

# Plant Disease Problems in Central India



INDIAN PHYTOPATHOLOGICAL SOCIETY

# Plant Disease Problems in Central India

*Edited by*

*K. Muralidharan and C.S. Reddy*

*Department of Plant Pathology*

*Directorate of Rice Research*

*Hyderabad 500 030*



INDIAN PHYTOPATHOLOGICAL SOCIETY

**Correct Citation : *Plant Disease Problems In Central India***  
**Editors : Muralldharan K and Reddy C S**  
**Proc Symp Central Zone**  
**Indian Phytopathological Society**  
**November 28-29, 1991 at**  
**Directorate of Rice Research**  
**Hyderabad 500 030 India**

# Pathometry of Fusarium Wilt of Pigeonpea

M.K. Naik and M.V. Reddy

Legumes Pathology, International Crops Research Institute for the Semi-Arid Tropics,  
Patancheru PO, Andhra Pradesh 502 324, India

**D**isease progress curves (DPC), apparent infection rates ( $r$ ) and area under disease progress curves (AUDPC) were obtained for Fusarium wilt-susceptible (ICP 2376), moderately susceptible (C11) and resistant (ICP 8863) pigeonpea cultivars in a vertisol wilt sick plot. Wilt progress was logarithmic with a low  $r$  (0.0034) in susceptible cultivar till 50 days after sowing (DAS). The  $r$  values reached 0.0271 at 95 DAS. The highest  $r$  obtained was 0.018 and 0.00067, respectively in the moderately susceptible and resistant cultivars. AUDPC in the susceptible, moderately susceptible and resistant cultivars were 742, 562, and 17, respectively. The low values of  $r$  and AUDPC estimated in ICP 8863 confirm its resistance to wilt. The  $r$  value coupled with AUDPC for wilt can be useful in evaluation of pigeonpeas for wilt resistance and for studying influence of various other factors such as crop mixtures, rotations, pathotypes on wilt incidence.

**Additional keywords:** *Cajanus cajan* infection rate; Disease progress curve; Inoculum density; Resistance; Wilt sick plot

One of the reasons for low seed yield of pigeonpea in the SemiArid Tropics is the occurrence of diseases, particularly *Fusarium wilt* (*Fusarium udum* Butler) which causes substantial losses (Kannaiyan et al. 1984). In recent years, much progress has been made in measuring and analyzing the epidemics of foliar diseases but our understanding about the measurement and quantitative aspects of soil-borne diseases is rudimentary. Analysis of disease variables in meaningful units help in better understanding of diseases and as well help in taking policy decisions about disease management. Hence, we used disease progress curves, area under the disease progress curve (AUDPC), apparent rate of infection ( $r$ ), and inoculum density and disease relationship (ID-D) to study the epidemiology of fusarium wilt of pigeonpea.

## MATERIALS AND METHODS

The experiment was conducted in a well established *Fusarium* wilt sick plot in vertisol at Patancheru, India. The sick plot was developed during 1975 to 1977 by growing wilt susceptible lines such as ICP 6997 and incorporating the wilted plants into soil. Sickness of the plot was subsequently maintained by inter-planting susceptible lines (1:2 or 1:4) with germplasm accessions and breeding material. The sick plot contained 4945 colony forming units (cfu per gram of soil). Sowing were made June in 1991 with the inter- and intra-row spacings of 60 cm and 20 cm, respectively. The trial was conducted under rainfed conditions (annual rainfall 718.9 mm). *F. udum* population in wilt sick soil was estimated at sowing using melachite

green medium (Singh and Chaube 1970). Three cultivars; ICP 8863 (resistant), C-11 (moderately susceptible) and ICP 2376 (susceptible) were used in the study. The experimental design used was RBD with 2 replications. The wilted plants in different cultivars were recorded at 15 days interval starting from 30 days after sowing.

#### Computation of apparent rate of infection (r)

Procedures or equations to calculate the apparent rate of infection at different stages have been established by Vanderplank (1963). Hence, the following formula with scope for introduction of correction factor was directly used for computing infection rate:

$$r = \frac{1}{(t_2 - t_1)} \times \left[ \log_e \frac{1}{(1 - Y_2)} - \log_e \frac{1}{(1 - Y_1)} \right]$$

where  $y_1$  and  $y_2$  = disease incidence levels at time  $t_1$  and  $t_2$  (1-y) = correction factor, which allows for a decreasing proportion of healthy plant left for infection.

#### Estimation of area under disease progress curves (AUDPC)

The following formula given by Wilcoxon et al. (1975) was used.

$$AUDPC = \sum_{i=1}^k \frac{1}{2} (y_i + y_{i+1}) \times d$$

where,  $y_i$  = disease incidence at the end of week  $i$ ;  $k$  = number of successive evaluations of disease; and  $d$  = interval between two evaluations.

#### Plotting the disease progress curves

Disease incidence was plotted in a cartesian plane taking  $\log_e (1/1-y)$  on the ordinate (Y axis) and the time on the abscissa

(X axis) where (1-y) is the correction factor for disease level.

