic with numerous single disease areas present plus up to

two banding patterns; 7= numerous single disease areas, banding patterns and necrotic margins well defined, 8= all leaf tissue necrotic except for centre of leaf; 9= all leaf tissue necrotic except midrib and some adjoining leaf tissue. Category 4 on the scale is considered a critical threshold, where well defined banding begins, indicating susceptibility (Davis, 1990). In this study scores were based on disease incidence (number of leaves attacked by disease on the plant) and severity (relative percentage of the total leaf area covered by uredinia). To facilitate line selection, a range of severity was recorded for each plot: 10 plants per plot were selected, each plant was rated, and the mean calculated for each entry.

For determination of grain yield in the Greytown trial, each plot was hand-harvested and grain mass and moisture percentage were recorded. Prior to statistical analyses, the rounded mean of each plot for each rating was converted to percentage leaf area diseased (Davis *et al.*., 1988). In this conversion 0 corresponds with absence of disease symptoms and 1 to 9 represent 0.68% (low) and 96% (highest) diseased leaf area, respectively (Table 1.2, Chapter 1).

Except AUDPC and grain yield values, all data were subjected to logarithmic transformation using natural logarithm (LN). Logarithmic transformation is more powerful in cases where the coefficient of variation (CV) is higher and homogeneity is not observed (Van Rooyen, personal communication). Data were submitted to statistical tests of homogeneity before analysis of variance (ANOVA). The statistical software programme Agrobase 98 was used for analysis of the following rust severity parameters: (1) disease severity means for each rating; (2) means of all three ratings; (3) cumulative severity (sum of all ratings); (4) area under disease progress curve (AUDPC) and grain yield for Greytown. AUDPC was determined from the first to the last evaluation according to the following formula:

$$AUDPC = (P1 + P2)/2 * w1 + (P2 + P3)/2 * w2 \qquad (1)$$

P1, P2, and P3 are means of the first, second and third ratings, and w1 and w2 are the number of weeks between ratings 1 and 2, and between rating 2 and 3, respectively. For both localities, Ermelo and Greytown, w1 and w2 were 3 and 2 weeks, corresponding to before anthesis, during anthesis and after anthesis. Grain yield was calculated as shelled grain mass per plot adjusted to 12.5% grain moisture and converted to tons per hectare, according to the following formula:

$$G_Y = (Wt\ Kg / Np) * Pp * (100 - H\%) / 87.5\%) * 1\ t / 1000\ Kg \qquad (2)$$

G_Y is grain yield in t /ha, Wt is grain mass in kg, Np is final stand (number of harvested plants), Pp is plant population (total number of plants/hectare) calculated from plot size, H% is percentage humidity taken after harvest and 87.5% is coefficient of humidity correction (100-12.5%).

The total rust severity variation (S_{ij}) on tested genotypes (G_i) across localities (E_j) were calculated using the classic model for Genotype x Environment interaction (G x E) (Fisher, 1918; 1925) (3). Thus, combined analyses of variance were performed on data of Ermelo and Greytown (mean, sum and AUDPC). Data were pooled for combined analysis (interaction genotypes by localities) across localities.

$$S_{ij} = \mu + G_i + E_j + GE_{ij} + e_{ij} \qquad (3)$$

In this model μ is the general mean of severity, G_I, E_j and GE_{ij} represent the effect of the genotype, environment and genotype x environment interaction respectively, and e_{ij} represents the average of random errors related with the r^{th} plot containing the i^{th} genotype in j^{th} environment. Differences in resistance were based on significant differences (LSD, $P < 0.05$) using log-transformed data for mean and sum values. Sum of squares was partitioned for inbred line/localities effects.

Evaluation of maize hybrids

Fifty-eight commercial hybrids (Table 3.2) from several South African seed companies were evaluated for rust resistance under natural infection at Petit and Greytown during the 1999/2000 growing season. The trial consisted of three replicates of four rows per plot in a randomised block design. Rows were 6 m long, spaced 0.76 m apart. Within-row spacing was 0.30 m, providing a total population of about 65,785 plants/hectare. Ten plants from the two centre rows of each plot were rated for rust severity as described previously. The first rating at Petit was taken when the ninth or tenth leaf was completely exposed, and the second and third ratings at early anthesis and after anthesis, respectively. At Greytown, rust was rated once, between two and three weeks after anthesis. Disease assessment and statistical analysis were done in the same way as previously described for inbred lines. Data were submitted to natural logarithmic transformation, except AUDPC values after which analysis of variance was performed on severity parameters for each locality. As the Greytown data consisted of only one rating, no genotype by environment interaction could be determined across localities. Spearman's rank correlations were used to determine consistency of resistance of cultivars over the two localities. Greytown data were correlated to the mean, sum and AUDPC values from Petit. Correlations between ratings and distribution of genotypes in different classes of severity were performed using the statistical programme Agrobase 98. The relationship between disease and yield of inbred lines at Greytown was studied using Statgraphics.

RESULTS AND DISCUSSION

There was considerable variation in rust development between Ermelo and Greytown on maize inbred lines screened for resistance during the 1999/2000 growing season. Although moderate temperatures and high

Table 3.2. Severity means of common rust on maize cultivars evaluated for resistance at Petit and Greytown (1999/2000 season)

		Petit																Greytown		
Entry	Name[a]	AUDPC[b]	Rank[c]	Rating1[d]	LogTD[e]	Rank	Rating2	LogTD	Rank	Rating3	LogT	Rank	Mean[f]	LogT	Ran	Sum[g]	LogT	Rank	Rating[h]	LogTD	Rank
49	CRN 3891	15.30	1	2.10	0.74	1	2.92	1.07	1	2.92	1.07	1	2.69	0.99	1	8.17	2.10	1	1.43	0.36	1
21	PHB 30H22	26.10	2	2.92	1.07	5	5.70	1.74	8	5.70	1.74	9	4.85	1.58	4	14.44	2.67	8	2.92	1.07	8
13	PAN 6414	26.10	3	2.92	1.07	3	5.70	1.74	5	5.70	1.74	11	4.85	1.58	3	14.44	2.67	5	2.92	1.07	5
7	PAN 6480	26.10	4	2.92	1.07	2	5.70	1.74	9	5.70	1.74	10	4.85	1.58	2	14.44	2.67	9	4.10	1.41	16
12	NS 9100	27.30	5	4.10	1.41	10	5.70	1.74	7	5.70	1.74	7	5.26	1.66	5	14.44	2.67	7	2.92	1.07	4
22	PAN 6242	28.50	6	5.70	1.74	23	5.70	1.74	2	5.70	1.74	2	5.70	1.74	9	14.44	2.67	2	4.10	1.41	11
24	QS 7608	28.50	7	5.70	1.74	20	5.70	1.74	6	5.70	1.74	6	5.70	1.74	8	14.44	2.67	6	2.92	1.07	6
4	PAN 6568	28.50	8	5.70	1.74	26	5.70	1.74	4	5.70	1.74	8	5.70	1.74	7	14.44	2.67	4	4.10	1.41	12
57	PAN 6734	28.50	9	5.70	1.74	24	5.70	1.74	3	5.70	1.74	3	5.70	1.74	6	14.44	2.67	3	5.16	1.64	26
11	CRN 3524	33.33	10	5.70	1.74	18	7.17	1.97	15	5.70	1.74	4	6.30	1.84	11	16.44	2.80	11	2.10	0.74	3
10	PAN 6710	33.33	11	5.70	1.74	32	7.17	1.97	10	5.70	1.74	5	6.30	1.84	12	16.44	2.80	10	5.16	1.64	22
3	CRN 3604	35.03	12	4.10	1.41	6	7.17	1.97	12	7.17	1.97	12	6.23	1.83	10	17.81	2.88	12	5.70	1.74	30
55	CRN 3852	36.23	13	5.70	1.74	19	7.17	1.97	14	7.17	1.97	14	6.75	1.91	14	17.81	2.88	13	2.01	0.70	2
35	PAN 6479	36.73	14	2.92	1.07	4	7.17	1.97	17	9.12	2.21	18	6.49	1.87	13	19.89	2.99	16	2.92	1.07	7
40	PAN 6615	39.13	15	5.70	1.74	22	7.17	1.97	16	9.12	2.21	16	7.46	2.01	16	19.89	2.99	14	5.16	1.64	27
41	SNK 2945	39.13	16	5.70	1.74	25	7.17	1.97	18	9.12	2.21	21	7.46	2.01	15	19.89	2.99	17	4.10	1.41	20
45	PAN 6777	41.07	17	7.17	1.97	37	7.17	1.97	13	9.12	2.21	15	7.92	2.07	18	19.89	2.99	15	4.10	1.41	10
30	PAN 6243	43.97	18	5.70	1.74	28	9.12	2.21	22	9.12	2.21	17	8.08	2.09	19	21.98	3.09	18	4.10	1.41	15
43	SNK 2957	43.97	19	7.17	1.97	38	7.17	1.97	11	11.47	2.44	25	8.76	2.17	21	22.42	3.11	19	5.16	1.64	21
1	PAN 6146	44.70	20	4.10	1.41	9	11.47	2.44	27	7.17	1.97	13	7.77	2.05	17	22.87	3.13	20	4.10	1.41	17
38	PAN 6573	46.87	21	5.70	1.74	21	9.12	2.21	19	11.47	2.44	28	8.85	2.18	23	24.53	3.20	24	5.16	1.64	29
14	SNK 2972	47.02	22	4.10	1.41	8	9.12	2.21	23	10.91	2.39	22	8.41	2.13	20	24.05	3.18	22	5.16	1.64	25
42	PAN 6823	50.15	23	7.17	1.97	40	9.12	2.21	20	10.91	2.39	23	9.30	2.23	24	24.05	3.18	21	4.10	1.41	9
2	SNK 2472	51.70	24	5.70	1.74	27	11.47	2.44	28	11.47	2.44	27	9.58	2.26	25	27.11	3.30	26	9.12	2.21	41
16	PAN 6364	51.78	25	5.16	1.64	16	10.91	2.39	24	9.12	2.21	19	8.76	2.17	22	24.29	3.19	23	4.10	1.41	13
5	SNK 2682	53.63	26	7.17	1.97	42	11.47	2.44	29	11.47	2.44	29	10.18	2.32	28	27.11	3.30	27	4.10	1.41	18
44	CRN 3815	54.98	27	9.12	2.21	44	9.12	2.21	21	13.87	2.63	33	10.80	2.38	29	26.84	3.29	25	6.49	1.87	32
28	PAN 6043	55.88	28	7.17	1.97	41	10.91	2.39	25	11.47	2.44	30	10.18	2.32	27	27.11	3.30	28	5.16	1.64	28
47	SNK 2969	56.62	29	5.16	1.64	12	13.87	2.63	30	9.12	2.21	20	9.58	2.26	26	27.66	3.32	29	7.85	2.06	40
9	CRN 3760	59.82	30	9.12	2.21	49	11.47	2.44	26	13.87	2.63	35	11.82	2.47	34	29.67	3.39	30	6.49	1.87	35
51	SNK 2626	60.72	31	7.17	1.97	43	13.87	2.63	36	11.47	2.44	26	11.13	2.41	30	29.96	3.40	32	9.12	2.21	43
52	CRN 1598	60.72	32	7.17	1.97	39	13.87	2.63	31	11.47	2.44	24	11.13	2.41	31	29.96	3.40	31	6.49	1.87	33
54	PAN 6561	63.03	33	5.70	1.74	34	13.87	2.63	35	13.87	2.63	31	11.13	2.41	32	32.14	3.47	33	7.17	1.97	37
19	PAN 6332	63.03	34	5.70	1.74	17	13.87	2.63	32	13.87	2.63	32	11.36	2.43	33	32.79	3.49	34	4.10	1.41	14
32	CRN 3549	67.28	35	5.70	1.74	33	13.87	2.63	34	16.61	2.81	37	12.18	2.50	35	35.16	3.56	35	6.49	1.87	34
37	PHB 3203	70.85	36	5.16	1.64	15	16.61	2.81	37	13.87	2.63	34	12.55	2.53	36	35.52	3.57	37	5.16	1.64	24
33	PAN 6335	73.08	37	11.47	2.44	55	13.87	2.63	33	16.61	2.81	36	14.15	2.65	39	35.16	3.56	36	15.33	2.73	51
36	SNK 2959	74.37	38	5.70	1.74	30	16.61	2.81	38	16.61	2.81	38	13.07	2.57	37	38.09	3.64	38	9.12	2.21	44

