HOST INFLUENCES ON EPIDEMIC DEVELOPMENT OF PEANUT LEAF SPOT CAUSED BY *Cercosporidium personatum* (BERK. AND CURT.) DEIGHTON

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ABSTRACT

Based on one cycle of pathogenesis due to peanut leaf spot fungus, *Cercosporidium personatum* (Berk. and Curt.) Deighton, resistant cultivars PI 259747 and EC 76446 (292) significantly prolonged the latent period of the disease by almost two times that of susceptible cultivar CES 101. This indicates 50% reduction in the potential number of nonoverlapping disease cycles that could be generated during the course of the epidemic. PI 259747 sustained a four-fold slower rate of lesion enlargement and six-fold less spore production rate than that of CES 101 based on data taken at 4, 5 and 6 weeks after inoculation. Sporulation rate was positively related to the rate of lesion enlargement ($r = 0.86$). Possessing some of the rate-reducing resistance traits, cultivars PI 259747, NC Acc 17133 (RF), PI 350680 and EC 76446 (292) significantly slowed down the progress of leaf spot epidemic in the field by 55-75% relative to that of CES 101. PI 259747 and NC Acc 17133 (RF) distinctively reduced the numerical increase of lesions by 85% relative to CES 101 within 6 weeks from disease onset.

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KEY WORDS: Peanut leaf spot. *Cercosporidium personatum.* Rate-reducing resistance. Infection rate.

INTRODUCTION

A weather-based computer program to generate daily peanut spray advisory aimed at enhancing the efficiency of fungicidal use against *Cercospora* leaf spot has been developed (Parvin et al., 1974; Phipps and Powell, 1984). This and the availability of increasingly new and useful knowledge regarding the component processes of *Cercospora* leaf spot epidemic should facili-

tate the development of an effective disease management system for peanut. Considered as an economical control strategy, host resistance must be an essential component in the disease management program whenever available. An intensified peanut breeding program to incorporate resistance traits to *Cercosporidium personatum* (Berk. and Curt.) Deighton has been underway (Subrahmanyam et al., 1985; Paningbatan and Ilag, 1984).

Resistance may reduce the initial amount of inoculum (Xo) or the multiplication rate of the pathogen, r (Vanderplank, 1975). Rate-reducing resistance is manifested in various ways such as latent period, infection frequency, sporulation capacity, lesion or colony size and infectious period (Berger, 1977; Parlevliet, 1979). The information about host influences on such components of an epidemic facilitates the prediction of the eventual speed through which peanut leaf spot disease develops. Fry (1982) aptly states that the first step in the development of an effective disease management program is the analysis of the disease dynamics so that the most appropriate control strategies could be identified.

The peanut leaf spot disease dynamics has not been fully investigated yet. Hence, this study was conducted to analyze the effects of host resistance on the various components of epidemic development of peanut leaf spot and on the infection rate of *C. personatum* in the Central Experiment Station of the University of the Philippines at Los Baños.

MATERIALS AND METHODS

Pot Experiment

Assessment of the Components of Rate-Reducing Resistance. Four peanut cultivars [CES 101, CES 2-25, EC 76466 (292), PI 259747] were grown in 30-cm pots with baked garden soil. Each cultivar was grown in five pots with two to three plants per pot. One plant per pot was randomly chosen to assess the individual components of resistance. The third and fourth leaves of each plant were tagged and uniformly inoculated with 5×10^5 spores/mL of water from 3-wk old onion agar cultures of *C. personatum* (Paningbatan, 1980). Both surfaces of the leaves were sprayed with the conidial suspension using a plastic atomizer until inoculum run-off. The treatments were replicated five times using the completely randomized design. The latent period (the time between inoculation and onset of sporulation), rates of lesion enlargement and sporulation were observed and recorded. Infectious period was not noted because the pathogen is a necrotrophic organism. Rates of lesion enlargement and sporulation were estimated through simple linear regressions of lesion size and spore yield per lesion over time, i.e., fourth, fifth and sixth week after inoculation. Lesion diameter was taken as the mean of the largest and shortest measurements. Spore count per lesion was determined with the aid of a hemacytometer by carefully suspending a lesion in one mL of water with 500 ppm Tween 80.

