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# On the relation between weather variables and sorghum ergot infection

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*Abstract*. Sorghum ergot (*Claviceps africana*) has had a significant impact on seed production and breeders' nurseries in Australia since it was first found in 1996. In this paper, 3 distinct key development stages of sorghum that are related to ergot infection were identified: flag leaf stage, pollen starch accumulation stage, and flowering period. Relationships between weather variables during these 3 stages and ergot severity as well as pollen viability were analysed using observed data from 2 field trials, a serial planting trial and a genotype trial, conducted at Gatton, Queensland. The duration of the flag leaf stage and of the flowering period was estimated from thermal time. An infection factor was introduced and calculated based on hourly temperature during the flowering period. This infection factor and the mean relative humidity at 0900 hours during the flowering period were the main factors influencing ergot infection. Mean daily minimum temperature during flag leaf stage also had a significant effect on ergot severity, although no significant relation was found between this mean daily minimum temperature and pollen viability. A linear regression model using the above 3 factors accounted for 94% of the environmentally caused variation in ergot severity observed in the genotype trial.

*Additional keywords*: *Claviceps africana*, *Sorghum bicolor*, pollen viability, development stage, climate, model.

## Introduction

Sorghum ergot, a disease caused by the fungus *Claviceps africana* (Frederickson *et al.* 1991), was first recorded in Australia in April 1996 (Ryley *et al*. 1996). It had been found in all sorghum regions of Australia, except seed production areas in southern New South Wales and the Ord River until 1997 (Ryley and Henzell 1999), but it has recently been identified in both regions (M. Ryley, unpubl. data). The disease has had a significant impact on seed production and breeders' nurseries in Queensland, necessitating regular applications of fungicides (Ryley 1998). In commercial grain production, direct yield losses have been minimal to date, but additional costs associated with harvesting problems due to honeydew on tillers have had a significant impact in some crops. Several cases of loss of milk production in sows, resulting in piglet death, and reduced milk production in dairy cattle have been demonstrated to be due to ergot-contaminated sorghum grain (Blaney *et al*. 1999*a*, 1999*b*).

Infection of sorghum by ergot occurs at anthesis when conidia of the fungus germinate on stigmas and the hyphae grow down the styles into the developing ovaries (Frederickson *et al.* 1991; Bandyopadhyay *et al.* 1998). Environmental conditions influence infection and disease development by their direct effects on the pathogen and through their effects on pollen production and fertilisation. The symptoms, causal organisms, biology, toxicity, and geographical distribution of ergot have been reviewed by Bandyopadhyay *et al*. (1998).

Fertilised ovaries are resistant to infection by ergot (Bandyopadhyay *et al*. 1998). Fertilisation is largely controlled by the amount of viable pollen, and by stigma receptivity. Adequate viable pollen combined with receptive stigmas results in extremely low infection rates even if ergot spores are abundant. There are genetic differences in viable pollen production between genotypes, while little is known about genetic differences in stigma receptivity.


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Three distinct crop physiological development stages are essential for pollen production and ergot infection (Fig. 1). The first stage is flag leaf stage. Low night temperature <13°C during flag leaf stage reduces pollen viability and may cause male sterility (Downes and Marshall 1971; Brooking 1976). According to Brooking (1976), the most sensitive physiological stage to low temperature is the late archesporial cell–pollen mother cell development period during the leptotene stage of meiosis. Leptotene of male meiosis commences in the apical florets as the flag leaf sheath emerges. Hence, low temperatures from flag leaf emergence to flag leaf fully expanded (flag leaf collar visible) can result in reduced pollen viability. The second stage is the pollen starch accumulation period. The pollen maturation phase of starch and proline accumulation takes place 4–5 days before anthesis (Brooking 1976). Unfavourable conditions during this period may affect the accumulation process and reduce the viability of pollen. The third stage is the flowering stage. Florets are potentially susceptible to infection during the period from the start to the end of flowering. According to Rosenthal and Gerik (1989) and Hammer *et al.* (1989) the flowering period can range from 4 to 12 days, depending on temperature and genotype.

Options for management of sorghum ergot are limited because there is no genetic resistance to the disease, and fungicides are economical only for seed production and parent seed blocks (Meinke and Ryley 1997). Management options, such as adjustment in planting dates to avoid the weather conditions favouring infection, and the use of agronomic practices that ensure uniform flowering, can minimise the impact of the disease (Ryley 1997). However, to manage the disease more effectively there is a need to better understand the underlying physiological processes involved.

It is particularly important to quantify the relationships between ergot infection and environmental factors more accurately. McLaren and Wehner (1990) analysed the relationship between infections caused by *Sphacelia sorghi* (*Claviceps africana*) and weather variables and found that the mean daily maximum temperature 1–5 days after commencement of pollen shed was highly correlated with disease incidence. They also found that mean night temperatures of <12°C, 3–4 weeks prior to flowering, significantly reduced pollen viability and increased susceptibility to infection (McLaren and Wehner 1992). Changes in ergot severity with flowering dates were closely correlated with differences in pollen viability (McLaren 1997). In order to quantify the