Table 3.2 (Cont.) Severity means of common rust on maize cultivars evaluated for resistance at Petit and Greytown (1999/2000 season)

		Petit																	Greytown		
Entry	Name[a]	AUDPC[b]	Rank[c]	Rating1[d]	LogTD[e]	Rank	Rating2	LogTD	Rank	Rating3	LogT	Rank	Mean[f]	LogT	Ran	Sum[g]	LogT	Rank	Rating[h]	LogTD	Rank
23	CRN 4502	80.25	39	4.10	1.41	7	20.09	3.00	41	16.61	2.81	40	13.74	2.62	38	42.10	3.74	41	7.85	2.06	38
8	SNK 2266	81.98	40	9.12	2.21	45	16.61	2.81	40	16.78	2.82	41	14.73	2.69	41	39.25	3.67	39	5.16	1.64	23
29	SNK 2401	82.18	41	5.16	1.64	14	20.09	3.00	42	16.61	2.81	39	14.44	2.67	40	42.10	3.74	40	10.91	2.39	47
18	LS 8502	87.62	42	5.70	1.74	35	16.61	2.81	39	19.30	2.96	43	14.73	2.69	42	42.95	3.76	42	6.49	1.87	31
53	SNK 2782	95.25	43	11.47	2.44	54	20.09	3.00	43	20.29	3.01	44	17.64	2.87	44	46.53	3.84	44	9.12	2.21	45
34	SNK 2721	95.32	44	9.12	2.21	50	20.29	3.01	44	16.78	2.82	42	16.12	2.78	43	44.70	3.80	43	7.85	2.06	39
27	CRN 7821B	110.90	45	9.12	2.21	47	24.29	3.19	45	24.29	3.19	46	19.30	2.96	46	54.05	3.99	45	7.17	1.97	36
25	SNK 2340B	115.77	46	5.16	1.64	13	24.29	3.19	46	29.67	3.39	47	20.49	3.02	47	59.74	4.09	47	16.61	2.81	55
6	PHB 3442	118.53	47	5.70	1.74	31	24.53	3.20	47	23.34	3.15	45	18.54	2.92	45	54.60	4.00	46	4.10	1.41	19
39	SNK 2811	129.10	48	5.16	1.64	11	29.67	3.39	50	29.67	3.39	48	21.54	3.07	48	65.37	4.18	48	9.12	2.21	42
50	PAN 6256	134.17	49	11.47	2.44	56	29.67	3.39	52	29.67	3.39	49	23.81	3.17	50	65.37	4.18	49	10.91	2.39	49
20	CRN 3818	140.37	50	5.70	1.74	29	28.22	3.34	48	34.12	3.53	52	22.87	3.13	49	68.72	4.23	50	13.20	2.58	50
48	PAN 6633	141.73	51	9.12	2.21	46	29.67	3.39	49	34.12	3.53	50	24.78	3.21	51	70.11	4.25	51	10.91	2.39	46
17	SNK 2778	151.67	52	11.47	2.44	57	29.67	3.39	51	41.26	3.72	53	27.66	3.32	53	77.48	4.35	53	15.96	2.77	53
15	CRN 3414	159.50	53	11.47	2.44	58	34.12	3.53	53	34.12	3.53	51	26.84	3.29	52	74.44	4.31	52	18.54	2.92	56
56	SNK 2147	186.47	54	7.17	1.97	36	41.26	3.72	54	47.94	3.87	54	32.46	3.48	54	95.58	4.56	54	10.91	2.39	48
31	SNK 2021	200.73	55	10.91	2.39	51	41.26	3.72	55	55.15	4.01	57	36.23	3.59	56	102.51	4.63	55	15.96	2.77	52
46	LS 8503	207.07	56	10.91	2.39	52	47.94	3.87	57	47.94	3.87	55	36.23	3.59	55	103.54	4.64	56	19.49	2.97	57
58	SNK 2975	215.67	57	11.47	2.44	53	47.94	3.87	56	55.15	4.01	56	38.09	3.64	57	109.95	4.70	57	16.61	2.81	54
26	PHB33A14	234.57	58	9.12	2.21	48	54.05	3.99	58	55.15	4.01	58	40.04	3.69	58	116.75	4.76	58	19.49	2.97	58
Mean		77.00		6.18	1.82		13.09	2.57		13.53	2.60		11.38	2.43		34.19	3.53		7.45	2.01	
LSD (0.05)		33.76			0.57			0.46			0.50			0.37			0.37			0.59	
Significance		**			**			**			*			**			**			**	
CV (%)		32.19			22.97			13.14			14.09			11.16			7.69			23.66	

[a] Name: maize cultivars from several South African seed companies.

[b] Area under disease progress curve from two weeks before anthesis to three weeks after anthesis.

[c] Ranking of genotypes according to the LSD values for each parameter analysed, where low values of severity represent resistant genotypes.

[d] Ratings of severity means assessed two weeks before anthesis, during anthesis, and three weeks after anthesis, respectively.

[e] LogTD: Transformed data using logarithm function (Ln).

[f] Mean rust severity for three ratings, measured as relative percent leaf area infected (0- 96% scale) (Davis *et al*., 1988).

[g] Cumulative plant leaf area diseased (sum of severity means of three ratings).

[h] Rating taken three weeks after anthesis in Greytown.

LSD ($P < 0.05$), CV (%) were calculated from logarithm values (LogTD).

**Highly significant ($P<0.05$) and * significant ($P<0.1$)

rainfall and humidity generally favour common rust development throughout South Africa (B.C. Flett and M.J. Bensch, unpublished), high rainfall during the 1999/2000 growing season at Ermelo (Fig. 3.1) appeared to be a limiting factor for disease development and adequate plant growth. Furthermore, the majority of inbred lines were poorly adapted to the Ermelo environment, more so than at than Greytown, this primarily being germplasm from Mozambique and Zimbabwe. Pataky and Eastburn (1993a) observed that local weather conditions may result in significant variation in development of common rust over localities and seasons. It is also well known that due to their biotrophic nature, the incidence and severity of rust fungi are correlated to plant vigour where vigorously growing plants favour disease development (Jugenheimer, 1958; Anonymous, 1977; Agrios, 1988).

Individual ANOVA, with regard to common rust severity and grain yield values for inbred lines tested, provided high CVs for the first and second ratings at Ermelo, relative to the third rating and all three ratings at Greytown. The high CV for severity ratings at Ermelo can possibly be explained by variation caused by environmental factors. Differences in host growth stage, as well as a more uniform distribution of inoculum later in the season, may also have influenced rust development at Ermelo. Headrick and Pataky (1987) observed that differences in partial resistance among hybrids were more evident on adult plants than seedlings, because several infection cycles would occur before evaluation of adult plants.

The mean rust severity values and grain yield of inbred lines and one hybrid evaluated at Greytown and Ermelo are given in Tables 3.3 and 3.4. According to LSD values at $P<0.05$, significant differences occurred among means for each rating, general mean (mean), cumulative means (sum), and AUDPC at both localities and for yield means at Greytown. Rust severity ranged from 0.68 to 13.20% for the first rating, 0.99 to 35.87% for the second rating, and from 0.99 to 95.58% for the third rating (Table

Table 3.3. Severity means of common rust and grain yield values of maize inbred lines evaluated for resistance at Greytown (1999/2000 season)