A replicate for each treatment consisted of the average number of spores counted in six chambers of the hemacytometer.

Field Experiment

Numerical Formation of Lesions. Five peanut genotypes [CES 12-12, CES 12-24, CES 12-26, PI 259747 and NC Acc 17133 (RF)] and three locally recommended cultivars (CES 101, UPL Pn4, BPI P9) with varying levels of resistance to leaf spots (IPB, 1982) were grown in single-row 5 x 1 m plots following the standard planting rate recommended by PCARRD (1978). Used as infector plants and systematically grown as line sources as described by Zadoks and Schein (1979) for every two rows of test plants, CES 101 plants were inoculated one week after emergence by spraying all the leaves with 3×10^4 conidia/mL sterile water from 3 week old onion agar cultures of *C. personatum*. Five sample plants per replicate were randomly chosen from a population of 55-60 plants, and every fifth oldest leaf on the main stem of each sample was tagged for weekly observation. The number of lesions was recorded weekly over 6 weeks. The numerical increase of typical lesions was computed by simple linear regression analyses.

Determination of Apparent Rates of Infection in Different Peanut Varieties. Due to the insufficient supply of seeds of CES 12-12, CES 12-24 and CES 12-26 cultivars which were used to determine the comparative numerical increase of lesions, seeds of CES 3-2, EC 76446 (292) and PI 350680 cultivars were used instead. The recommended planting practices for peanut described by PCARRD (1978) were followed. The seeds were drilled along the rows and each variety was planted on 5 x 2 m plots. The entire experimental field was surrounded by line sources composed of CES 101 plants spaced 4 meters from the test plants. The line sources were inoculated one week after emergence and served as primary sources of inoculum for the test cultivars. An isolated planting site was carefully chosen to minimize the entry of inocula other than the ones coming from the designated primary sources. Disease ratings were taken at 2-week intervals by visually estimating the proportion of leaf area infected. The apparent infection rate was estimated through simple linear regression of logits of diseased proportion of foliage per plant against time, i.e., fourth, sixth, eighth and tenth week after emergence.

RESULTS AND DISCUSSION

Table 1 shows the comparative latent period, infection frequency, rates of lesion expansion and lesion sporulation caused by *C. personatum* on four peanut test varieties based on one infection cycle. PI 259747 and EC 76446 (292), two previously confirmed resistant peanut genotypes to *C. personatum* in the field (IPB, 1982), significantly prolonged the latent period of the disease by about 11 days compared to CES 101, a susceptible cultivar. This delay in spore production implies a considerable reduction in the potential number

Table 1. Comparative infection frequency, latent period, rate of lesion enlargement and rate of sporulation in four peanut varieties inoculated with conidia of *C. personatum.*[1]

Host Cultivar	Infection Frequency (lesions/ cm^2)	Latent Period (days)	Rate of Lesion Expansion $(mm/wk)^2$	Rate of Sporulation (per 1000 lesions/ $week)^2$
CES 101	8.2a	13.2c	1.3a	17.8a
CES 2-25	6.6b	14.6b	0.8b	14.4a
EC 76446 (292)	4.4c	25.6a	0.6c	1.8c
PI 259747	2.2d	24.8a	0.3c	3.0b

[1]Values are means of five replications and each replication consisted of two leaves per plant.

[2]Simple linear regression coefficients based on aggregate data observed at fourth, fifth and sixth week after inoculation. In a column, values followed by a common letter are not significantly different at 5% level, DMRT.

of disease cycles that could be generated in the course of the epidemic. Assuming a 110-day peanut maturity period, ***C. personatum*** could produce approximately eight nonoverlapping disease cycles in CES 101 compared with only four in PI 259747. Delayed initial sporulation was similarly noted by Paningbatan and Ilag (1984) to be one of the major resistance traits of peanut to ***C. personatum.*** However, they also observed that high inoculum levels significantly shortened the periods of incubation and onset of sporulation. Inoculated with the same conidial concentration, PI 259747 produced an average of 2.2 lesions/ cm^2 leaf which was over 300% lower than the 8.2 lesions/cm^2 produced in CES 101. This agrees with the pattern observed by Paningbatan and Ilag (1984).