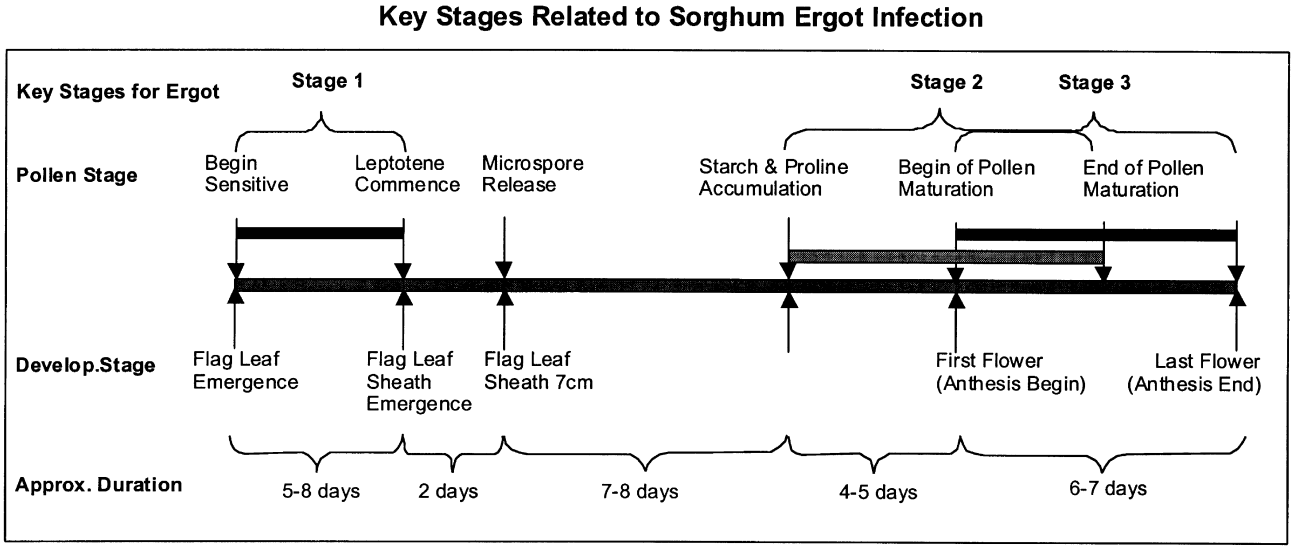


**Fig. 1.** Development stages of sorghum from flag leaf emergence to the end of flowering, and the essential development stages related to ergot infection. The approximate duration in days and the pollen development stages are also shown. The upper part of this figure was constructed based on Brooking (1976). The lower part indicates the key weather factor for ergot infection in each essential stage.

relationship between ergot potential and weather variables in South Africa, McLaren and Flett (1998) developed a regression model using the mean minimum temperature 23–27 days before flowering, and the mean daily maximum temperature and mean daily maximum relative humidity 1–5 days after flowering (±10% pollen shed). A preliminary climatic analysis using a rule-based method that incorporates these responses demonstrated the potential of using such information in crop management (Meinke and Ryley 1997). In the above models, a fixed duration of the sensitive stages was used. In fact, the occurrence and the duration of the stages sensitive to low day/night temperatures, as well as that of the flowering period, change with environmental factors, particularly temperature. The applicability of the responses to changes in key environmental factors and their specific thresholds need to be quantified for Australian conditions.

This study addresses the above-mentioned issue and reports findings obtained from experiments conducted near Gatton, Queensland, where the first Australian ergot outbreak was observed in 1996 (Ryley *et al*. 1996). In this paper we report on the physiologically based relations between sorghum growth and development, ergot infection, and weather conditions. The findings have been incorporated into a simple phenomenological model that builds on the original analysis by Meinke and Ryley (1997).

## Materials and methods

### *Experimental design*

#### *Serial planting trial*

In the serial planting trial, 3 sorghum genotypes (2 commonly grown hybrids designated here as variety A and B and an unnamed male sterile line) were planted near Gatton (27°55′S, 152°33′E) at 14-day intervals from 5 September 1997 to 3 March 1998, totalling 13 plantings. At each planting, 8 rows, each 5 m long and with a 0.4-m row spacing, of each genotype were planted with the male sterile line between the 2 hybrids. Panicles of the male sterile line in the first planting were inoculated with a suspension of macroconidia and secondary conidia at $1 \times 10^5$ conidia/mL when >70% of panicles had emerged stigmas. No further inoculations were done.

#### *Genotype trial*

Forty-eight genotypes were grown in a randomised block with 3 replicates and 2 planting dates: 2 March and 9 March 1998. The genotypes were selected in the Queensland Department of Primary Industries' sorghum breeding program on the basis of their variation in pollen production under low temperatures. Five hybrid cultivars, varying in pollen production, were also included. Each panicle was inoculated as outlined below.

### *Observation and data collection*

#### *Serial planting trial*

When at least 100 panicles in a planting had reached 10% anthesis (estimated by regular visual assessment), the pollen from 5 panicles was collected between 0900 and 1100 hours on 1.5% water agar in 90-mm plastic Petri dishes. A suspension of 2.5 g/L of iodine in water ethanol solution (7 : 3) was sprayed on to the surface of the water agar, and the dishes stored under refrigeration (9.5°C ± 0.5°C) for up to 1 week. Previous experiments indicated that the percentage viability of pollen did not change significantly over the period of storage. The dishes were examined under a compound microscope using transmitted light and the percentage of viable pollen grains (stained black–dark blue) was determined. One hundred pollen grains were counted on each dish, with 2 dishes per assessment. The flag leaf of each plant on which a panicle was flowering was marked with enamel paint. The percent florets with ergot infection was visually estimated on the marked plants.

#### *Genotype trial*

In each plot the quantity of pollen on each of 5 panicles with >10% flowering florets was rated visually (1, large cloud of pollen; 9, no pollen cloud) between 0900 and 1100 hours as the 'volume and density' of the pollen cloud resulting from flicking the panicle. On the second day of flowering, pollen from these panicles was collected on a single water agar plate, and the viability of the pollen was determined as described above. One or 2 days later, similar collections were made on 5 different panicles in each plot. The plants were marked with enamel paint as described above.

After pollen collection the panicles were inoculated with approximately 10 mL of a suspension of $1 \times 10^5$ macroconidia and secondary conidia/mL water. The severity of ergot infection on the selected panicles was visually rated 20–23 days after inoculation.

### *Simulation of the critical growth stages*

The end of the flag leaf stage for each planting was estimated using the APSIM sorghum model based on the recorded flowering date (Hammer and Muchow 1994). The starting date of flag leaf stage was simulated by assuming that the flag leaf begins to emerge when the third leaf prior to flag leaf is fully expanded (G. L. Hammer, pers. comm.), and that each of the last 3 leaves needs 20 degree-days to fully expand. Anthesis begins 100 degree-days after the full expansion of flag leaf. Duration of anthesis was calculated using a thermal time value of 128 degree-days with a base temperature of 3.2°C, based on the original observation data of Hammer *et al.* (1989). The pollen starch accumulation period is from 4–5 days prior to the first floret flowering to 4–5 days before the last floret flowering. Only the period prior to the pollen collection date was investigated because we collected pollen only once from each panicle during the flowering period.

### *Weather variables and calculation*

The weather data were recorded at a standard Bureau of Meteorology weather station located 100 m from the trials. Weather variables included daily maximum and minimum temperature, relative humidity at 0900 hours, and daily total rainfall. Mean values of daily maximum, minimum, and average temperatures, relative humidity at 0900 hours, and total rainfall during the flowering period were calculated. For the flag leaf period, mean values of daily minimum temperatures as well as the number of days with minimum temperature lower than 13°C were calculated. For each pollen collection date, mean maximum and minimum temperatures 1–5 days prior to collection date were calculated.

In order to increase the accuracy of thermal time calculation, hourly temperatures were simulated based on the method proposed by Goudriaan and van Laar (1994). The thermal time needed for predicting the flowering period and flag leaf stage was then estimated from these hourly temperatures. Hourly temperatures were also used to identify that part of each day during the flowering period when temperatures were favourable for infection.