Entry	Code/Name[a]	AUDPC[b]	Rank[c]	Rating1[d]	LogTD[e]	Rank	Rating2	LogTD	Rank	Rating	LogTD	Rank	Mean[f]	LogTD	Rank	Sum[g]	LogTD	Rank	Y(t/ha)[h]	Rank
16	L9798-750	5.51	1	0.68	-0.39	2	0.99	-0.01	2	1.43	0.36	2	1.23	0.21	1	3.25	1.18	2	2.13	22
93	ZCML-390	5.75	2	0.99	-0.01	3	0.99	-0.01	1	0.99	-0.01	1	1.28	0.25	2	2.97	1.09	1	1.67	49
47	L9798-813	8.35	3	0.68	-0.39	1	2.10	0.74	5	2.10	0.74	4	1.63	0.49	3	4.90	1.59	3	2.51	7
21	L9798-760	9.06	4	0.99	-0.01	5	2.10	0.74	6	2.10	0.74	5	1.77	0.57	4	5.31	1.67	5	1.83	38
100	ZCML-312	9.32	5	1.43	0.36	12	1.43	0.36	3	2.01	0.70	3	1.90	0.64	5	5.00	1.61	4	2.17	20
96	ZCML-204	10.26	6	0.99	-0.01	6	2.10	0.74	9	2.92	1.07	9	2.05	0.72	7	6.17	1.82	7	1.45	73
25	L9798-768	10.26	7	0.99	-0.01	4	2.10	0.74	4	2.92	1.07	6	2.05	0.72	6	6.17	1.82	6	1.46	71
32	L9798-781	10.97	8	1.43	0.36	13	2.10	0.74	12	2.92	1.07	12	2.25	0.81	9	6.75	1.91	9	1.77	42
33	L9798-782	10.97	9	1.43	0.36	11	2.10	0.74	11	2.92	1.07	15	2.25	0.81	8	6.75	1.91	8	2.57	5
95	ZCML-393	11.68	10	2.10	0.74	17	2.10	0.74	7	2.92	1.07	7	2.44	0.89	10	7.32	1.99	11	0.83	97
29	L9798-777	11.68	11	2.10	0.74	22	2.10	0.74	8	2.92	1.07	14	2.44	0.89	11	7.32	1.99	10	2.11	23
10	L9798-737	12.88	12	2.10	0.74	30	2.10	0.74	10	4.10	1.41	18	2.83	1.04	15	8.50	2.14	15	1.40	81
94	ZCML-392	13.97	13	1.43	0.36	9	2.92	1.07	15	2.92	1.07	8	2.48	0.91	12	7.46	2.01	12	2.14	21
26	L9798-769	14.46	14	0.99	-0.01	7	2.92	1.07	19	4.10	1.41	21	2.77	1.02	14	8.33	2.12	14	1.77	41
15	L9798-748	14.68	15	2.10	0.74	36	2.92	1.07	16	2.92	1.07	11	2.83	1.04	16	8.50	2.14	16	1.38	82
37	L9798-791	14.94	16	2.10	0.74	21	3.06	1.12	21	2.92	1.07	13	2.75	1.01	13	8.25	2.11	13	1.54	65
68	L9798-856	16.37	17	1.43	0.36	10	2.92	1.07	14	5.70	1.74	27	3.53	1.26	20	10.59	2.36	20	1.66	54
19	L9798-758	17.08	18	2.10	0.74	50	2.92	1.07	20	5.70	1.74	32	3.63	1.29	23	10.91	2.39	22	2.22	16
83	MZLP-25	17.08	19	2.10	0.74	45	2.92	1.07	17	5.70	1.74	31	3.63	1.29	22	10.91	2.39	23	2.44	10
66	L9798-854	17.68	20	2.92	1.07	52	2.92	1.07	18	4.10	1.41	19	3.39	1.22	19	10.18	2.32	19	2.48	8
67	L9798-855	17.68	21	2.10	0.74	48	4.10	1.41	34	2.92	1.07	10	3.13	1.14	17	9.39	2.24	17	2.09	26
89	MZLP-67	17.81	22	2.10	0.74	26	2.92	1.07	13	5.16	1.64	22	3.53	1.26	21	10.59	2.36	21	2.37	11
35	L9798-788	20.08	23	2.10	0.74	19	4.10	1.41	28	5.70	1.74	28	4.06	1.40	26	12.18	2.50	26	1.74	43
18	L9798-756	20.08	24	2.10	0.74	39	4.10	1.41	29	5.70	1.74	36	4.06	1.40	29	12.18	2.50	29	2.20	19
53	L9798-825	20.08	25	2.10	0.74	24	4.10	1.41	31	5.70	1.74	24	4.06	1.40	25	12.18	2.50	25	2.48	9
17	L9798-752	20.08	26	2.10	0.74	46	4.10	1.41	35	5.70	1.74	38	4.06	1.40	28	12.18	2.50	28	1.65	55
91	ZCML-387	20.08	27	2.10	0.74	18	4.10	1.41	26	5.70	1.74	34	4.06	1.40	27	12.18	2.50	27	1.40	79
9	L9798-736	20.68	28	2.92	1.07	70	4.10	1.41	30	4.10	1.41	20	3.78	1.33	24	11.36	2.43	24	1.67	52
11	L9798-738	21.88	29	2.92	1.07	55	4.10	1.41	25	5.70	1.74	25	4.39	1.48	32	13.20	2.58	32	1.62	57
72	L9798-865	22.01	30	2.10	0.74	49	4.10	1.41	36	7.17	1.97	49	4.71	1.55	39	14.15	2.65	39	1.42	76
99	ZCML-216	22.02	31	1.43	0.36	14	4.10	1.41	24	6.49	1.87	39	4.06	1.40	30	12.18	2.50	30	1.46	72
69	L9798-859	22.61	32	2.10	0.74	33	5.70	1.74	49	3.71	1.31	16	4.10	1.41	31	12.30	2.51	31	1.69	47
50	L9798-817	23.03	33	2.05	0.72	16	5.70	1.74	51	5.70	1.74	26	4.48	1.50	36	13.46	2.60	36	2.83	3
30	L9798-779	23.08	34	2.10	0.74	34	5.70	1.74	41	5.70	1.74	23	4.48	1.50	34	13.46	2.60	34	1.68	48
54	L9798-827	23.08	35	2.10	0.74	23	5.70	1.74	46	5.70	1.74	29	4.48	1.50	35	13.46	2.60	35	2.26	14
55	L9798-829	23.08	36	2.10	0.74	40	5.70	1.74	47	5.70	1.74	37	4.48	1.50	37	13.46	2.60	37	1.51	68
92	ZCML-389	23.08	37	2.10	0.74	35	5.70	1.74	59	5.70	1.74	35	4.48	1.50	38	13.46	2.60	38	1.10	92
57	L9798-832	23.68	38	4.10	1.41	79	4.10	1.41	37	5.70	1.74	33	4.85	1.58	41	14.59	2.68	41	1.71	45
48	L9798-815	23.81	39	2.92	1.07	65	4.10	1.41	32	7.17	1.97	48	4.95	1.60	43	14.88	2.70	43	1.63	56
59	L9798-730	23.81	40	2.92	1.07	56	4.10	1.41	33	7.17	1.97	46	4.81	1.57	40	14.44	2.67	40	1.42	77
20	L9798-759	23.82	41	2.10	0.74	47	3.71	1.31	23	7.17	1.97	44	4.48	1.50	33	13.46	2.60	33	2.22	17
24	L9798-765	23.93	42	2.10	0.74	27	4.10	1.41	27	9.12	2.21	59	5.31	1.67	46	15.96	2.77	46	2.07	28

Table 3.3 (Cont.) Severity means of common rust and grain yield values of maize inbred lines evaluated for resistance at Greytown (1999/2000 season)

Entry	Code/Name[a]	AUDPC[b]	Rank[c]	Rating1[d]	LogTD[e]	Rank	Rating2	LogTD	Rank	Rating	LogTD	Rank	Mean[f]	LogTD	Rank	Sum[g]	LogTD	Rank	Y(t/ha)[h]	Rank
22	L9798-762	24.88	43	2.92	1.07	54	5.70	1.74	54	5.70	1.74	30	4.85	1.58	42	14.59	2.68	42	2.02	30
71	L9798-863	25.01	44	2.10	0.74	31	5.70	1.74	57	7.17	1.97	40	5.05	1.62	44	15.18	2.72	44	1.61	60
90	ZCML-386	25.01	45	2.10	0.74	25	5.70	1.74	40	7.17	1.97	43	5.05	1.62	45	15.18	2.72	45	1.97	32
74	L9798-870	26.22	46	1.43	0.36	15	5.70	1.74	48	9.12	2.21	54	5.58	1.72	49	16.61	2.81	49	1.25	89
36	L9798-790	26.81	47	2.92	1.07	66	5.70	1.74	43	7.17	1.97	47	5.47	1.70	48	16.44	2.80	48	2.07	29
65	L9798-852	26.81	48	2.92	1.07	57	5.70	1.74	56	7.17	1.97	42	5.37	1.68	47	16.12	2.78	47	1.58	62
58	L9798-833	26.93	49	2.10	0.74	44	5.70	1.74	44	9.12	2.21	64	5.70	1.74	52	17.12	2.84	52	1.84	36
1	L9798-714	26.93	50	2.10	0.74	37	5.70	1.74	50	9.12	2.21	57	5.70	1.74	50	17.12	2.84	50	1.71	46
13	L9798-745	28.61	51	4.10	1.41	74	5.70	1.74	45	7.17	1.97	45	5.81	1.76	55	17.46	2.86	56	2.56	6
12	L9798-741	28.61	52	4.10	1.41	77	5.70	1.74	42	7.17	1.97	53	5.81	1.76	54	17.46	2.86	54	2.32	13
76	MZLP-05	28.61	53	4.10	1.41	72	5.70	1.74	52	7.17	1.97	41	5.81	1.76	53	17.46	2.86	53	1.11	91
31	L9798-780	28.73	54	2.92	1.07	59	5.70	1.74	60	9.12	2.21	67	6.05	1.80	58	18.17	2.90	57	1.35	84
86	MZLP-31	28.73	55	2.92	1.07	64	5.70	1.74	55	9.12	2.21	56	6.05	1.80	57	18.17	2.90	58	1.98	31
46	L9798-812	28.75	56	2.10	0.74	43	5.16	1.64	38	9.12	2.21	55	5.81	1.76	56	17.46	2.86	55	1.53	67
97	ZCML-202	29.46	57	1.38	0.32	8	3.71	1.31	22	3.71	1.31	17	3.22	1.17	18	9.68	2.27	18	1.42	75
70	L9798-862	29.82	58	2.10	0.74	32	7.17	1.97	61	7.17	1.97	51	5.70	1.74	51	17.12	2.84	51	1.86	35
84	MZLP-26	31.60	59	2.92	1.07	62	5.70	1.74	58	10.91	2.39	68	6.69	1.90	63	20.09	3.00	63	1.91	34
23	L9798-764	31.75	60	2.10	0.74	29	7.17	1.97	69	9.12	2.21	60	6.23	1.83	59	18.73	2.93	59	1.83	37
63	L9798-849	33.55	61	2.92	1.07	53	7.17	1.97	65	9.12	2.21	58	6.49	1.87	60	19.49	2.97	60	2.07	27
4	L9798-720	33.55	62	2.92	1.07	61	7.17	1.97	73	9.12	2.21	66	6.55	1.88	61	19.69	2.98	61	3.01	2
14	L9798-747	33.55	63	2.92	1.07	63	7.17	1.97	70	9.12	2.21	63	6.55	1.88	62	19.69	2.98	62	2.10	24
75	MZLP-02	34.26	64	5.70	1.74	88	5.70	1.74	53	11.47	2.44	75	7.61	2.03	71	22.87	3.13	71	1.50	69
43	L9798-807	35.23	65	5.70	1.74	84	7.17	1.97	64	7.17	1.97	50	6.75	1.91	65	20.29	3.01	65	2.66	4
5	L9798-725	35.23	66	5.70	1.74	87	7.17	1.97	63	7.17	1.97	52	6.75	1.91	64	20.29	3.01	64	1.58	63
28	L9798-774	35.35	67	4.10	1.41	73	7.17	1.97	62	9.12	2.21	61	6.89	1.93	67	20.70	3.03	67	2.20	18
45	L9798-810	35.35	68	4.10	1.41	75	7.17	1.97	72	9.12	2.21	62	6.89	1.93	66	20.70	3.03	66	1.32	85
7	L9798-731	35.47	69	2.92	1.07	69	7.17	1.97	67	11.47	2.44	73	7.32	1.99	68	21.98	3.09	68	3.38	1
49	L9798-816	35.47	70	2.92	1.07	60	7.17	1.97	71	11.47	2.44	72	7.39	2.00	69	22.20	3.10	69	0.95	95
62	9798-846	36.54	71	2.10	0.74	28	7.17	1.97	66	13.87	2.63	76	7.77	2.05	72	23.10	3.14	72	1.79	40
77	MZLP-11	38.81	72	2.10	0.74	38	5.16	1.64	39	16.78	2.82	83	8.94	2.19	77	26.84	3.29	77	0.70	98
73	L9798-734	39.07	73	5.70	1.74	81	7.17	1.97	68	11.47	2.44	74	8.25	2.11	75	24.78	3.21	75	1.03	93
88	MZLP-52	40.16	74	4.10	1.41	76	9.12	2.21	77	9.12	2.21	65	7.46	2.01	70	22.42	3.11	70	1.61	59
64	L9798-850	40.29	75	2.92	1.07	58	9.12	2.21	78	11.47	2.44	71	8.08	2.09	73	24.29	3.19	73	1.59	61
87	MZLP-34	41.23	76	2.92	1.07	51	9.12	2.21	81	10.91	2.39	69	8.08	2.09	74	24.29	3.19	74	1.73	44
44	L9798-808	43.89	77	5.70	1.74	83	9.12	2.21	82	11.47	2.44	70	8.85	2.18	76	26.58	3.28	76	2.35	12
98	ZCML-205	46.62	78	2.10	0.74	42	9.12	2.21	80	16.78	2.82	84	9.58	2.26	79	28.79	3.36	79	0.93	96
81	MZLP-22	46.76	79	5.70	1.74	82	9.12	2.21	79	13.87	2.63	79	9.68	2.27	80	29.08	3.37	80	1.29	87
2	L9798-715	50.21	80	5.16	1.64	80	8.67	2.16	74	13.87	2.63	77	9.39	2.24	78	28.22	3.34	78	2.09	25
38	L9798-794	51.57	81	5.70	1.74	85	11.47	2.44	86	13.87	2.63	78	10.49	2.35	82	31.50	3.45	82	1.96	33
85	MZLP-28	53.23	82	2.92	1.07	68	11.47	2.44	88	16.78	2.82	81	10.70	2.37	83	32.14	3.47	83	1.42	78
82	MZLP-23	53.80	83	2.10	0.74	20	10.91	2.39	84	16.78	2.82	85	10.07	2.31	81	30.27	3.41	81	1.62	49
61	L9798-842	57.62	84	2.10	0.74	41	9.12	2.21	75	29.67	3.39	93	14.01	2.64	87	41.68	3.73	87	1.30	86

Table 3.3 (Cont.) Severity means of common rust and grain yield values of maize inbred lines evaluated for resistance at Greytown (1999/2000 season)