CES 101 displayed the biggest lesions over a period of time among the test varieties (Table 1). PI 259747 significantly exhibited more than four-fold reduction in lesion growth rate compared to CES 101. Simple linear regression analysis of the lesion diameter against time showed that CES 101 (1.3 mm/wk) sustained the fastest rate of lesion enlargement which was over four times faster than PI 259747 (0.3 mm/wk) (Fig. 1). This confirms the findings of Paningbatan and Ilag (1984) that the most resistant

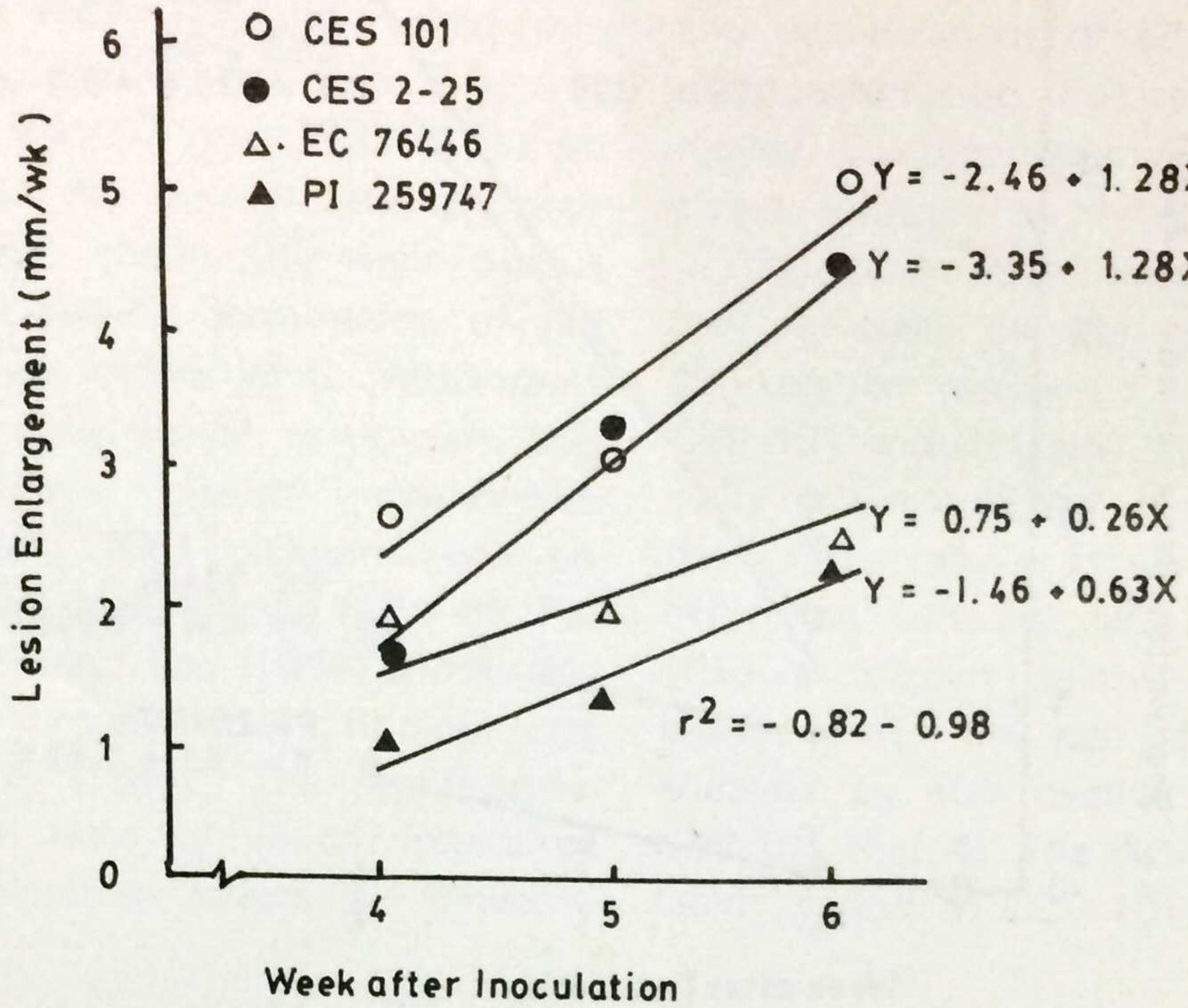

Figure 1. The rate of lesion enlargement in different peanut cultivars inoculated with *C. personatum*.

cultivar exhibited a lesion growth rate which was 60% lower than that of the most susceptible one. Moreover, they estimated that CES 101 would take approximately 10 days to attain 10-mm^2 lesion whereas PI 259747 would take about 47 days. Similar effects on lesion size in other host-pathogen systems were described by Parlevliet (1979).