### *Correlation analysis and the regression model*

Based on the physiological understanding of the infection cycle, correlation coefficients were calculated between pollen viability, ergot severity, and the mean/total values of weather variables during the critical developmental stages. The correlations between pollen viability and

mean daily minimum temperature during the flag leaf stage, and temperatures during the pollen starch accumulation stage, were investigated. For ergot severity, correlations with all the calculated variables (mean values of temperatures, relative humidity, and the total rainfall) were analysed.

Regression analyses were performed to determine the relationships between ergot severity and the weather variables during the critical periods. A regression model was established using the most significant factors ($P < 0.05$).

**Table 1. Correlations between mean ergot severity and the mean values of daily minimum temperature in flag leaf stage ($T_{flm}$), maximum, minimum, and average temperature, infection factor, relative humidity, and rainfall in the flowering period**

The last line shows the correlation between pollen viability and mean daily minimum temperature in the flag leaf stage in both trials
Infection factor ranges from 0.0 to 1.0, and describes the relative effect of temperature on ergot infection. Significant correlations in **bold**

| Variable | Genotype trial $n = 17$ | | Serial planting trial $n = 11$[A] | |
|---|---|---|---|---|
| | *R* | *P* | *r* | *P* |
| Flag leaf stage $T_{flm}$ | 0.36 | >0.100 | –0.46 | >0.100 |
| Maximum temperature | 0.03 | >0.100 | **–0.75** | **<0.010** |
| Minimum temperature | **0.83** | **<0.001** | **–0.55** | **<0.050** |
| Average temperature | **0.85** | **<0.001** | **–0.72** | **<0.010** |
| Infection factor | **0.86** | **<0.001** | **0.65** | **<0.010** |
| Relative humidity | **0.83** | **<0.001** | 0.40 | >0.100 |
| Rainfall | **0.80** | **<0.010** | 0.03 | >0.100 |
| $T_{flm}$ *v*. pollen viability | 0.41 | >0.100 | 0.42 | >0.100 |

[A] One data set missing in the serial planting trial.

## Results and discussion

### *Flag leaf stage minimum temperature, pollen viability, and ergot severity*

In both the serial planting and genotype trials the mean daily minimum temperatures during the flag leaf stage for most flowering dates were higher than 13°C, the threshold temperature identified by Downes and Marshall (1971) below which pollen viability is affected. The number of days with daily minimum temperature <13°C in the flag leaf stage was less than the threshold of 5 days (Downes and Marshall 1971) for all flowering dates in the serial planting trial and 5 days only for 2 flowering dates in the genotype trial. Accordingly, there was no significant relationship between pollen viability and the mean daily minimum temperature during flag leaf period (TMFLG) (Table 1, Fig. 2). There was a slightly increasing trend in pollen viability with increasing

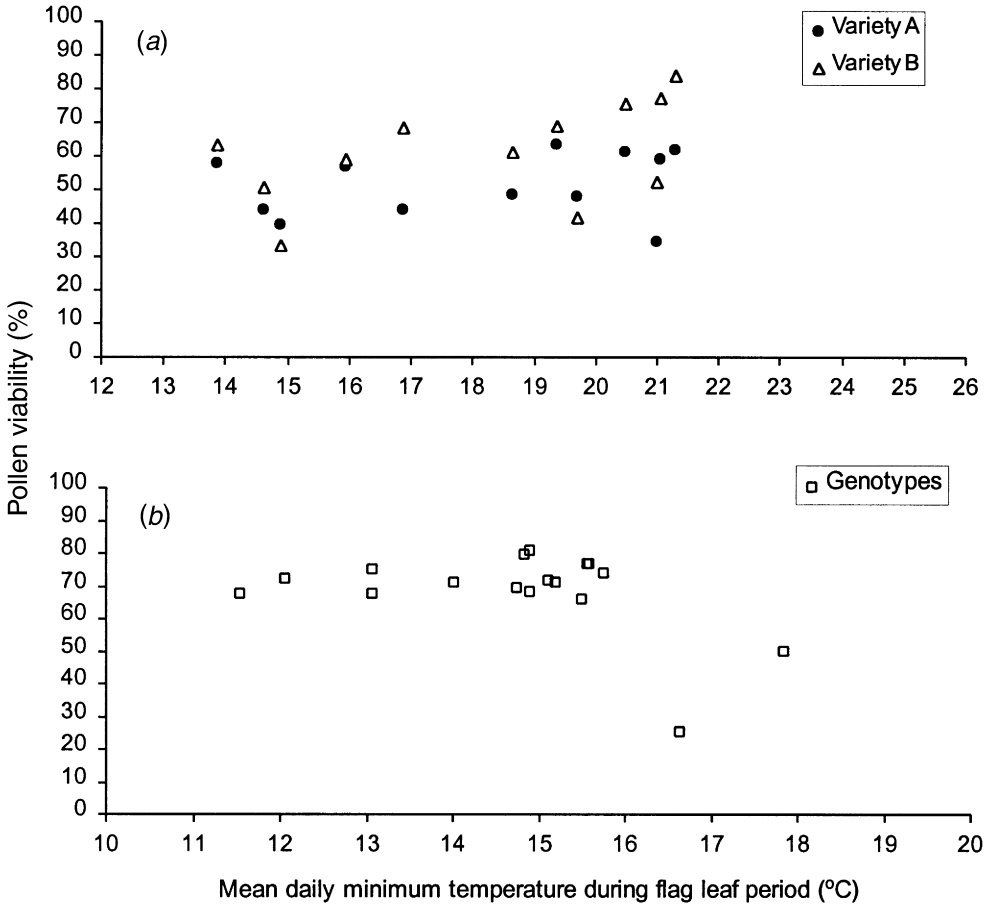


**Fig. 2.** Relationship between pollen viability and the mean daily minimum temperature during the flag leaf stage in the (*a*) serial planting trial and (*b*) genotype trial.

TMFLG in the serial planting data (Fig. 2*a*). Ergot severity did not correlate with this mean minimum temperature (Table 1), but there appears to be a non-linear relationship with a breakdown occurring at approximately 15°C in the serial planting trial (Fig. 3*a*). A similar relationship was found between pollen viability and ergot severity; that is, ergot severity increased sharply with decreasing pollen viability below 55–60% (Fig. 4). The highest ergot severity in the serial planting trial was associated with the lowest TMFLG and the lowest pollen viability. The complete scatter in the genotype data could be attributed to differential interactions between individual genotypes and temperature. Brooking (1979) and McLaren (1997) found that sorghum genotypes differed in their ability to tolerate low night temperatures during the pre-flowering period. The critical temperature during this period varies with genotypes. Further study is necessary to investigate the genotype effect on sensitivity to low night temperatures.