Entry	Code/Name[a]	AUDPC[b]	Rank[c]	Rating1[d]	LogTD[e]	Rank	Rating2	LogTD	Rank	Rating	LogTD	Rank	Mean[f]	LogTD	Rank	Sum[g]	LogTD	Rank	Y(t/ha)[h]	Rank
80	MZLP-21	62.09	85	7.17	1.97	93	10.91	2.39	83	16.78	2.82	82	11.82	2.47	84	35.52	3.57	84	0.64	99
101	PHB3394	67.40	86	9.12	2.21	97	13.87	2.63	90	16.61	2.81	80	13.46	2.60	85	40.45	3.70	85	ND	ND
52	L9798-824	67.65	87	4.10	1.41	71	9.12	2.21	76	34.12	3.53	97	16.28	2.79	92	48.42	3.88	92	1.66	53
34	L9798-784	67.84	88	5.70	1.74	86	11.47	2.44	85	29.67	3.39	94	15.80	2.76	90	46.99	3.85	90	1.21	90
42	L9798-805	67.84	89	5.70	1.74	89	11.47	2.44	89	29.67	3.39	95	15.80	2.76	89	46.99	3.85	89	1.57	64
56	L9798-830	69.87	90	4.10	1.41	78	16.61	2.81	95	20.09	3.00	86	13.74	2.62	86	41.26	3.72	86	1.43	74
40	L9798-798	76.95	91	7.17	1.97	90	16.61	2.81	93	20.29	3.01	87	15.03	2.71	88	45.15	3.81	88	1.54	66
60	L9798-837	83.20	92	2.92	1.07	67	13.87	2.63	91	32.14	3.47	96	17.99	2.89	94	54.05	3.99	94	1.40	80
78	MZLP-18	83.37	93	7.17	1.97	91	16.61	2.81	96	23.34	3.15	89	16.12	2.78	91	48.42	3.88	91	1.80	39
27	L9798-773	91.32	94	13.20	2.58	101	16.61	2.81	94	24.29	3.19	91	18.54	2.92	95	55.15	4.01	95	1.67	50
39	L9798-796	91.81	95	7.17	1.97	92	11.47	2.44	87	44.70	3.80	99	22.42	3.11	98	67.36	4.21	98	1.37	83
79	MZLP-19	101.42	96	9.49	2.25	98	15.96	2.77	92	23.34	3.15	88	16.78	2.82	93	50.40	3.92	93	1.49	70
6	L9798-728	102.54	97	8.67	2.16	95	20.29	3.01	98	29.67	3.39	92	20.09	3.00	97	60.34	4.10	97	1.67	51
3	L9798-724	104.46	98	8.67	2.16	94	24.29	3.19	100	24.29	3.19	90	19.11	2.95	96	57.40	4.05	96	2.23	15
41	L9798-801	123.64	99	10.91	2.39	99	20.29	3.01	99	36.97	3.61	98	23.10	3.14	99	69.41	4.24	99	1.28	88
8	L9798-733	136.05	100	11.47	2.44	100	20.09	3.00	97	66.02	4.19	100	32.79	3.49	100	98.49	4.59	100	1.01	94
51	L9798-819	199.59	101	9.12	2.21	96	35.87	3.58	101	95.58	4.56	101	47.47	3.86	101	142.59	4.96	101	0.47	100
Grand mean		37.72		2.83	1.04		5.75	1.75		8.33	2.12		5.93	1.78		17.81	2.88		1.74	
LSD (0.05)		23.45			0.60			0.59			0.69			0.50			0.51		0.54	
Significance		**			**			**			**			**			**		**	
CV (%)		46.08			42.47			24.99			24.03			20.89			13.21		22.79	

[a] L9798, lines from ARC-Grain Crops Institute, Potchefstroom, South Africa; MZLP, lines from INIA-National Agriculture Research Institute, Mozambique; ZCML, lines from CIMMYT- International Maize and Wheat Improvement Centre, Zimbabwe and PHB3394 susceptible hybrid (control).

[b] Area under disease progress curve from three weeks before anthesis to two weeks after anthesis.

[c] Ranking of genotypes according to the LSD values for each parameter analysed, where for severity low values represent resistant genotypes.

[d] Ratings of severity means assessed three weeks before anthesis, during anthesis, and two weeks after anthesis, respectively.

[e] LogTD: Transformed values using natural logarithm (LN).

[f] Mean rust severity for three ratings, measured as relative percentage leaf area infected in a 0-96% scale (Davis *et al*., 1988).

[g] Cumulative plant leaf area diseased (sum of severity means of three ratings).

[h] Shelled grain weight per plot adjusted to 12.5% grain moisture and converted to tons per hectare.

LSD ($P < 0.05$), CV (%) were calculated from logarithm values (LogTD) and AUDPC form percentage values.

** Highly significant ($P<0.05$)and * significant ($P<0.1$)

Table 3.4. Severity means of common rust on maize inbred lines evaluated for resistance at Ermelo (1999/2000 season)

Entry	Code/Name	UDPC[b]	Rank[c]	Rating1[d]	LogTD[e]	Rank	Rating2	LogTD	Rank	Rating3	LogTD	Rank	Mean[f]	LogTD	Rank	Sum[g]	LogTD	Rank
47	L9798-813	4.80	1	0.68	-0.39	1	0.68	-0.39	1	2.10	0.74	4	1.15	0.14	1	3.46	1.24	1
50	L9798-817	6.69	2	0.99	-0.01	13	0.99	-0.01	4	2.10	0.74	2	1.45	0.37	4	4.35	1.47	4
34	L9798-784	6.69	3	0.99	-0.01	10	0.99	-0.01	7	2.10	0.74	6	1.45	0.37	3	4.35	1.47	3
97	ZCML-202	7.17	4	0.68	-0.39	2	1.43	0.36	8	2.10	0.74	3	1.45	0.37	2	4.35	1.47	2
95	ZCML-393	7.19	5	0.68	-0.39	4	0.99	-0.01	6	2.92	1.07	10	1.65	0.50	5	4.90	1.59	5
35	L9798-788	8.82	6	2.10	0.74	67	1.43	0.36	18	1.43	0.36	1	1.72	0.54	6	5.16	1.64	6
64	L9798-850	9.03	7	0.97	-0.03	8	1.43	0.36	11	2.92	1.07	13	1.82	0.60	7	5.47	1.70	7
94	ZCML-392	9.30	8	2.10	0.74	58	1.43	0.36	9	2.10	0.74	5	1.93	0.66	8	5.81	1.76	8
96	ZCML-204	9.57	9	0.68	-0.39	3	1.43	0.36	10	4.10	1.41	21	2.23	0.80	14	6.62	1.89	13
31	L9798-780	9.57	10	0.68	-0.39	5	1.43	0.36	20	4.10	1.41	32	2.10	0.74	10	6.30	1.84	10
30	L9798-779	9.79	11	1.43	0.36	27	1.43	0.36	14	2.92	1.07	11	1.99	0.69	9	5.99	1.79	9
18	L9798-756	10.26	12	0.99	-0.01	15	2.10	0.74	40	2.92	1.07	16	2.14	0.76	11	6.42	1.86	11
36	L9798-790	10.40	13	2.01	0.70	46	0.99	-0.01	2	2.92	1.07	8	2.20	0.79	12	6.62	1.89	12
54	L9798-827	10.50	14	2.10	0.74	73	1.43	0.36	17	2.92	1.07	14	2.25	0.81	15	6.75	1.91	15
77	MZLP-11	11.46	15	0.99	-0.01	12	2.10	0.74	38	4.10	1.41	25	2.48	0.91	18	7.46	2.01	18
38	L9798-794	11.49	16	0.68	-0.39	7	1.43	0.36	13	5.16	1.64	44	2.72	1.00	28	8.08	2.09	28
39	L9798-796	11.59	17	2.01	0.70	47	1.43	0.36	12	2.92	1.07	12	2.29	0.83	16	6.89	1.93	16
9	L9798-736	11.68	18	2.10	0.74	68	2.10	0.74	48	2.92	1.07	15	2.44	0.89	17	7.32	1.99	17
10	L9798-737	11.73	19	1.43	0.36	19	0.99	-0.01	3	5.16	1.64	34	2.94	1.08	34	8.85	2.18	34
11	L9798-738	12.14	20	1.43	0.36	17	2.10	0.74	34	4.10	1.41	28	2.61	0.96	24	7.85	2.06	24
32	L9798-781	12.17	21	1.43	0.36	32	2.10	0.74	32	4.10	1.41	26	2.69	0.99	27	8.08	2.09	26
26	L9798-769	12.17	22	1.43	0.36	38	2.10	0.74	51	4.10	1.41	27	2.69	0.99	26	8.08	2.09	27
53	L9798-825	12.17	23	1.43	0.36	31	2.10	0.74	30	4.10	1.41	20	2.61	0.96	23	7.85	2.06	23
20	L9798-759	12.17	24	1.43	0.36	39	2.10	0.74	31	4.10	1.41	31	2.69	0.99	25	8.08	2.09	25
63	L9798-849	12.17	25	1.43	0.36	36	2.10	0.74	28	4.10	1.41	24	2.61	0.96	22	7.85	2.06	22
93	ZCML-390	12.21	26	0.99	-0.01	11	1.43	0.36	19	5.16	1.64	45	2.86	1.05	33	8.58	2.15	33
29	L9798-777	12.58	27	1.73	0.55	41	0.99	-0.01	5	2.92	1.07	9	2.48	0.91	21	7.46	2.01	21
25	L9798-768	12.79	28	1.43	0.36	26	2.01	0.70	25	2.92	1.07	17	2.20	0.79	13	6.62	1.89	14
68	L9798-856	12.88	29	2.10	0.74	59	2.10	0.74	46	4.10	1.41	22	2.83	1.04	30	8.50	2.14	30
17	L9798-752	12.88	30	2.10	0.74	82	2.10	0.74	53	4.10	1.41	23	2.83	1.04	32	8.50	2.14	32
22	L9798-762	12.88	31	2.10	0.74	80	2.10	0.74	29	4.10	1.41	29	2.83	1.04	31	8.50	2.14	31
71	L9798-863	13.97	32	1.43	0.36	35	2.92	1.07	64	2.92	1.07	7	2.48	0.91	19	7.46	2.01	19
16	L9798-750	14.08	33	2.10	0.74	50	2.10	0.74	36	5.70	1.74	50	3.29	1.19	37	9.87	2.29	37
4	L9798-720	14.08	34	2.10	0.74	51	2.10	0.74	55	5.70	1.74	47	3.29	1.19	38	9.87	2.29	38
58	L9798-833	14.10	35	1.43	0.36	37	2.10	0.74	44	5.16	1.64	43	3.03	1.11	35	9.12	2.21	35
24	L9798-765	14.71	36	1.43	0.36	16	2.01	0.70	23	3.71	1.31	18	2.48	0.91	20	7.46	2.01	20
1	L9798-714	14.81	37	2.10	0.74	76	2.10	0.74	50	5.16	1.64	37	3.32	1.20	40	9.87	2.29	40
28	L9798-774	14.81	38	2.10	0.74	74	2.10	0.74	49	5.16	1.64	41	3.32	1.20	41	9.87	2.29	41
60	L9798-837	14.81	39	2.10	0.74	56	2.10	0.74	42	5.16	1.64	39	3.32	1.20	39	9.87	2.29	39
21	L9798-760	15.30	40	4.10	1.41	99	1.43	0.36	16	4.10	1.41	30	3.46	1.24	43	10.38	2.34	43
7	L9798-731	15.90	41	2.01	0.70	45	2.10	0.74	45	5.16	1.64	42	3.67	1.30	45	10.91	2.39	45