Among the test cultivars, the sporulation rate varied in the same manner as the rate of lesion enlargement (Table 1). Based on the aggregate data on spore yield per lesion taken at 4, 5 and 6 weeks after inoculation, EC 76446 (292) and PI 259747 respectively reduced spore production by 90 and 80% compared to CES 101. The relatively high spore count per lesion in CES 101 and CES 2-25 was caused by their distinctly high sporulation rate (Fig. 2). The rate of spore production averaged across the host cultivars varied positively with the rate of lesion growth (r^2 = 0.86) (Fig. 3). This finding supports Parlevliet's (1979) postulate that the lesion size reflects the growth rate of the pathogen and therefore its spore production. Moreover, the study of Habgood (1977) on partial resistance of barley cultivars to *Rhynchosporium secalis* also showed that spore production varied positively with lesion size.

Within 6 weeks after the onset of the disease, the rates of lesion formation varied remarkably in the different

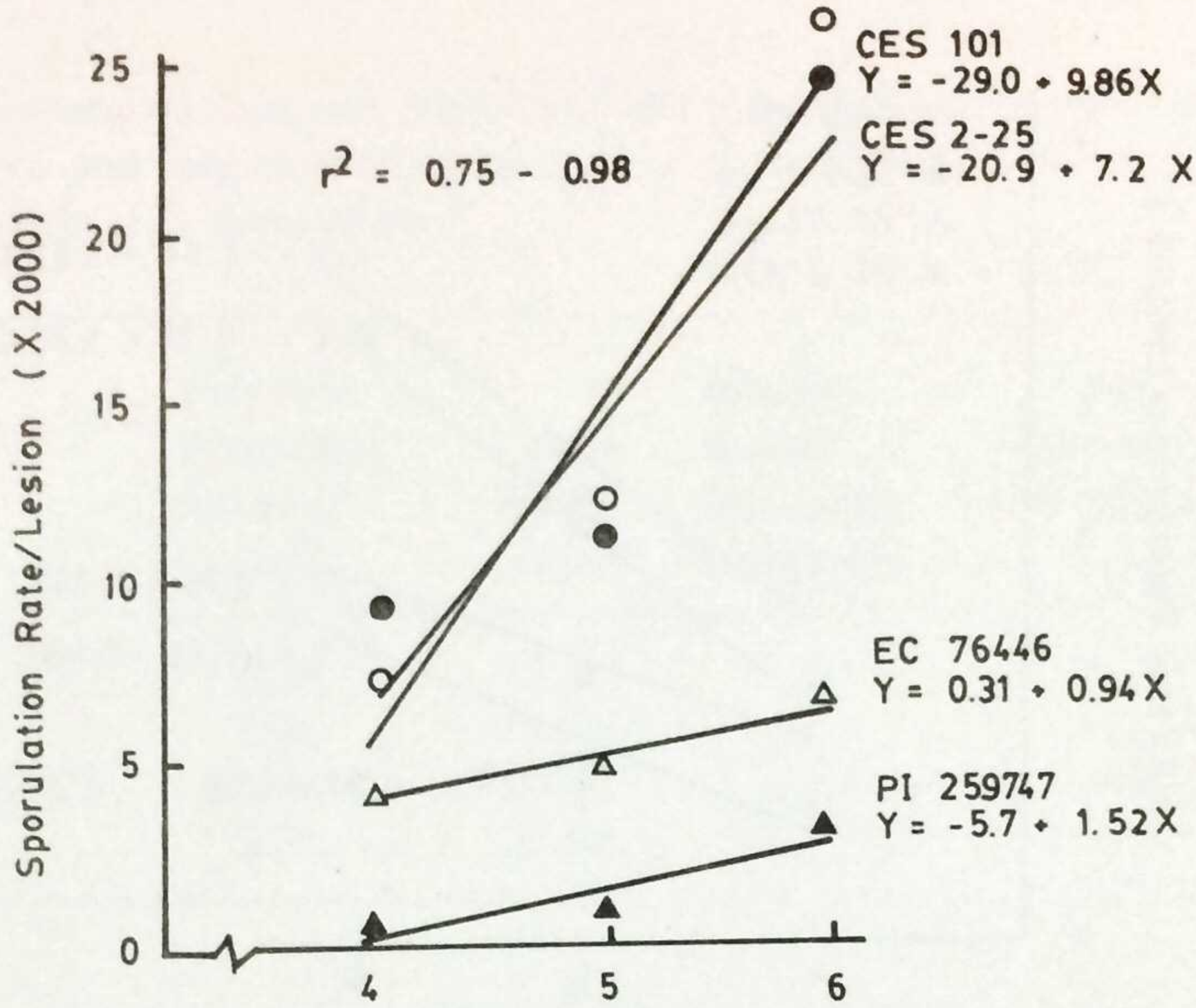

Figure 2. The comparative rates of sporulation of lesions in *C. personatum* — infected peanut cultivars.