*Pollen starch accumulation stage and pollen viability*

We investigated the relationship between pollen viability and the mean value of daily maximum, minimum, and average temperatures 1–5 days prior to pollen collection. In the serial planting trial, mean temperature had a larger effect than minimum and maximum temperature, and the mean temperature over the 24 h before collection gave the most significant relationship (Fig. 5*a*). When the mean temperature over 5 days prior to pollen collection was plotted against pollen viability, the points became scattered, especially at temperatures above 24°C (Fig. 5*b*). Pollen viability decreased with decreasing temperature below 23–24°C. At temperatures above 24°C there was a trend of decreased pollen viability with increasing temperatures, although the points were scattered (Fig. 5*a*). Dhopte (1984) also found that pollen fertility was significantly reduced by 50% at night temperatures both below and above an optimum 23°C during the floret differentiation stage. In the genotype trial, no relation was found between pollen viability and temperatures during the starch accumulation stage, perhaps due to strong genotype effects.

Brooking (1976) also investigated the effect of temperatures 5 days prior to anthesis on pollen proline levels and final seed set. He found that night temperatures of 10°C and above during this period had no effect on pollen proline level and final seed set. Compared with the proline levels in his

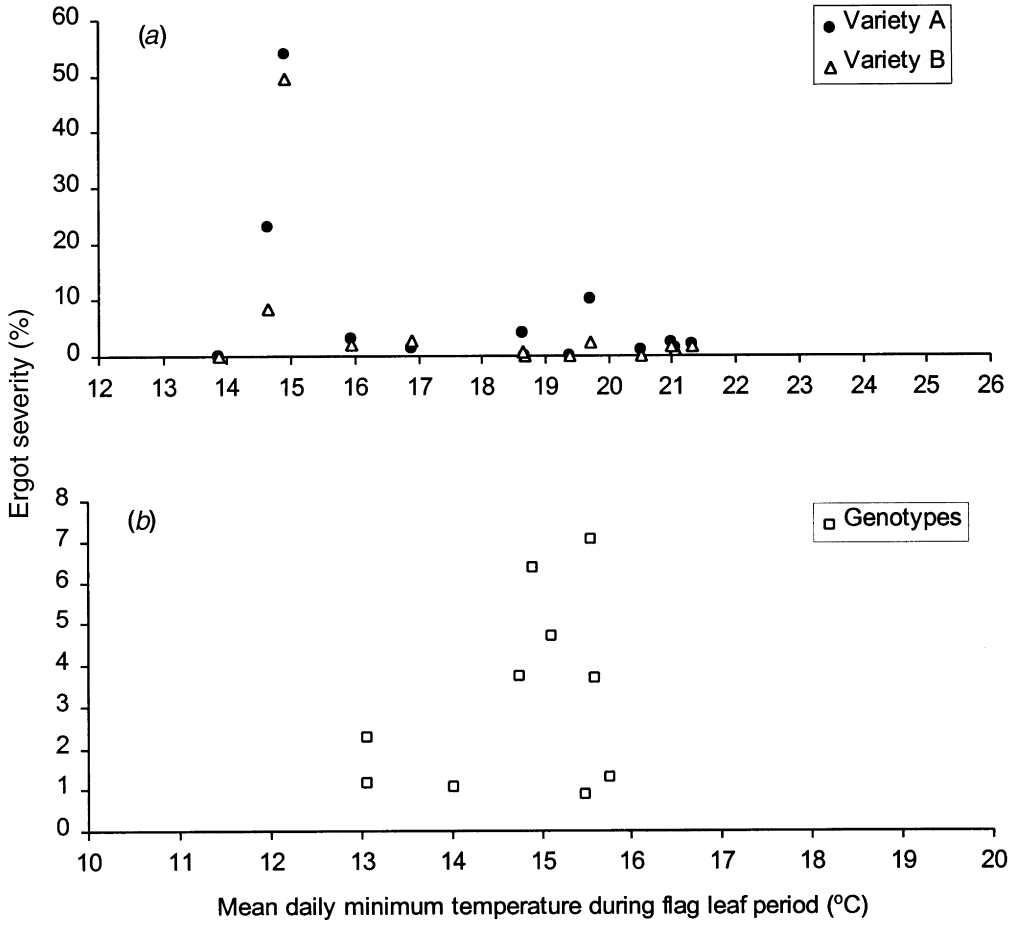


**Fig. 3.** Relationship between ergot severity and the mean daily minimum temperature during the flag leaf stage: (*a*) for the 2 genotypes in the serial planting trial; (*b*) mean ergot severity value for all genotypes in the genotype trial.

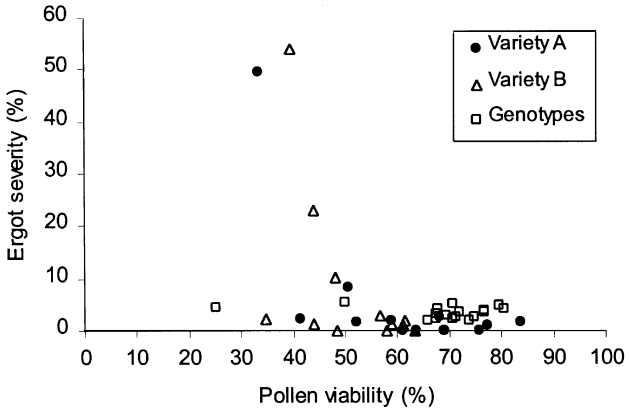


**Fig. 4.** Relationship between pollen viability and ergot severity.

control treatment, 'it would appear that day conditions during maturation had more influence on proline levels than did night temperatures'.