Table 3.4 (Cont.) Severity means of common rust on maize inbred lines evaluated for resistance at Ermelo (1999/2000 season)

Entry	Code/Name	UDPC[b]	Rank[c]	Rating1[d]	LogTD[e]	Rank	Rating2	LogTD	Rank	Rating3	LogTD	Rank	Mean[f]	LogTD	Rank	Sum[g]	LogTD	Rank
55	L9798-829	16.40	42	2.53	0.93	88	1.43	0.36	21	3.71	1.31	19	2.72	1.00	29	8.17	2.10	29
42	L9798-805	16.73	43	2.10	0.74	60	2.10	0.74	35	6.49	1.87	52	3.86	1.35	47	11.59	2.45	47
13	L9798-745	16.86	44	2.53	0.93	89	2.10	0.74	39	4.10	1.41	33	3.25	1.18	36	9.78	2.28	36
65	L9798-852	16.88	45	0.68	-0.39	6	2.92	1.07	63	7.17	1.97	64	3.63	1.29	44	10.91	2.39	44
66	L9798-854	17.10	46	1.43	0.36	18	2.92	1.07	68	5.16	1.64	35	3.39	1.22	42	10.07	2.31	42
15	L9798-748	17.81	47	2.92	1.07	91	2.10	0.74	27	7.17	1.97	67	4.26	1.45	62	12.81	2.55	59
43	L9798-807	17.82	48	2.10	0.74	84	2.01	0.70	26	7.17	1.97	66	3.90	1.36	48	11.70	2.46	48
87	MZLP-34	17.93	49	2.10	0.74	66	2.10	0.74	47	9.12	2.21	79	4.48	1.50	65	13.46	2.60	64
100	ZCML-312	19.01	50	2.10	0.74	53	2.92	1.07	60	7.17	1.97	57	4.10	1.41	53	12.30	2.51	53
19	L9798-758	19.01	51	2.10	0.74	83	2.92	1.07	70	7.17	1.97	63	4.26	1.45	61	12.81	2.55	61
12	L9798-741	19.01	52	2.10	0.74	77	2.92	1.07	77	7.17	1.97	58	4.10	1.41	57	12.30	2.51	57
76	MZLP-05	19.01	53	2.10	0.74	52	2.92	1.07	66	7.17	1.97	59	4.10	1.41	56	12.30	2.51	56
86	MZLP-31	19.01	54	2.10	0.74	57	2.92	1.07	67	7.17	1.97	61	4.10	1.41	54	12.30	2.51	54
14	L9798-747	19.01	55	2.10	0.74	70	2.92	1.07	76	7.17	1.97	56	4.10	1.41	55	12.30	2.51	55
61	L9798-842	19.01	56	2.10	0.74	86	2.92	1.07	78	7.17	1.97	60	4.26	1.45	60	12.81	2.55	60
52	L9798-824	19.15	57	1.43	0.36	30	2.10	0.74	37	11.47	2.44	87	5.10	1.63	77	15.18	2.72	76
70	L9798-862	19.28	58	0.99	-0.01	14	2.01	0.70	22	8.67	2.16	72	3.97	1.38	50	11.82	2.47	50
56	L9798-830	19.73	59	2.10	0.74	49	2.92	1.07	72	6.49	1.87	53	4.14	1.42	58	12.43	2.52	58
88	MZLP-52	19.97	60	1.43	0.36	21	2.92	1.07	75	6.23	1.83	51	3.82	1.34	46	11.47	2.44	46
91	ZCML-387	20.08	61	2.10	0.74	64	4.10	1.41	86	5.70	1.74	46	4.06	1.40	52	12.18	2.50	52
57	L9798-832	20.08	62	2.10	0.74	75	4.10	1.41	93	5.70	1.74	49	4.06	1.40	51	12.18	2.50	51
92	ZCML-389	20.22	63	1.43	0.36	24	2.92	1.07	71	9.12	2.21	80	4.57	1.52	67	13.74	2.62	67
48	L9798-815	20.22	64	1.43	0.36	20	2.92	1.07	58	9.12	2.21	77	4.66	1.54	70	14.01	2.64	70
85	MZLP-28	20.82	65	2.80	1.03	90	2.10	0.74	43	9.12	2.21	78	5.16	1.64	80	15.49	2.74	80
99	ZCML-216	20.93	66	2.10	0.74	79	2.92	1.07	62	9.12	2.21	75	4.81	1.57	74	14.44	2.67	74
98	ZCML-205	21.30	67	1.43	0.36	23	4.10	1.41	92	7.17	1.97	55	4.57	1.52	66	13.60	2.61	66
37	L9798-791	21.88	68	2.10	0.74	65	2.92	1.07	65	8.67	2.16	73	4.66	1.54	69	14.01	2.64	69
72	L9798-865	21.88	69	2.10	0.74	55	2.92	1.07	74	8.67	2.16	70	4.90	1.59	75	14.73	2.69	75
27	L9798-773	22.01	70	2.10	0.74	62	4.10	1.41	90	7.17	1.97	62	4.71	1.55	73	14.15	2.65	73
81	MZLP-22	22.73	71	2.10	0.74	72	2.10	0.74	52	13.87	2.63	91	6.05	1.80	87	18.17	2.90	87
74	L9798-870	23.09	72	1.43	0.36	28	2.92	1.07	73	10.91	2.39	83	5.26	1.66	83	15.80	2.76	83
23	L9798-764	23.11	73	1.43	0.36	34	3.71	1.31	82	7.17	1.97	54	4.26	1.45	59	12.81	2.55	62
2	L9798-715	23.46	74	6.49	1.87	101	2.10	0.74	54	5.70	1.74	48	5.16	1.64	79	15.49	2.74	79
67	L9798-855	23.80	75	4.44	1.49	100	2.10	0.74	33	5.16	1.64	36	4.66	1.54	71	14.01	2.64	72
5	L9798-725	23.82	76	2.10	0.74	87	3.71	1.31	85	7.17	1.97	65	4.48	1.50	64	13.46	2.60	65
89	MZLP-67	23.86	77	1.43	0.36	40	2.53	0.93	57	9.12	2.21	76	4.71	1.55	72	14.01	2.64	71
45	L9798-810	23.93	78	2.10	0.74	69	4.10	1.41	89	9.12	2.21	81	5.16	1.64	78	15.49	2.74	78
59	L9798-730	24.44	79	2.10	0.74	81	3.53	1.26	80	5.16	1.64	38	3.94	1.37	49	11.82	2.47	49
84	MZLP-26	25.02	80	1.43	0.36	25	2.92	1.07	69	13.87	2.63	94	6.17	1.82	90	18.36	2.91	90
73	L9798-734	25.02	81	1.43	0.36	29	2.92	1.07	61	13.87	2.63	93	6.17	1.82	92	18.36	2.91	91

Table 3.4 (Cont.) Severity means of common rust on maize inbred lines evaluated for resistance at Ermelo (1999/2000 season)

Entry	Code/Name	UDPC[b]	Rank[c]	Rating1[d]	LogTD[e]	Rank	Rating2	LogTD	Rank	Rating3	LogTD	Rank	Mean[f]	LogTD	Rank	Sum[g]	LogTD	Rank
69	L9798-859	25.22	82	0.97	-0.03	9	3.71	1.31	84	8.67	2.16	71	4.62	1.53	68	13.87	2.63	68
33	L9798-782	25.25	83	1.43	0.36	33	2.01	0.70	24	10.49	2.35	82	5.10	1.63	76	15.33	2.73	77
46	L9798-812	25.60	84	2.92	1.07	97	2.92	1.07	59	10.91	2.39	84	6.05	1.80	85	17.99	2.89	85
49	L9798-816	25.62	85	2.10	0.74	78	5.16	1.64	96	5.16	1.64	40	4.26	1.45	63	12.81	2.55	63
6	L9798-728	25.71	86	2.10	0.74	71	3.71	1.31	83	9.03	2.20	74	5.21	1.65	82	15.64	2.75	82
79	MZLP-19	26.07	87	2.10	0.74	54	2.10	0.74	41	13.33	2.59	90	6.17	1.82	91	18.36	2.91	88
62	L9798-846	26.80	88	2.10	0.74	85	4.10	1.41	91	10.91	2.39	85	6.05	1.80	86	17.99	2.89	86
44	L9798-808	27.16	89	2.01	0.70	42	2.10	0.74	56	13.33	2.59	89	6.11	1.81	88	18.36	2.91	89
3	L9798-724	28.60	90	2.92	1.07	93	4.10	1.41	94	10.91	2.39	86	6.17	1.82	89	18.54	2.92	92
40	L9798-798	28.62	91	2.92	1.07	95	5.16	1.64	98	7.17	1.97	68	5.21	1.65	81	15.64	2.75	81
75	MZLP-02	32.40	92	2.01	0.70	43	1.43	0.36	15	15.33	2.73	96	7.46	2.01	94	22.42	3.11	94
90	ZCML-386	32.47	93	2.92	1.07	92	5.16	1.64	97	11.47	2.44	88	6.96	1.94	93	20.70	3.03	93
83	MZLP-25	34.54	94	2.05	0.72	48	4.44	1.49	95	7.85	2.06	69	5.37	1.68	84	16.12	2.78	84
101	PHB3349	35.20	95	4.10	1.41	98	4.10	1.41	88	16.61	2.81	97	8.67	2.16	97	26.05	3.26	97
80	MZLP-21	37.65	96	1.43	0.36	22	6.49	1.87	99	13.87	2.63	92	7.61	2.03	96	22.87	3.13	96
8	L9798-733	41.38	97	2.01	0.70	44	2.92	1.07	79	23.34	3.15	99	9.58	2.26	99	28.79	3.36	99
82	MZLP-23	42.11	98	2.92	1.07	96	3.71	1.31	81	15.33	2.73	95	7.46	2.01	95	22.42	3.11	95
78	MZLP-18	50.80	99	2.10	0.74	63	7.85	2.06	100	16.78	2.82	98	9.49	2.25	98	28.22	3.34	98
41	L9798-801	54.97	100	2.10	0.74	61	4.10	1.41	87	32.79	3.49	101	13.87	2.63	100	41.68	3.73	100
51	L9798-819	67.48	101	2.92	1.07	94	9.12	2.21	101	25.79	3.25	100	14.73	2.69	101	44.26	3.79	101
Grand Mean		19.68		1.76	0.57		2.39	0.87		6.15	1.82		3.76	1.32		11.28	2.42	
LSD (0.05)		16.19			0.78			0.82			0.85			0.66			0.66	
Significance		**			*			**			**			**			**	
CV (%)		60.90			102.60			70.23			34.78			36.79			20.09	

[a] L9798, lines from ARC-Grain Crops Institute, Potchefstroom, South Africa; MZLP, lines from INIA-National Agriculture Research Institute, Mozambique; ZCML, lines from CIMMYT- International Maize and Wheat Improvement Center, Zimbabwe and PHB3394 susceptible hybrid (control).

[b] Area under disease progress curve from three weeks before anthesis to two weeks after anthesis.

[c] Ranking of genotypes according to the LSD values for each parameter analysed, where low values represent resistant genotypes.

[d] Ratings of severity means assessed three weeks before anthesis, during anthesis, and two weeks after anthesis, respectively.

[e] LogTD: Transformed values using natural logarithm (LN).

[f] Mean rust severity for three ratings, measured as relative percentage leaf area infected in a 0-96% scale (Davis *et al*., 1988).

[g] Cumulative plant leaf area diseased (sum of severity means of three ratings).

LSD (P=0.05) and CV (%) were calculated from logarithmic values (LogTD) except for AUDPC.

** Highly significant (P<0.05) and * significant (P<0.1).

3.3). For the same assessment periods at Ermelo severity ranged between 1.48 to 6.4%, 1.48 and 9.12%, and between 1.43 and 32.79%.