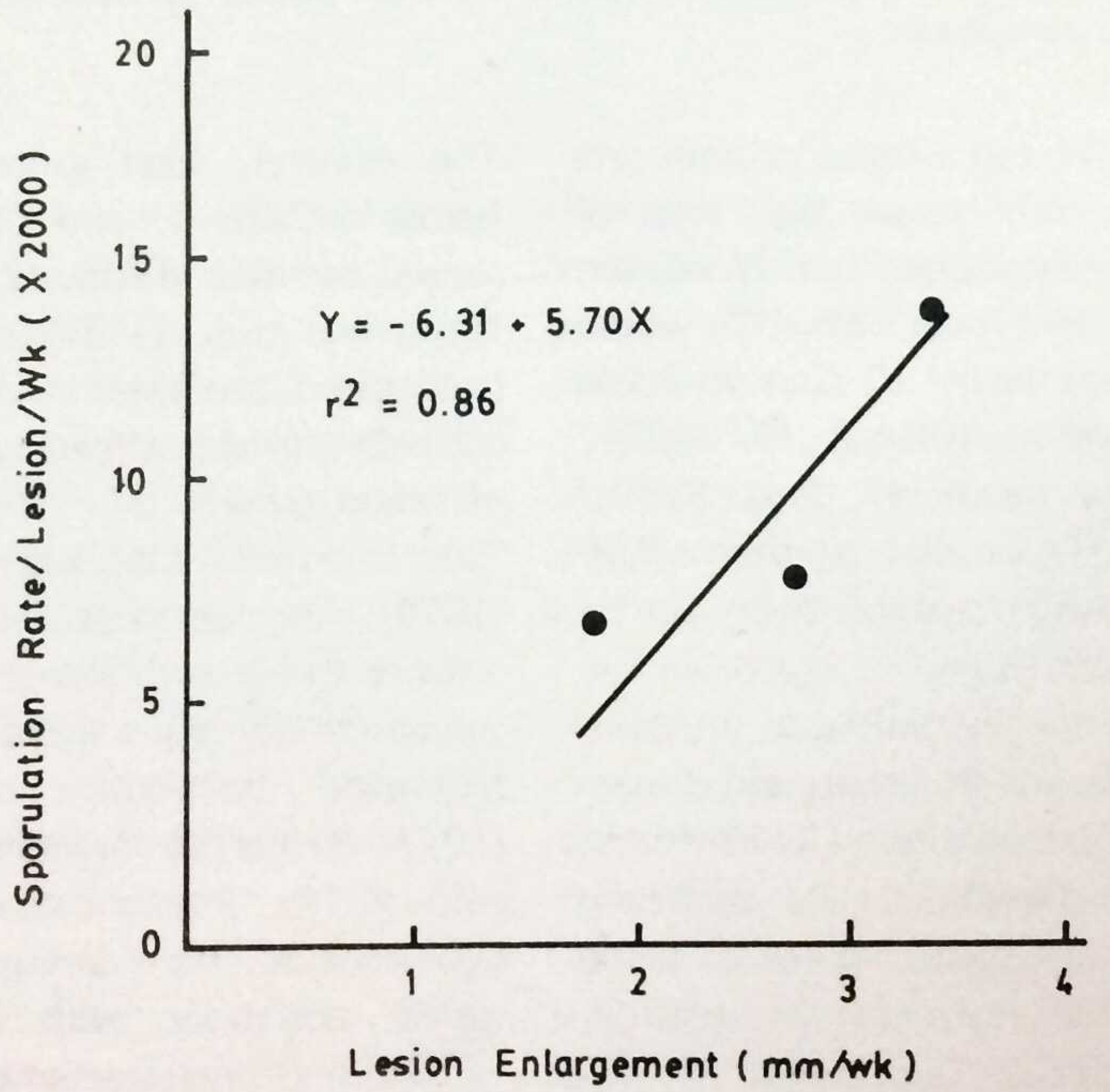

Figure 3. The relationship between the rate of lesion enlargement and the rate of spore production of *C. personatum* averaged across four peanut cultivars.

peanut varieties (Table 2). Decline in rates of lesion formation ranged from 30-85% relative to CES 101. PI 259747 and NC Acc 17133 (RF) both exhibited the slowest rate of lesion formation among the eight peanut varieties tested. Germination of and entry into leaflets of *C. personatum* conidia were similar in resistant and susceptible peanut genotypes (ICRISAT, 1981). Observations on controlled inoculations done by Paningbatan and Ilag (1984) and in this study support the findings of ICRISAT (1981). The significantly different rates of lesion formation might therefore reflect the inherent variations of peanut cultivars to resist the establishment of *C. personatum* after penetration. Future histopathological investigations would provide direct evidence to these differences.

The comparative infection rates of the pathogen on the eight peanut cultivars are presented in Table 3. CES 101 and UPL Pn4, two commercially grown cultivars, sustained relatively faster rates of infection than the other test cultivars. NC Acc 17133 (RF), a resistant variety, remarkably slowed down the rate of leaf spot epidemic by 40% relative to that of CES 101 (Fig. 4). The distinctly short latent period in CES 101 (Table 1)