*Weather variables during flowering period*

The analysed weather variables during the flowering period included mean values of daily maximum temperature, daily minimum temperature, daily average temperature, relative humidity at 0900 hours, and total rainfall. In the serial planting trial, ergot severity was negatively correlated with all temperatures, with the mean daily maximum temperature being the most significant, followed by the mean daily average temperature (Table 1, Fig. 6*a*, *c*). In the genotype trial, mean daily maximum temperatures varied <1°C across flowering dates (Fig. 6*b*), which may explain the lack of correlation between maximum temperature and ergot severity (Table 1). However, mean daily minimum and average temperatures were highly positively correlated with ergot severity (Figs 6*d*, 7). Mean daily average temperature tended to have a larger effect than mean daily minimum temperature (Table 1). Comparing the temperature ranges and the correlation coefficients between temperatures and ergot severity in the 2 trials (Fig. 6, Table 1), there was an increase in ergot severity with increasing temperature in the genotype trial, but a decline in ergot severity with increasing temperature in the serial planting trial. The opposite trends in the 2 trials were primarily due to differences in temperature ranges under which the trials were conducted. The maximum ergot severity occurred at a mean daily average temperature of

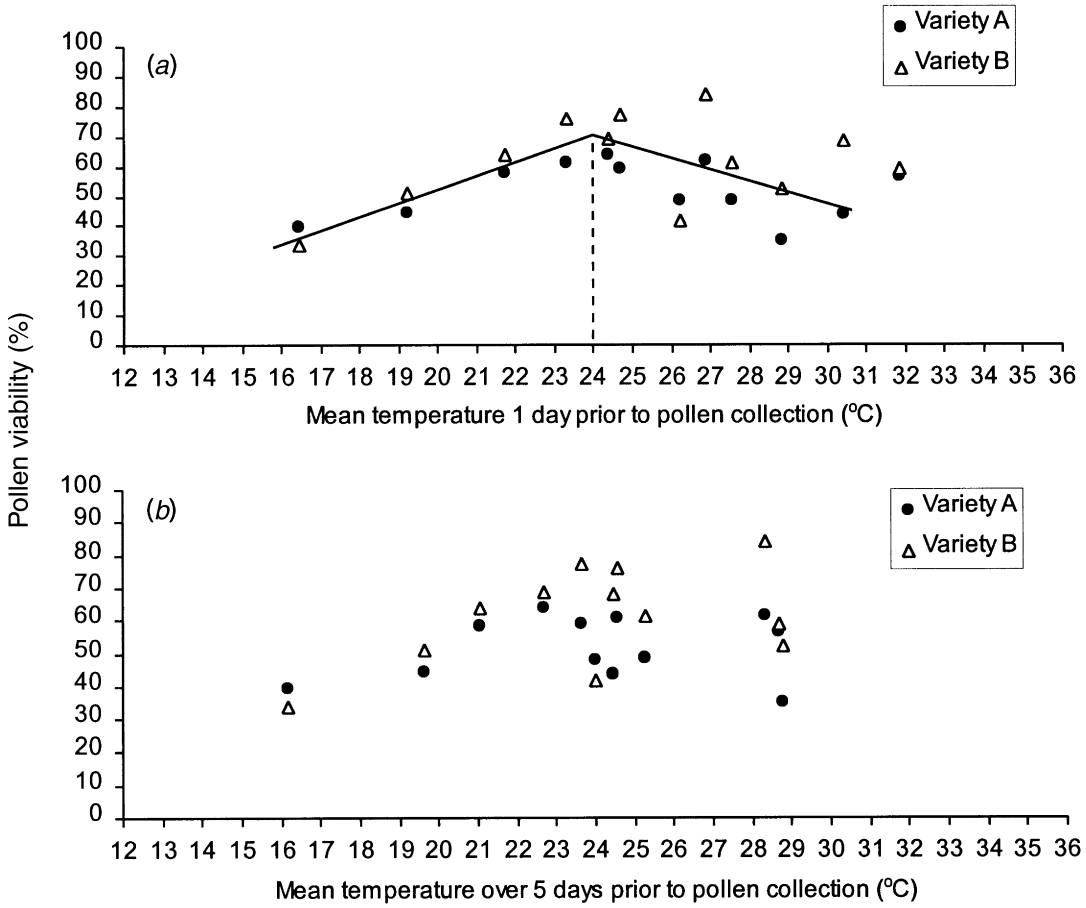


**Fig. 5.** Relationship between pollen viability and the mean temperature during the (*a*) 1- and (*b*) 5-day period prior to pollen collection. The mean temperatures were calculated using the minimum temperature on each collection date and the maximum temperatures on each previous day. Line in (*a*) was fitted by eye.

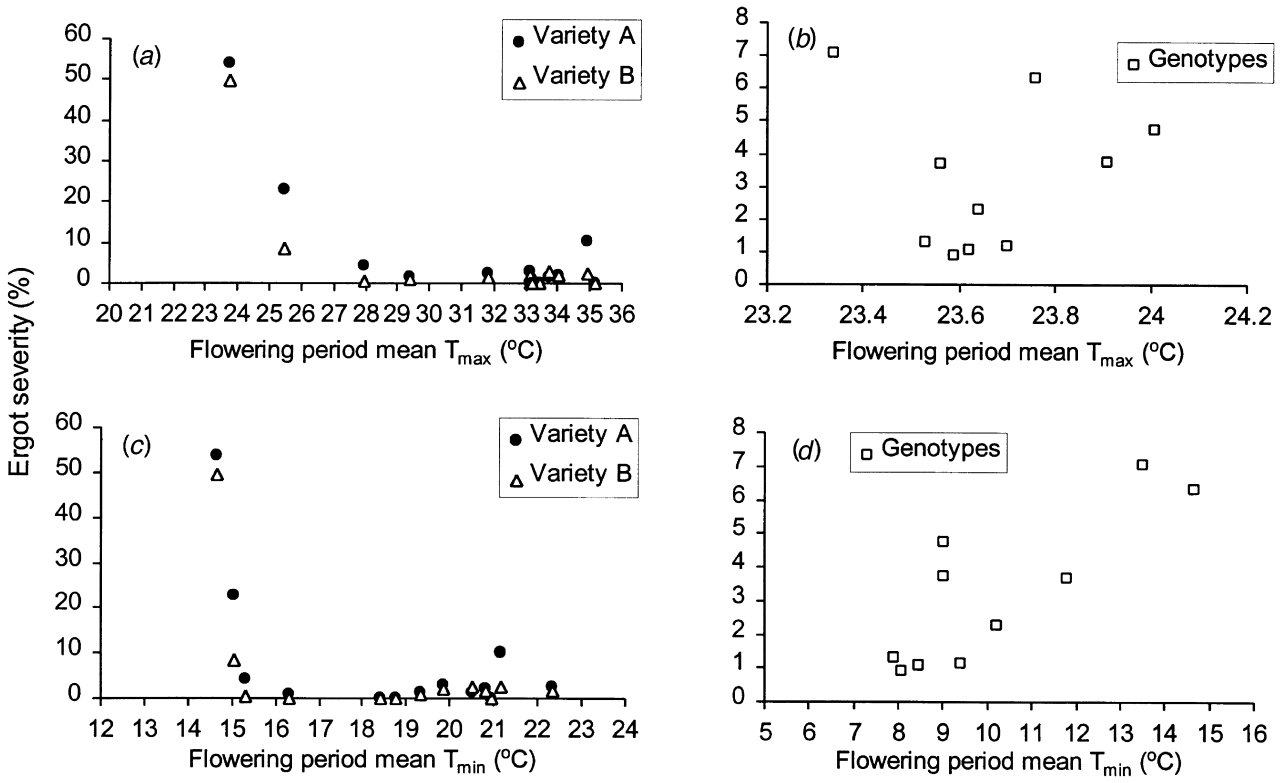


**Fig. 6**. Relationship between ergot severity and mean daily maximum ($T_{max}$) and minimum ($T_{min}$) temperatures in the (*a*,*c*) serial planting and (*b*,*d*) genotype trial.