#### Inoculum density and disease relation (ID-D)

Eight soil dilutions from wilt sick plot at ICRISAT (4945 cfu/g soil) were prepared in 8"-diameter pots by mixing the sick soil with sterilized soil. Populations of *F. udum* in different dilutions were estimated by using a selective medium (malachite green). Each soil sample was plated on five petridishes for estimating the inoculum. These inoculum dilutions were used to measure wilt incidence in the three genotype (ICP 2370, C-11 and ICP 8863).

### RESULTS AND DISCUSSION

Log transformation as advocated by Vanderplank (1963) was used for linearization of disease progress curve. Initially the progress was conspicuously logarithmic both in susceptible and moderately-susceptible genotypes owing to low rate of infection and thereafter the disease progress switched over to non-logarithmic phase due to higher infection rate. The incidence level reached a plateau at the termination of the season. The term "logarithmic" usually refers to low infection rate of the disease in disease progress curve. In case of pigeonpea wilt though infection occurs early (with in a week after sowing) disease symptoms appear only after a month. Disease progress is slow during the vegetative phase (4 months) and accelerates only in the reproductive phase. The reasons for such disease pattern are not clear. Some of the possible explanations for higher incidence of wilt in the reproductive phase include: increased susceptibility of pigeonpea to wilt in the reproductive stage compared to vegetative stage; slow colonization of *F. udum* in the host due to woody nature of the plant; and availability

**Table 1** Apparent rate of infection (*r*) and area under disease progress curve (AUDPC) of *Fusarium wilt* of pigeonpea

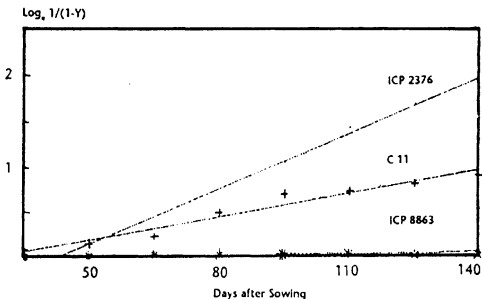
Days after sowing	ICP 2376 (Susceptible)		C-11 (Moderately susceptible)		ICP 8863 (Resistant)	
	<i>r</i>	AUDPC	<i>r</i>	AUDPC	<i>r</i>	AUDPC
35	-	-	-	-	-	-
50	0.0037	110	0.0045	147	0	0
65	0.0089	232	0.0055	245	0.00670	7.01
80	0.0265	510	0.0180	438	0.00013	16.61
95	0.0271	837	0.0136	665	0	19.2
110	0.0257	1055	0.0026	765	0	19.2
125	0.0170	1181	0.0052	806	0.00047	24.3
140	0.0223	1261	0.0059	861	0	29.4

of more moisture in the vegetative stage than in the reproductive stage. The disease progress curve as a result varied slightly from that of typical monocyclic disease pattern.

When the growth curve was analysed in terms of apparent rate of infection (*r*), the initial *r* was 0.0037 and reached the highest *r* of 0.0271 at 95 DAS in the susceptible ICP 2376 (Table 1 and Figure 1). In moderately susceptible genotype, C-11, the initial rate of infection was 0.0045 but reached the highest rate of 0.0136 at 95 DAS. The resistant genotype ICP 8863 had a very low

infection rate of 0.0067 in the logarithmic phase till the end. It is inferred that as the amount of disease increases, the amount of susceptible host roots available for infection would not remain constant. Therefore, at the termination of the growing season the growth curve reached a plateau. The initial low *r* in the logarithmic phase accelerated due to higher colonization in the next phase.

Area under the disease progress curve (AUDPC) is another way of expressing the course of disease development and has been considered a reliable and convenient



**Figure 1.** Progress of *Fusarium wilt* in vertisol

procedure for epidemic analysis. The greatest change in AUDPC value was recorded between 80 and 95 DAS in both susceptible and moderately susceptible genotypes.

Vanderplank (1963) utilizing the data of Ware and Young (1933) depicted a logarithmic growth rate followed by non-logarithmic disease progress for cotton wilt incited by *Fusarium oxysporum* f.sp. *vasinfectum*. The rate of disease increase in the present experiment was similar to that of cotton wilt except for a short duration in each phase of the disease. Such log transformation and calculation of  $r$  has been stressed as a superior technique for epidemic analysis by Vanderplank (1963). On the other hand, Wilcoxon et al. (1975) considered estimation of AUDPC to be a more appropriate technique for epidemic analysis than the  $r$  value. In case of pigeonpea wilt, both the amount of wilt (indicated by AUDPC) and the time at which wilt occurs (indicated by  $r$ ) are important. Wilting before pod set causes a total loss and wilting at later stages, results in a partial loss. Hence both  $r$  and AUDPC are important in epidemic analysis.