Averaged over inbred lines, rust severity ranged from 1.23 to 47.47% and 1.15 to 14.73% at Greytown and Ermelo respectively, corresponding to AUDPC values of 5.51 to 199.59 for Greytown, and 4.8 to 67.48 for Ermelo (Tables 3.3 and 3.4). These ratings were generally of the same magnitude as reported in other field experiments, e. g. for sweet corn hybrids and open pollinated varieties evaluated for their reaction to common rust under artificial infection in the field over three years, where severity ranged between 0-46% and 0-8.5 (0-9 scale) (Pataky *et al.*, 1998). Gonzalez (2000) reported severity ranges between 5-35% on *Rp* inbred lines (R168-background) in Argentina. Previously, Pataky (1987b) reported variation in severity on sweet corn germplasm screened for resistance in Illinois. Ranges of 0-60%, 0-50% and 0-30% were observed in 1984, 1985 and 1986 respectively. The general means for severity and AUDPC for Greytown were 5.94% and 37.72, and 3.76% and 19.68 respectively, about two times higher than at Ermelo. For both localities the cumulative severity (sum of three ratings) also showed more significant differences ($P< 0.05$).

Means for genotype by environment interaction for common rust severity showed high significant differences between localities, inbred entries and locality by entry interaction, representing considerable differences in infection levels among lines between localities. These differences were observed in terms of disease incidence per se and the ranking of lines across localities. In general there was a high correlation between AUDPC, severity means (mean) and cumulative severity (sum) (r=0.98) and between mean and sum (r=1.00). The ranking of each parameter can be used for comparing genotypes across localities, and it is known that the disease damage at harvest is proportional to AUDPC (Van Rooyen, personal communication). In the present study, AUDPC values were used to rank genotypes. However, in the Greytown trial, AUDPC did

not significantly explain variation in yield (R^2=0.18) (Fig. 3.2A-F). A significant relationship was observed only for the third rating (R^2=0.24), mean and sum (R^2=0.21). According to the AUDPC values, the best genotypes for both localities were L9798-813 followed by ZCML-390, ZCML-393, L9798-750 and ZCML-204 (Table 3.5). The most susceptible line at both localities was L9798-819, followed by L9798-801, L9798-733 and MZLP-18 (Table 3.5).

Evaluated genotypes were distributed in classes of severity according to their level of resistance and susceptibility to the pathogen (Fig. 3.3). Many genotypes were in low groups of severity, representing higher potential of resistance between genotypes to common rust. Inbred lines that expressed reduced disease symptoms at all growth stages are potential sources of partial resistance. According to Pataky (1986) hybrids were considered as possessing partial resistance when they showed reduced numbers of uredia per leaf area at all growth stages. In the present study a considerable number of other lines appeared to be moderately resistant or moderately susceptible to common rust whereas several others were susceptible.

The high natural rust incidence that occurred at Petit in the 1999/2000 seasons resulted in high levels of rust severity on the 58 maize cultivars tested (Table 3.2). In the same trial at Greytown, rust incidence and severity were limited, possibly due to early planting. Thus, mean infection levels in the cultivars differed considerably between localities. At Petit rust severity ranged from 2.10 to 11.47% for the first rating, from 2.92 to 54.05% for the second rating, and from 2.92 to 55.15% for third rating. At Greytown, the single severity assessment after anthesis ranged between 1.43 to 19.49%. Averaged over hybrids, rust severity ranged from 2.69 to 40.04%. According to all disease assessments CRN 3891 was the most resistant hybrid at both sites. PHB 30H22 and NS 9100 were consistently ranked in the most rust resistant group whereas PAN 6414 and PAN 6480 also showed high levels of resistance. PHB 33A14, SNK 2975, LS

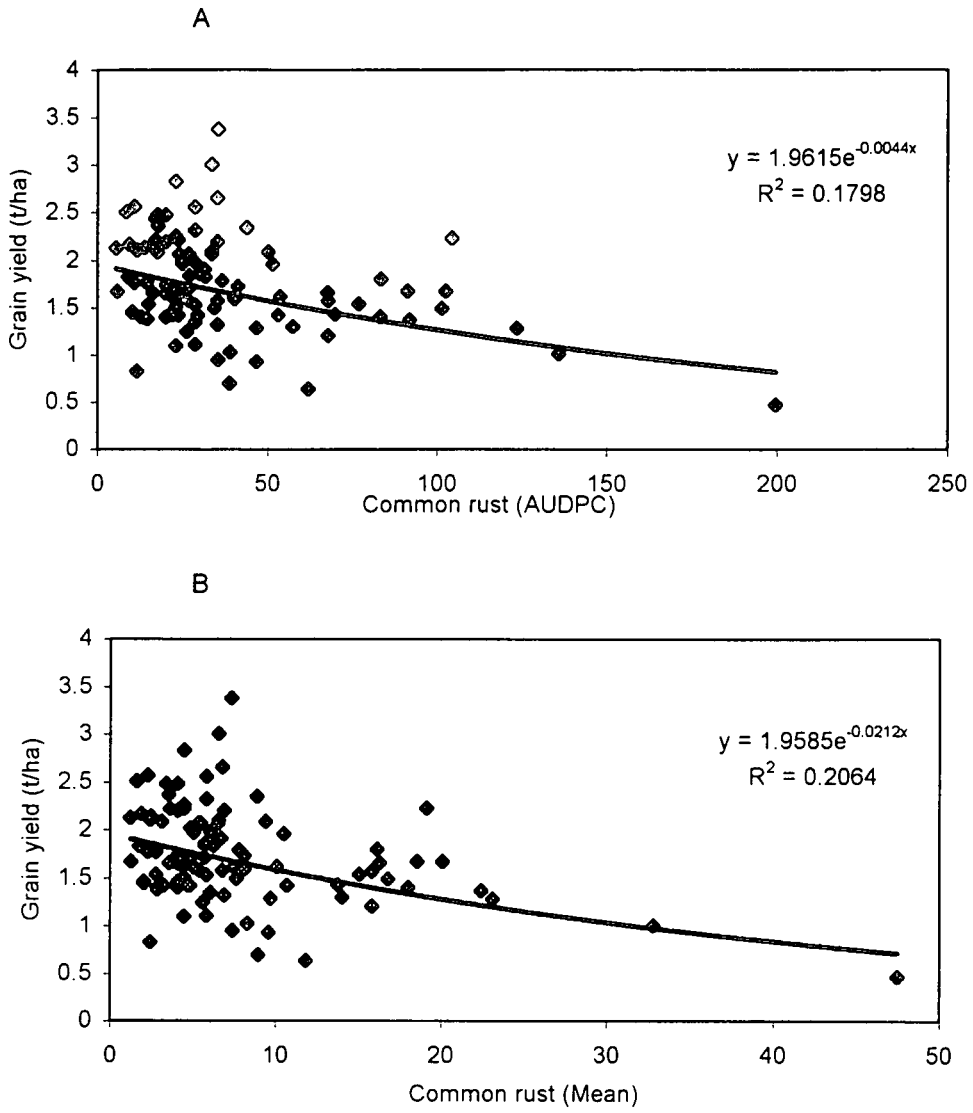


Fig. 3.2. The relationship between maize grain yield and AUDPC (A) and mean rust rating (B) at Greytown.

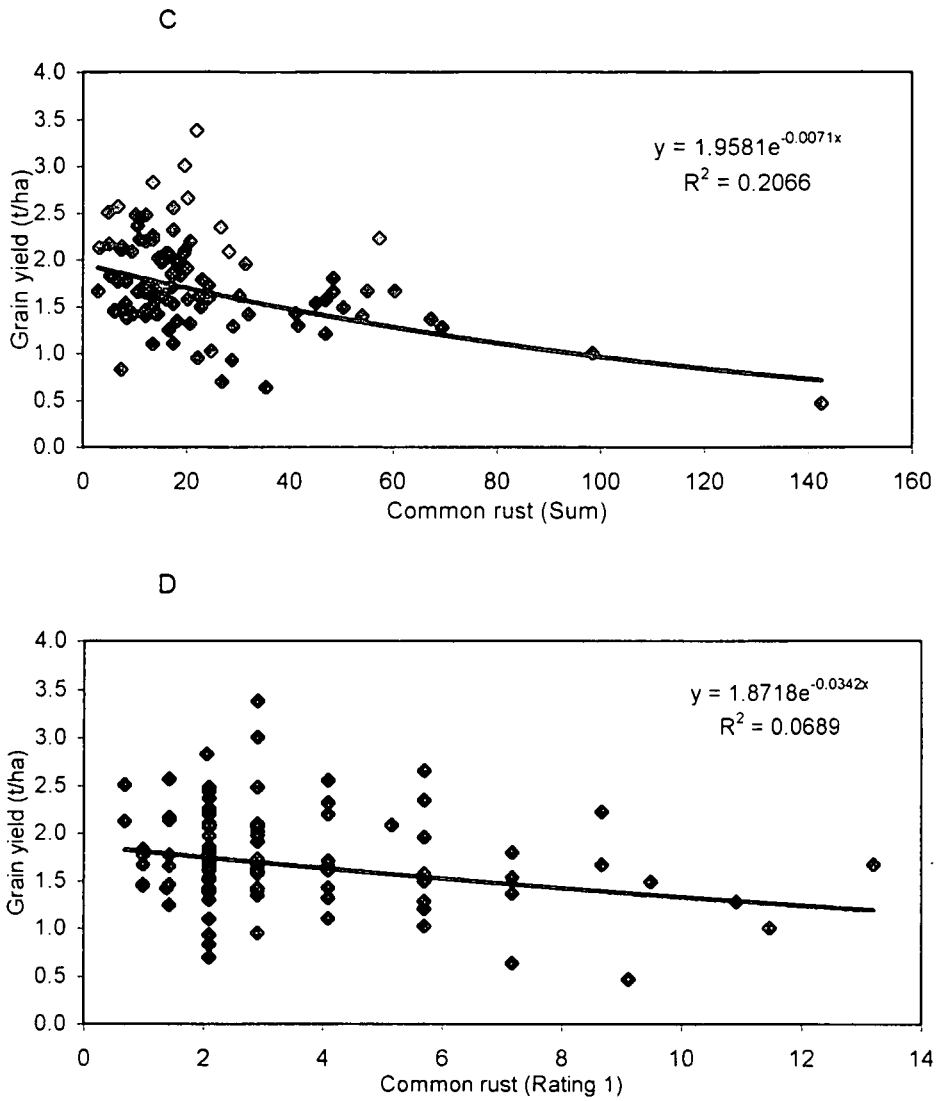


Fig. 3.2. The relationship between maize grain yield and cumulative (C) and first (D) rust ratings at Greytown.