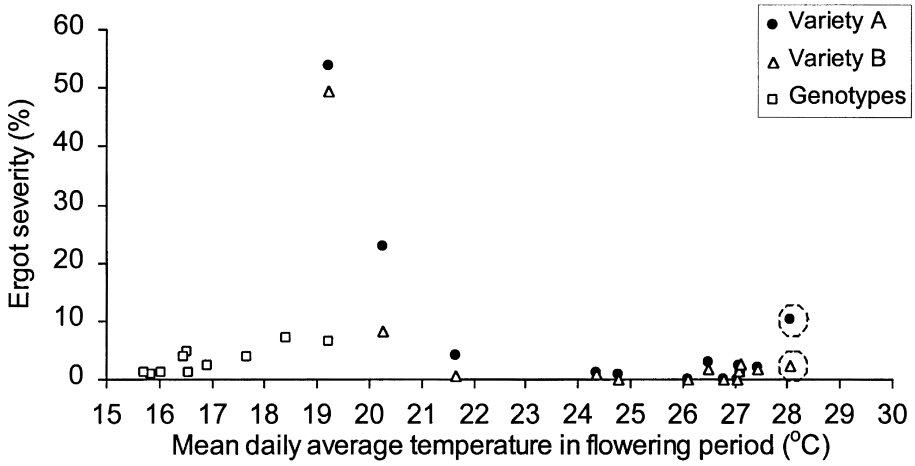


**Fig. 7.** Relationship between ergot severity and mean daily average temperatures during the flowering period in the serial planting and genotype trials.

approximately 19.5°C or a mean daily minimum temperature near 14.5°C for both trials (Figs 6, 7). The temperature range of the genotype trial was lower than the optima, whereas the temperature range of the serial planting trial was higher than the optima. Anahosur and Patil (1982) and McLaren and Wehner (1990) also found that a temperature of 19±1°C was optimal for ergot infection. Recent experimental evidence (S. A. Bhuiyan, pers. comm.) indicates that the minimum, optimum, and maximum temperature for macroconidium germination of Australian isolates of *C. africana* is 8°C, 20°C, and 32°C, respectively.

Based on these results an infection factor ($f$) was developed by modifying the equation of McLaren and Wehner (1990):

$$f = \exp(-0.026T^2+1.014T-9.8865) \quad (1)$$

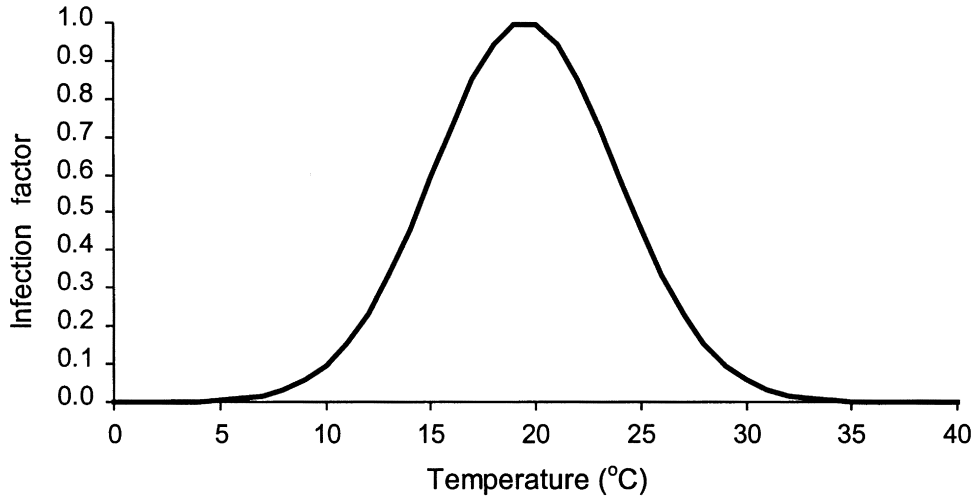


**Fig. 8.** Relationship between ergot infection and the mean temperature during the flowering period (infection period).

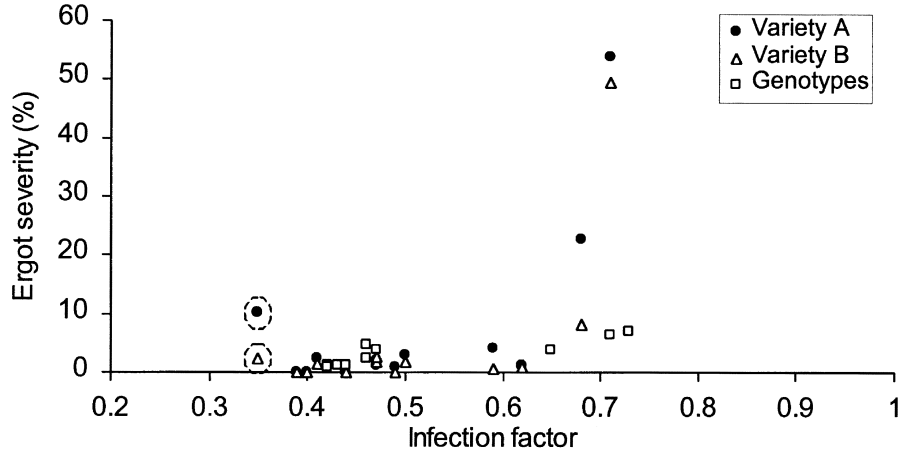


**Fig. 9**. Relationship between observed ergot severity and mean value of the infection factor calculated using hourly temperatures during the flowering period in the serial planting and genotype trials.

where $T$ is the temperature (°C) at a particular time in the flowering period. The infection factor $f$, which ranges from 0.0 to 1.0, describes the temperature response of the infection process, with 0.0 meaning no infection would occur at these temperatures, and 1.0 that the temperature is most favourable for infection. Fig. 8 displays the shape of this factor. Fig. 9 displays the relationship between the calculated mean $f$ values ($F$) during the flowering period using hourly temperatures for both trials, and severity. In both trials, ergot severity increased with increasing infection factor. Except for the severity values on one flowering date (circled), infection occurred when $F > 0.4$. In both trials the observed ergot severity values were significantly positively correlated with the introduced infection factor (Table 1).