#### Inoculum density and disease (ID-D)

Wilt was not observed at 1:200 dilution of wilt sick soil (15 cfu/g soil) in susceptible ICP 2376, but it occurred at 1:100 (34 cfu/g) dilution (Figure. 2) with incidence increasing up to 1:10 dilution (261 cfu/g soil). Wilt incidence in moderately susceptible C-11 was observed at 1:25 dilution (111 cfu/g soil) and increased at 1:5 dilution (517 cfu/g soil). No wilt was observed at 1:200, 1:100 and 1:50 dilutions. The resistant ICP 8863 had a very low wilt incidence (5%) in undiluted sick soil. In other crops, the number of propagules of *Fusarium* in soil also had a direct relationship with severity of wilt (Netzer, 1976). However, Cook (1968) did not detect a plateau for *Fusarium* foot rot of cereals, even at 104 propagules/g of soil. He concluded that in addition to inoculum, there are other factors involved in disease severity. Douglas (1970) favoured the theory of ID-D only over an intermediate range of inoculum concentrations.

Log-log transformation suggested by Baker et al. (1967) was utilized for the study of ID-D relationship in pigeonpea

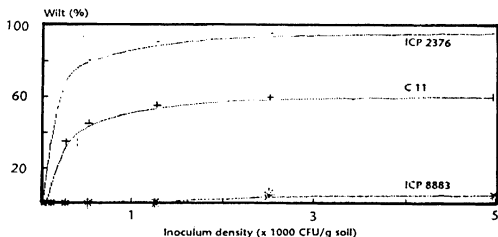
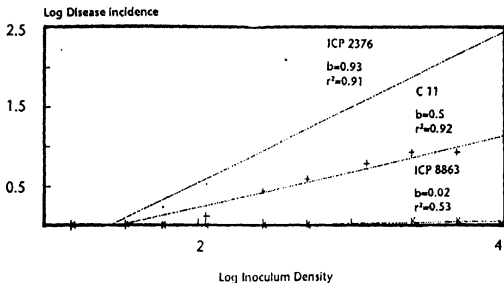


Figure 2. Inoculum density and disease relationship in *Fusarium* wilt of pigeonpea



**Figure 3.** Inoculum density and disease model for *Fusarium* wilt of pigeonpea

wilt (Figure 3). The ID-D indicated a slope value of 0.93 and 0.5 for susceptible and moderately susceptible genotypes, respectively with 91% correlation (Figure 3)

In this model, it is assumed that the root tip moves through the soil and activate increasing proportions of inoculum. Slope values similar to the present values have been reported for related host-pathogen systems. Mitchell and Hurwitz (1975) demonstrated a slope of near 1.0 for *Pythium* infection of rye. In damping-off of radish, the slope values conformed to this model (Benson and Baker 1974). However, variation in slope values, especially in vascular wilt, is common because of the influence of symptom expression by environmental factors and the absence of symptom expression in the host plant (Baker and Phillips 1962).

The infection rate and AUDPC obtained in this study confirm the resistance of ICP 8863 to wilt. The  $r$  value in a single figure reveals the nature of the pathogen, variety of the host-plant under cultivation and prevalent environmental conditions. The  $r$

value coupled with AUDPC for wilt is helpful in evaluation of pigeonpeas for wilt resistance and for studying the influence of crop seed mixtures, rotations and pathotypes on wilt incidence. Similarly, the ID-D is useful in studying inoculum density in relation to crop sequence, crop density, environmental effects and for comparison of control measures.

#### REFERENCES

- Baker R, Maurer CL, Maurer RA (1967) Ecology of plant pathogens in soil. VII Mathematical models and inoculum density. *Phytopathology* 57: 662-666
- Baker R Phillips DJ (1962) Obtaining pathogen-free stock by shoot tip culture. *Phytopathology* 52: 1242-1244
- Benson DM, Baker R (1974) Epidemiology of *Rhizoctonia solani* pre-emergence damping-off of radish : Inoculum potential and disease potential interaction. *Phytopathology* 64: 957-962



- Cook RJ** (1968) *Fusarium* foot rot and foot rot of cereals in the Pacific North West. *Phytopathology* **58**: 127-131
- Douglas DR** (1970) The effect of inoculum concentrations on the apparent resistance to muskmelons to *Fusarium oxysporum* f. sp. *melonis*. *Can J Bot* **48**: 687-693
- Kannalyan J, Nene YL, Reddy MV, Ryan JG, Raju TN** (1984) Prevalence of pigeonpea diseases and associated crop losses in Asia, Africa and the Americas. *Tropical Pest Manage* **30**: 62-71
- Mitchell R, Hurwitz E** (1975) Suppression of *Pythium debaryanum* by lytic rhizosphere bacteria. *Phytopathology* **55**: 156-158
- Netzer D** (1976) Physiological races and soil population level of *Fusarium* wilt of watermelon. *Phytophthora* **4**: 1 131136
- Singh R S, Chaube HS** (1970) A technique for estimation of hyphal and conidial propagules of *Fusarium* in soil. *Labdev J Sci Technol* **8**: 172-174
- Vanderplank JE** (1963) *Plant Diseases: Epidemics and Control*. Acad Press, New York
- Ware JO, Young VH** (1934) Control of cotton wilt and rust. *Univ Arkansas (Fayetteville) Agri Expt Sta Bull* **308**:23
- Wilcoxson RD, Skvand B, Atif AH** (1975) Evaluation of wheat cultivars for ability to retard the development of stem rust. *Ann Appl Biol* **80**: 275-281