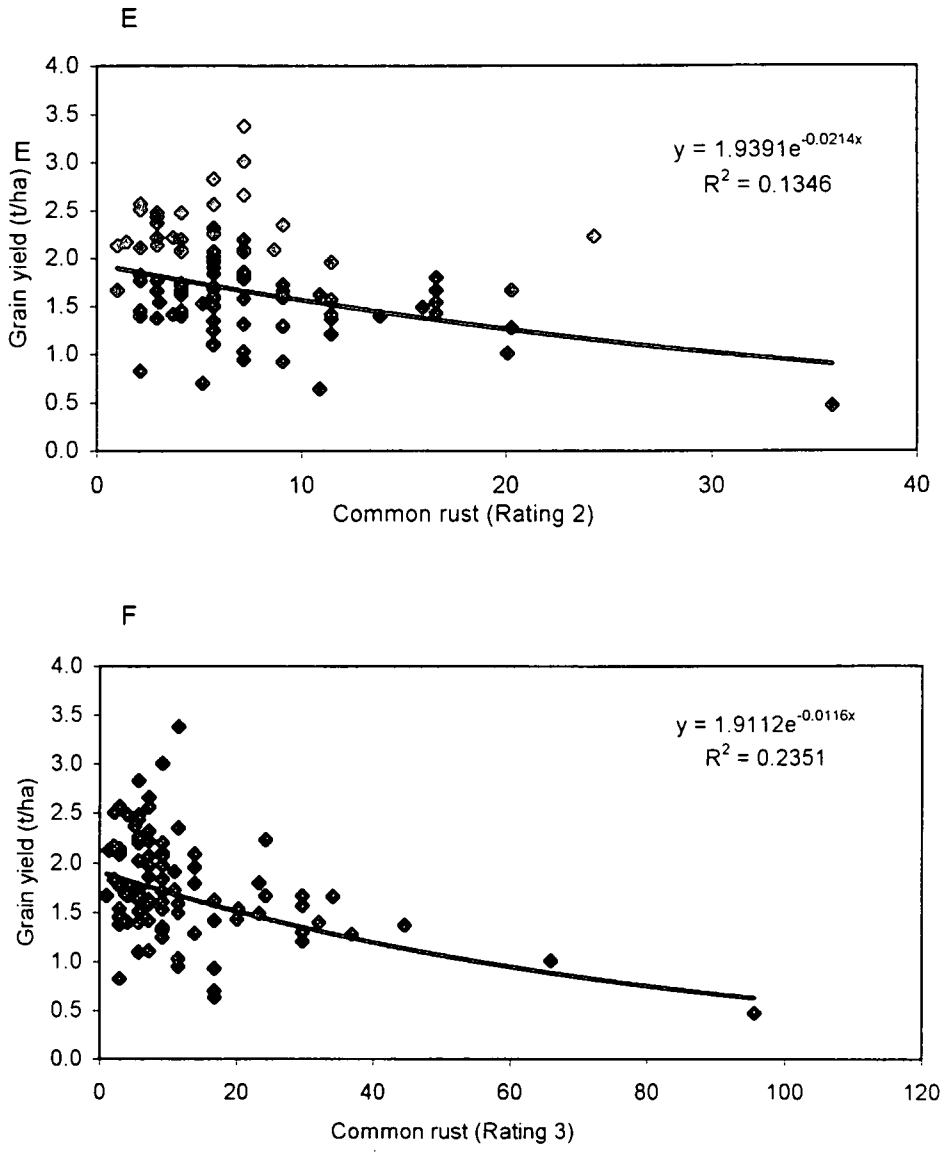


Fig. 3.2. The relationship between maize grain yield and the second (E) and third (F) rust ratings at Greytown.

Table 3.5. Genotype by environment interaction in common rust severity on inbred lines tested across Ermelo and Greytown (1999/2000 season)

Entry	Code/Name[a]	AUDPC[b]	Rank[c]	Mean[d]	LogTD[e]	Rank	Sum[f]	LogTD	Rank
47	L9798-813	6.58	1	1.37	0.32	1	4.12	1.42	1
93	ZCML-390	8.98	2	1.68	0.52	2	5.04	1.62	2
95	ZCML-393	9.44	3	2.00	0.69	4	6.00	1.79	4
16	L9798-750	9.80	4	1.89	0.64	3	5.67	1.74	3
96	ZCML-204	9.92	5	2.14	0.76	6	6.41	1.86	6
25	L9798-768	11.53	6	2.13	0.76	5	6.41	1.86	5
32	L9798-781	11.57	7	2.45	0.90	9	7.37	2.00	9
94	ZCML-392	11.64	8	2.19	0.78	8	6.57	1.88	8
29	L9798-777	12.13	9	2.46	0.90	10	7.40	2.00	10
21	L9798-760	12.18	10	2.48	0.91	11	7.45	2.01	11
10	L9798-737	12.31	11	2.89	1.06	16	8.65	2.16	16
26	L9798-769	13.32	12	2.74	1.01	15	8.22	2.11	15
100	ZCML-312	14.17	13	2.62	0.96	13	7.87	2.06	13
35	L9798-788	14.45	14	2.63	0.97	14	7.91	2.07	14
68	L9798-856	14.63	15	3.15	1.15	20	9.47	2.25	20
50	L9798-817	14.86	16	2.55	0.94	12	7.67	2.04	12
18	L9798-756	15.17	17	2.94	1.08	17	8.82	2.18	17
53	L9798-825	16.13	18	3.24	1.18	22	9.75	2.28	22
9	L9798-736	16.18	19	3.03	1.11	19	9.10	2.21	19
15	L9798-748	16.25	20	3.47	1.25	27	10.41	2.34	27
30	L9798-779	16.44	21	2.99	1.10	18	8.98	2.20	18
17	L9798-752	16.48	22	3.38	1.22	24	10.16	2.32	25
54	L9798-827	16.79	23	3.17	1.15	21	9.52	2.25	21
11	L9798-738	17.01	24	3.38	1.22	23	10.15	2.32	23
66	L9798-854	17.39	25	3.39	1.22	26	10.16	2.32	26
20	L9798-759	18.00	26	3.47	1.25	28	10.43	2.35	28
19	L9798-758	18.05	27	3.94	1.37	38	11.82	2.47	38
33	L9798-782	18.11	28	3.38	1.22	25	10.15	2.32	24
97	ZCML-202	18.32	29	2.16	0.77	7	6.49	1.87	7
37	L9798-791	18.41	30	3.57	1.27	33	10.73	2.37	33
36	L9798-790	18.61	31	3.47	1.25	29	10.43	2.35	29
22	L9798-762	18.88	32	3.71	1.31	35	11.13	2.41	35
31	L9798-780	19.15	33	3.55	1.27	32	10.68	2.37	32
24	L9798-765	19.32	34	3.64	1.29	34	10.94	2.39	34
71	L9798-863	19.49	35	3.54	1.27	31	10.64	2.37	31
55	L9798-829	19.74	36	3.49	1.25	30	10.49	2.35	30
91	ZCML-387	20.08	37	4.04	1.40	39	12.15	2.50	39
58	L9798-833	20.52	38	4.17	1.43	42	12.49	2.53	42
67	L9798-855	20.74	39	3.83	1.34	36	11.50	2.44	36
89	MZLP-67	20.84	40	4.08	1.41	40	12.21	2.50	40
1	L9798-714	20.87	41	4.34	1.47	43	13.03	2.57	43
99	ZCML-216	21.48	42	4.42	1.49	49	13.26	2.59	49
92	ZCML-389	21.65	43	4.53	1.51	51	13.57	2.61	51
65	L9798-852	21.85	44	4.41	1.48	47	13.22	2.58	47
57	L9798-832	21.88	45	4.43	1.49	50	13.30	2.59	50
72	L9798-865	21.95	46	4.82	1.57	58	14.47	2.67	58
48	L9798-815	22.02	47	4.79	1.57	57	14.40	2.67	57
13	L9798-745	22.74	48	4.34	1.47	44	13.04	2.57	44
63	L9798-849	22.86	49	4.12	1.42	41	12.37	2.52	41
12	L9798-741	23.81	50	4.88	1.59	59	14.66	2.69	59
76	MZLP-05	23.81	51	4.88	1.59	60	14.66	2.69	60
4	L9798-720	23.82	52	4.65	1.54	52	13.97	2.64	52
86	MZLP-31	23.87	53	4.98	1.61	61	14.95	2.71	61
69	L9798-859	23.92	54	4.36	1.47	46	13.09	2.57	46
59	L9798-730	24.13	55	4.36	1.47	45	13.09	2.57	45
70	L9798-862	24.55	56	4.75	1.56	54	14.25	2.66	54
74	L9798-870	24.66	57	5.42	1.69	68	16.25	2.79	68
64	L9798-850	24.66	58	3.85	1.35	37	11.55	2.45	37
28	L9798-774	25.08	59	4.77	1.56	55	14.32	2.66	55
7	L9798-731	25.69	61	5.18	1.65	64	15.53	2.74	64
83	MZLP-25	25.81	62	4.42	1.49	48	13.26	2.59	48

Table 3.5 (Cont.) Genotype by environment interaction in common rust severity on inbred lines tested across Ermelo and Greytown (1999/2000 season)

Entry	Code/Name[a]	AUDPC[b]	Rank[c]	Mean[d]	LogTD[e]	Rank	Sum[f]	LogTD	Rank
77	MZLP-11	25.14	60	4.71	1.55	53	14.13	2.65	53
14	L9798-747	26.28	63	5.20	1.65	65	15.61	2.75	65
43	L9798-807	26.53	64	5.14	1.64	63	15.46	2.74	63
46	L9798-812	27.18	65	5.92	1.78	71	17.73	2.88	71
23	L9798-764	27.43	66	5.14	1.64	62	15.46	2.74	62
84	MZLP-26	28.31	67	6.42	1.86	75	19.26	2.96	75
90	ZCML-386	28.74	68	5.92	1.78	72	17.76	2.88	72
5	L9798-725	29.53	69	5.51	1.71	69	16.56	2.81	69
87	MZLP-34	29.58	70	6.04	1.80	74	18.05	2.89	74
45	L9798-810	29.64	71	5.95	1.78	73	17.87	2.88	73
88	MZLP-52	30.07	72	5.32	1.67	67	15.99	2.77	67
49	L9798-816	30.55	73	5.63	1.73	70	16.91	2.83	70
38	L9798-794	31.53	74	5.32	1.67	66	15.96	2.77	66
62	L9798-846	31.67	75	6.83	1.92	77	20.45	3.02	77
73	L9798-734	32.05	76	7.11	1.96	79	21.33	3.06	79
75	MZLP-02	33.33	77	7.52	2.02	83	22.60	3.12	84
98	ZCML-205	33.96	78	6.62	1.89	76	19.83	2.99	76
81	MZLP-22	34.75	79	7.67	2.04	85	22.94	3.13	85
44	L9798-808	35.53	80	7.37	2.00	81	22.13	3.10	81
2	L9798-715	36.84	81	6.96	1.94	78	20.91	3.04	78
85	MZLP-28	37.03	82	7.44	2.01	82	22.31	3.11	82
34	L9798-784	37.27	83	4.79	1.57	56	14.34	2.66	56
61	L9798-842	38.32	84	7.71	2.04	87	23.06	3.14	86
42	L9798-805	42.29	85	7.82	2.06	88	23.34	3.15	88
52	L9798-824	43.40	86	9.09	2.21	91	27.17	3.30	91
56	L9798-830	44.80	87	7.54	2.02	84	22.58	3.12	83
82	MZLP-23	47.96	88	8.69	2.16	89	26.05	3.26	89
60	L9798-837	49.01	89	7.71	2.04	86	23.10	3.14	87
80	MZLP-21	49.87	90	9.49	2.25	93	28.45	3.35	93
101	PHB3349	51.30	91	10.78	2.38	96	32.39	3.48	96
39	L9798-796	51.70	92	7.15	1.97	80	21.50	3.07	80
40	L9798-798	52.79	93	8.83	2.18	90	26.52	3.28	90
27	L9798-773	56.67	94	9.35	2.24	92	27.99	3.33	92
79	MZLP-19	63.75	95	10.18	2.32	94	30.42	3.42	94
6	L9798-728	64.13	96	10.25	2.33	95	30.78	3.43	95
3	L9798-724	66.53	97	10.86	2.39	97	32.62	3.49	97
78	MZLP-18	67.09	98	12.34	2.51	98	37.04	3.61	98
8	L9798-733	88.72	99	17.76	2.88	99	53.25	3.98	99
41	L9798-801	89.31	100	17.90	2.89	100	53.79	3.99	100
51	L9798-819	133.54	101	26.44	3.28	101	79.44	4.38	101
Grand mean		28.70		4.71	1.55		16.34	2.65	
LSD (L)(0.05)		1.69			0.12			0.12	
LSD (E)(0.05)		11.95			0.35			0.35	
LSD (LxE)(0.05)		16.90			0.51			0.51	
Sign.(LxE)		**			**			**	
CV(LxE)(%)		51.50			28.20			16.50	

[a] L9798, lines from ARC-Potchefstroom, South Africa; MZLP, lines from INIA-Mozambique; ZCML, lines from CIMMYT-Zimbabwe and PHB3394 susceptible hybrid (control).