McLaren and Wehner (1990) found that the mean daily maximum temperature 1–4 days after commencement of pollen shed (when temperature ranged from 22° to 34°C) was negatively correlated with ergot 'incidence' (equates with severity in our trials). McLaren and Flett (1998) analysed the correlation between ergot severity and the pentad values of weather variables during pre- and post-flowering. They found that the highest correlations were between mean ergot severity and mean maximum temperature 1–5 days after first flowering (negative), mean minimum temperature 23–27 days before flowering (negative), and mean maximum relative humidity 1–5 days after flowering (positive). They also concluded that ergot severity decreases with increasing temperature and is negligible at temperatures

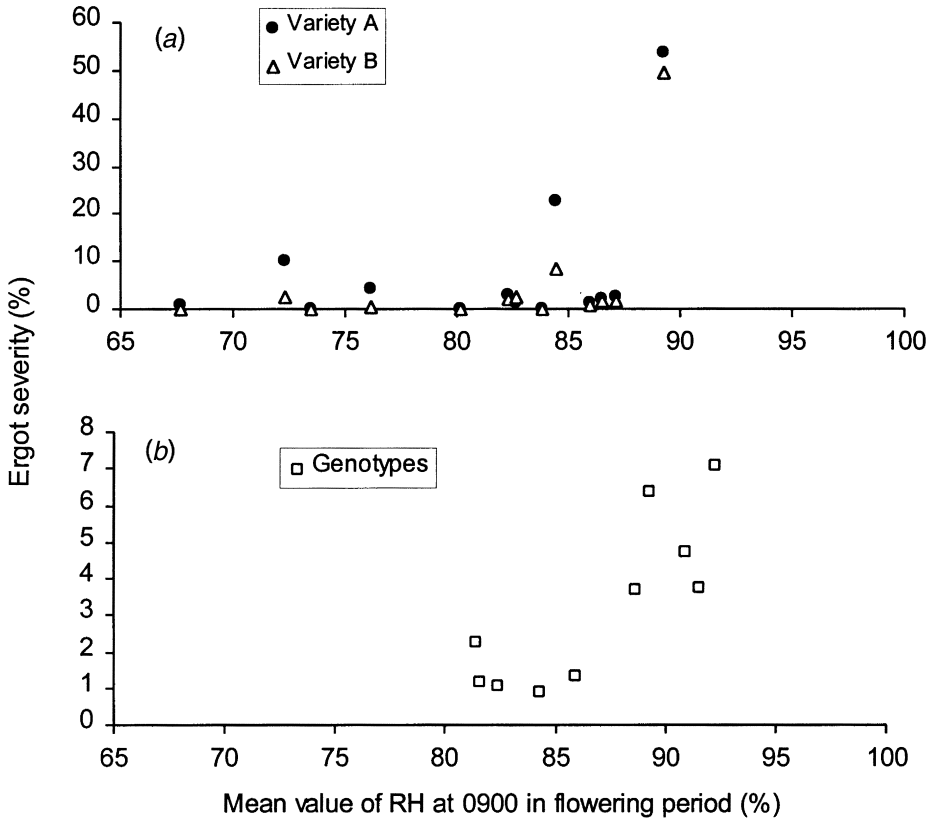


**Fig. 10.** Relationship between ergot severity and the mean value of relative humidity (RH) at 0900 hours during the flowering period in the serial planting and genotype trials.

>28–30°C. Our data from the serial planting trial (temperature range: 22–35°C) support these conclusions (Fig. 6*a*). However, at mean temperatures lower than approximately 19°C, which commonly occurred in the genotype trial, maximum temperature was not correlated with ergot severity (Fig. 6*b*), whereas, ergot severity increased with increasing (minimum) temperatures (Fig. 6*d*). This illustrates the importance of considering the temperature response of the infection process.

Fig. 10 displays the relationship between ergot severity and the mean relative humidity at 0900 hours for both the serial planting trial (Fig. 10*a*) and the genotype test (Fig. 10*b*). In the genotype trial, humidity was significantly correlated with ergot severity ($R$ = 0.83), whereas no significant correlation was observed between the two in the serial planting trial (Table 1). However, in the serial planting trial there was an apparent increase in ergot severity as relative humidity increased above 80% (Fig. 10*a*). Fig. 11 displays the combined effect of relative humidity and infection factor on ergot severity. Ergot severity increased with increasing infection factor and relative humidity, except for one flowering date. A significant correlation was also found between ergot severity and total rainfall during the flowering period in the genotype trial ($r$ = 0.80, Table 1). In these trials, high rainfall was, naturally, correlated with high relative humidity. High rainfall may inhibit pollen dispersal and also wash pollen from the stigmas, thereby affecting the pollination process. The role of high intensity rain *per se* on the infection process is not clear.

### *Critical stages and critical factors*

The use of the APSIM model to calculate the onset and duration of the flag leaf stage and to calculate the duration of the flowering period should be more accurate than using a fixed number of days. The development rate of a sorghum plant is very dependent on temperature and other environmental factors. The effect can be quite large. For example, in the serial planting trial the calculated length of the flag leaf period ranged from 5 to 7 days and the calculated duration of the flowering period lasted 6–9 days. By comparison, under the cooler conditions experienced by the genotype trial, the duration of these 2 periods ranged from 7 to 8 days and from 9 to 11 days, respectively. McLaren and Flett (1998) found that low minimum temperatures 3–4 weeks before flowering adversely affected pollen viability and hence predisposed panicles to infection. In our trials, the calculated duration from full expansion of the flag leaf to first anthesis ranged from 8 to 16 days. The use of a sorghum growth model, such

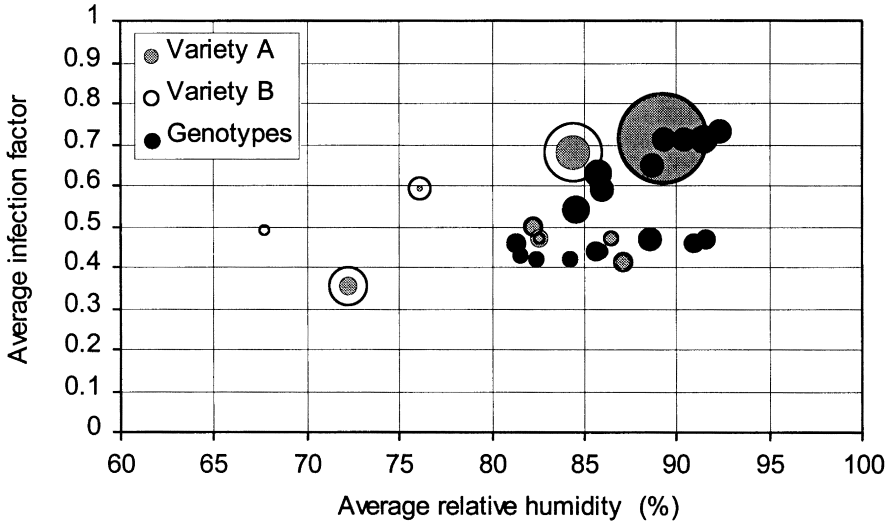


**Fig. 11.** Effect of mean relative humidity and mean infection factor during the flowering period on ergot severity. The area of the circles represents the ergot severity. The biggest circle represents an ergot severity value of 53.8%.

as APSIM, to calculate the onset and duration of the critical growth stages will allow the ergot prediction model to be used across a wide range of environments and so help in ergot management.

In our trials, temperature and relative humidity during the flowering period were the most critical factors influencing ergot infection (Table 1). At temperatures higher than 19.5°C (serial planting trial), ergot severity was negatively correlated with temperatures, whereas at temperatures below 19.5°C (genotype test) the correlation was positive. This may be related to the temperature response of the pathogen, in that infection increases with increasing temperature below an optimum of 19.5°C and then declines with temperatures above the optimum. Based on the present data, infection is likely to occur (potential infection) if the infection factor in the flowering period is >0.3 and the mean relative humidity at 0900 hours is >70% (Figs 10, 11).

*The regression model*

A regression model was established based on the data obtained from the genotype trial. Using the mean daily relative humidity (*RH*) at 0900 hours and the mean hourly infection factor (*F*) during the flowering period, Eqn 2 was obtained with $R^2 = 0.88$:

$$Y = 10.169\,F + 0.2548RH - 24.1955 \quad (2)$$

where *Y* is the mean ergot severity (%) across genotypes. By introducing the mean daily minimum temperature during the flag leaf stage ($T_{flm}$) into the equation, the $R^2$ value was increased to 0.94:

$$Y = 9.9953\,F + 0.379\,RH - 0.7532T_{flm} - 23.799 \quad (3)$$

All the 3 factors are significant in the regression model with $P < 0.05$, although $T_{flm}$ was not correlated with the observed ergot severity (Table 1, Fig. 3).

There was a poor correlation between the observed severity on the 2 hybrids on flowering days in the serial planting trial and severity values predicted by Eqn 3. The above equations were developed from using the mean values of severity for all genotypes that flowered on the same day during the genotype trial, and may not be applicable to individual genotypes, which undoubtedly differ in their response to climatic conditions, particularly temperature. In addition, only one inoculation was conducted in the serial planting trial, when the male sterile line in the first planting flowered. Infection of sorghum flowers of the hybrids and the male sterile line in subsequent plantings was from secondary conidia which were formed on panicles that had flowered earlier. Weather factors, particularly temperature and relative humidity, have a significant, but as yet poorly defined, influence on the production and dispersal of secondary conidia. Consequently, the relationships between ergot severity and climatic variables during natural infection would be expected to be different from those obtained from the genotype test where all flowering panicles were inoculated. However, the equations retrospectively predicted severe ergot outbreaks, which occurred at Gatton in mid April 1996 (Ryley *et al*. 1996) and at Monto, central Queensland, in late April–early May 1997 (M. Ryley, unpubl.data).

*Existing problems and further research needs*

Although many research papers have been published on sorghum ergot, there are still many unanswered questions about the biology and epidemiology of the disease. Because

fertilised ovaries are resistant to infection, flowering behaviour is one of the most important factors for ergot infection. Factors which prolong the period from floret opening to fertilisation will promote ergot infection (Futrell and Webster 1965).

When pollen and conidia are placed concurrently on the stigma, pollen germinates within 30 min and fertilisation takes place within another 2–12 h, whereas conidia require 8–24 h to germinate and several days to reach the ovary (Bandyopadhyay *et al*. 1992). That is, pollen tubes reach the embryo sac faster than do the colonising hyphae. However, the quantity of viable pollen needed for successful fertilisation of most of the florets is unknown. If the stigmas remain unfertilised, it is not clear how long they remain receptive to pollen and ergot and how environmental factors affect the receptivity of stigmas and the rates of pollination and fertilisation. Furthermore, the quantity and viability of pollen might be affected by genetic and environmental factors, e.g. temperatures (Dhopte 1984) and humidity (Lansac *et al.* 1994), beyond an optimum. Studies on the influence of environmental factors on stigma receptivity, pollen production, and the infection process are essential.

It is also important to gain a better understanding of the phenology of flowering in sorghum, when it is vulnerable to ergot infection. Under our conditions an individual panicle requires 4–12 days for all spikelets to flower. Flowering proceeds basipetally on a panicle as glumes of spikelets in a horizontal plane open at about the same time (Ayyangar and Rao 1931; Stephens and Quinby 1934). On any day of the flowering period a panicle may have an upper region of spikelets with dried anthers that have dehisced pollen, a middle region of spikelets with yellow anthers shedding fresh pollen, and a basal portion of immature florets (Pendleton *et al*. 1994). The proportion of those stages varies daily for particular panicles and is controlled by panicle flowering rate determined mainly by temperatures. Therefore the quantity of viable pollen and the numbers of receptive stigmas differ from day to day in the total flowering period (Patil and Goud 1980). The quantity of viable pollen and stigma receptivity should be investigated on several days instead of one day in the flowering period. Pollen viability should be measured by pollen germination rather than pollen starch (as measured by iodine staining). Clarification of whether temperature in the starch accumulation stage influences pollen quantity and viability is needed. The relative sensitivities of genotypes to pre-flowering low night temperatures in the pollen production process also need to be studied.

The model developed by McLaren and others in South Africa was based on data generated from inoculated trials, as is the model reported here. These predictive models have application in resistance screening, when genotypes flowering on different days are exposed to different ergot 'potentials' *sensu* McLaren and Flett (1998) and McLaren (1997). As outlined previously the models may not be applicable under conditions of natural infection. Evidence from the serial planting trials suggests that many of the interactions between variables such as temperature and relative humidity and ergot severity are non-linear. Further research of this type is currently being continued in Australia, and also needs to be conducted overseas. In addition, the interactions between climatic variables on secondary sporulation, spore dispersal, and infection need to be quantified, in order to better define critical periods during the day. These studies will help us to better understand the weather conditions which lead to sorghum ergot epidemics, and give us the ability to predict outbreaks and take preventative action, particularly in seed production blocks.

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