[b] Area under disease progress curve from three weeks before anthesis to two weeks after anthesis.

[c] Ranking of genotypes according to the LSD values for each parameter analysed.

[d] Mean rust severity for three ratings, measured as percentage leaf area infected (0-96% scale) (Davis *et al*., 1988)

[e] LogTD: Transformed values using natural logarithm (LN).

[f] Cumulative plant leaf area diseased (sum of severity means of three ratings).

LSD (P = 0.05) and CV (%) from logarithmic values (LogTD) of mean and sum and AUDPC values.

** Highly significant and (P<0.05) * significant (P<0.1)

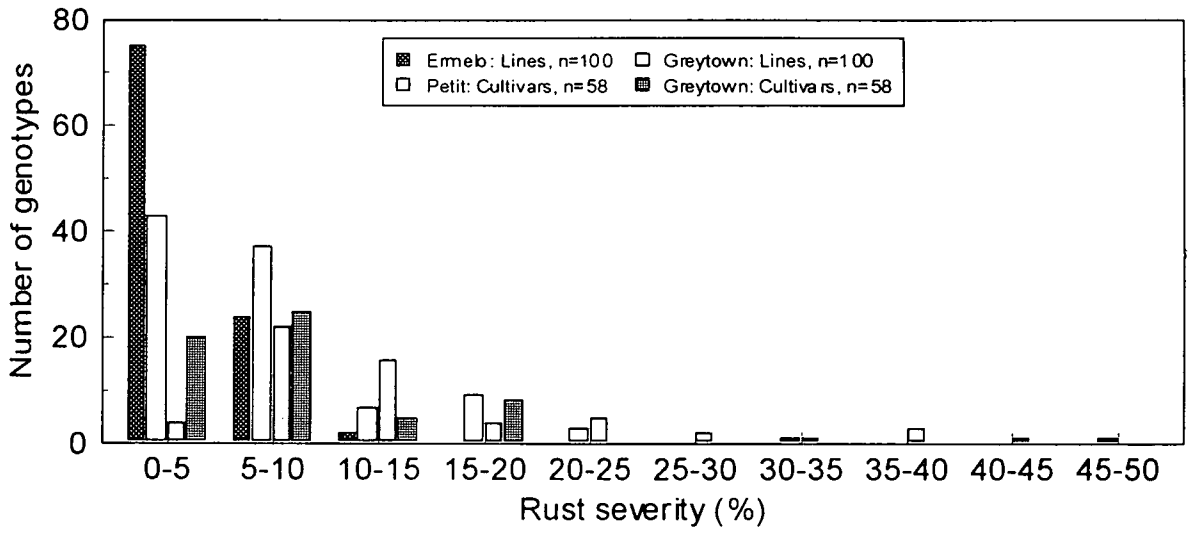


Fig. 3.3. Distribution of maize genotypes according to their level of resistance to common rust.

8503, SNK 2021, SNK 2147, CRN 3414, SNK 2778 and PAN 6256 were most susceptible across environments and ratings.

The objective of this study was a detailed screening of common rust resistance in maize germplasm originating from several sources. As little background information on the most suitable localities, inoculation techniques and disease assessment protocols were available, limitations in experimental procedure were expected. In general, the field screening techniques applied to inbred line evaluation were adequate. Epidemic levels of rust infection were achieved in experimental plots at both localities. The adverse weather conditions affected disease development at Ermelo and confirmed literature reports that environmental variation is strong in field evaluations. It was clear, however, that more emphasis should be placed on uniform distribution of inoculum, especially within a large experimental area as in the present study. More control over inoculum distribution will consequently reduce variation among blocks and therefore provide more meaningful differentiation among entries. Several inbred lines gave indications of useful resistance within and across localities (Fig. 3.3). However, these results should be confirmed at other localities and possibly also in artificial infection studies using individual pathotypes of *P. sorghi*. The results from Petit emphasised the lack of genetic resistance to common rust among several commercial cultivars used in South Africa. Not one of the tested genotypes was completely resistant. Identification of resistant genotypes, specifically with high levels of partial resistance, and their incorporation in elite commercial germplasm appears to be one of the most prudent future goals for maize breeders.

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SUMMARY

Common rust, caused *by Puccinia sorghi* Schw., is a major disease in maize (*Zea mays* L.) producing regions throughout the world and can result in high yield losses on susceptible genotypes when the environment is favourable for epidemic development.

To understand the principal aspects related to *P. sorghi*, a literature survey concerning the biology of the pathogen, host range, host-pathogen interaction, and economic importance of the disease, was conducted. Furthermore, different methods of disease management are reviewed, with emphasis on genetic control.

To determine pathogenic variability in *Puccinia sorghi* in South Africa, rust-infected maize leaves were collected during the 1999/2000 season. Isolates collected in the field were increased on susceptible plants and inoculated onto maize differential lines carrying different *Rp* genes for resistance to *P. sorghi.* Seven pathotypes, namely, A, B, C, D, E, F and G, were differentiated in the greenhouse. Pathotype B was the most virulent and occurred in Gauteng, KwaZulu-Natal, and Mpumalanga. Pathotype A, virulent only on $Rp3^A$ and $Rp3^B$, was widely distributed, occurring in six provinces. Pathotype C was detected in Mpumalanga and the Northern Province, D in the Northern Province whereas pathotype E was collected from North West, and F and G from KwaZulu-Natal. No virulence was detected for genes $Rp1^C$, $Rp1^G$, $Rp1^L$, $Rp3^D$ and $Rp3^F$. All isolates were virulent on $Rp3^B$. The occurrence of virulence for most *Rp* genes suggests that monogenic resistance is of little value for future protection of maize cultivars against common rust in South Africa.

One hundred maize inbred lines from Mozambique, South Africa and Zimbabwe, as well as 58 South African hybrids, were evaluated for resistance to common rust under field conditions over different localities in South Africa during the 1999/2000 season. Inbred lines were planted at Ermelo and Greytown and inoculated by a mixture of spores from previously identified *P. sorghi* pathotypes. Cultivars were tested under conditions of natural common rust infection at Greytown and Petit. Disease severity was assessed prior to anthesis, at anthesis,

and after anthesis at all localities except the Greytown cultivar trial, which was assessed only once after anthesis. Disease was scored on a 0-9 scale converted to percentage leaf diseased area (0-96% scale). The percentage values were log-transformed using the natural logarithm function. Analyses of variance were carried out for each severity parameter (individual rating, mean, sum and AUDPC [area under the disease progress curve]) within, and (mean, sum and AUDPC) across localities. Significant genetic variation existed between genotypes for resistance to *P. sorghi* at each and across localities. Some inbred lines showed significant levels of partial resistance to the pathogen and may be used in resistance breeding programmes. Most hybrids showed susceptible reaction types to common rust and are thus potentially vulnerable to yield losses under conditions of epidemic occurrence of P. *sorghi.*

In general, similar to other cereal rust pathosystems, it is apparent that monogenic rust resistance in maize will not be durable. Emphasis in breeding should therefore be placed on horizontal resistance, with particular attention to sources of resistance, screening procedures and heritability. This study will hopefully serve as an important source of information for future research of common maize rust in southern Africa.

OPSOMMING

Roes, veroorsaak deur *Puccinia sorghi* Schw., is 'n belangrike siekte van mielies (*Zea mays* L.) wêreldwyd en kan betekenisvolle oesverliese veroorsaak onder omgewingstoestande gunstig vir epidemiese ontwikkeling.

Ten einde die belangrikste aspekte met betrekking tot die biologie van patogeen, gasheerreeks, gasheer-patogeen interaksies en ekonomiese belang te verstaan, is `n literatuuroorsig saamgestel. Verskillende metodes van siektebeheer, met spesiale klem op genetiese beheer, is ook ingesluit.

Patogeniese variasie in *P. sorghi* in Suid-Afrika is gedurende die 1999/2000 seisoen bestudeer. Mielieblare geïnfekteer met roes is in die veld versamel en roeskulture is op vatbare plante in 'n glashuis vermeerder. Isolate is gesuiwer en vervolgens op roes-differensiërende lyne, elk met 'n bepaalde *Rp*-geen vir bestandheid, geïnokuleer. Sewe patotipes, nl. A, B, C, D, E, F en G, is geïdentifiseer. Patotipe B was mees virulent en is in Gauteng, KwaZulu-Natal en Mpumalanga waargeneem. Patotipe A, slegs virulent op $Rp3^A$ en $Rp3^B$, was wydverspreid in ses provinsies. Patotipe E het in Noordwes voorgekom terwyl F en G in KwaZulu-Natal, C in Mpumalanga en die Noordelike Provinsie, en D in die Noordelike Provinsie, onderskeidelik, versamel is. Geen virulensie is vir die gene $Rp1^C$, $Rp1^G$, $Rp1^L$, $Rp3^D$ en $Rp3^F$ waargeneem nie. Alle isolate was virulent op $Rp3^B$. Die voorkoms van virulensie vir meeste *Rp*-gene dui aan dat monogeniese weerstand van min waarde in die toekomstige beskerming van mieliekultivars teen roes in Suid-Afrika sal wees.

Eenhonderd ingeteelde mielielyne vanaf Mosambiek, Suid-Afrika en Zimbabwe, en 58 Suid-Afrikaanse kultivars, is vir bestandheid teen roes onder veldtoestande gedurende 1999/2000 geëvalueer. Die lyne is op Ermelo en Greytown geplant en met 'n mengsel van spore van vooraf-bepaalde patotipes van *P. sorghi* geïnokuleer. Die kultivars is onder toestande van natuurlike infeksie op Greytown en Petit getoets. Siektegraad is voor, tydens, en na antese bepaal, behalwe vir die kultivarproef te Greytown waar roesinfeksie slegs eenmalig na antese beraam is. Siektegraad is gemeet op `n 0-9 skaal waarna die waardes

omgeskakel is na `n persentasieskaal (0-96%). Die persentasie-waardes is vervolgens getransformeer na natuurlike logaritmes. Analise van variansie is uitgevoer op elke siekteparameter (individuele raming, gemiddelde raming, som van ramings en AOSVK [area onder die siektevorderingskurwe]) binne, en (gemiddeld, som en AOSVK) oor lokaliteite. Betekenisvolle genetiese variasie vir reaksie teenoor *P. sorghi* infeksie is waargeneem by elke lokaliteit, maar ook oor lokaliteite. Sekere ingeteelde lyne het hoë vlakke van gedeeltelike weerstand teen roes getoon en kan in weerstandstelingsprogramme gebruik word. Die meeste basters was egter vatbaar, wat aandui dat hulle potensieel gevoelig is vir oesverliese tydens toestande gunstig vir epidemiese ontwikkeling van mielieroes.

Soortgelyk aan ander graanroes-patosisteme is dit duidelik dat monogeniese weerstand in mielies nie volhoubaar teen roes sal wees nie. Die klem in weerstandsteling moet dus val op horisontale weerstand, met spesifieke verwysing na toepaslike bronne, evaluasieprosedures en erflikheid. Hierdie studie sal hopelik dien as `n belangrike bron van inligting vir toekomstige navorsing op mielieroes in suidelike Afrika.