Table 2. Mean number of lesions and rate of lesion formation due to *C. personatum* per fifth oldest leaf of each peanut cultivar.

Host Cultivar	Mean Number of Lesions — Weeks After Inoculation						Rate of Lesion Formation/ week[1]
	1	2	3	4	5	6	
CES 101	0	1.0	2.5	5.8	78.2	137.1	34.8a
BPI P9	0	1.0	2.3	5.3	65.7	87.1	24.0b
UPL Pn4	0	0.7	2.5	3.2	34.0	43.3	11.7f
CES 12-12	0	0.8	3.0	4.3	31.7	52.2	13.2e
CES 12-24	0	0.6	2.6	4.7	44.5	63.5	16.7d
CES 12-26	0	1.0	3.1	3.8	39.0	81.5	19.7c
PI 259747	0	0.1	0.8	1.7	6.1	22.5	5.0g
NC Acc 17133 (RF)	0	0.3	1.4	2.4	12.5	22.7	5.6g

[1]Values are based on simple regression coefficients of three replications; a replicate consisted of five random plants from a population of 55-60. In a column, values followed by a common letter are not significantly different at 1% level, DMRT.

Table 3. Proportion of diseased leaf area per plant and infection rates of *C. personatum* on peanut cultivars with different levels of resistance.

Host Cultivar	Weeks after Emergence				Infection Rate (r)[1]	Percent Reduction in r based on CES 101
	4	6	8	10		
CES 101	.018	.023	.057	.350	.55	—
BPI P9	.010	.013	.057	.100	.43	22
UPL Pn4	.012	.022	.040	.240	.52	5
PI 259747	.003	.004	.050	.013	.23	58
EC 76446 (292)	.003	.003	.006	.010	.22	60
NC Acc 17133 (RF)	.007	.007	.010	.015	.13	76
PI 350680	.003	.003	.005	.010	.21	62
CES 3-2	.008	.017	.057	.100	.45	18

[1] Regression coefficients based on simple linear regression analysis of logits of percent diseased leaf area with time.

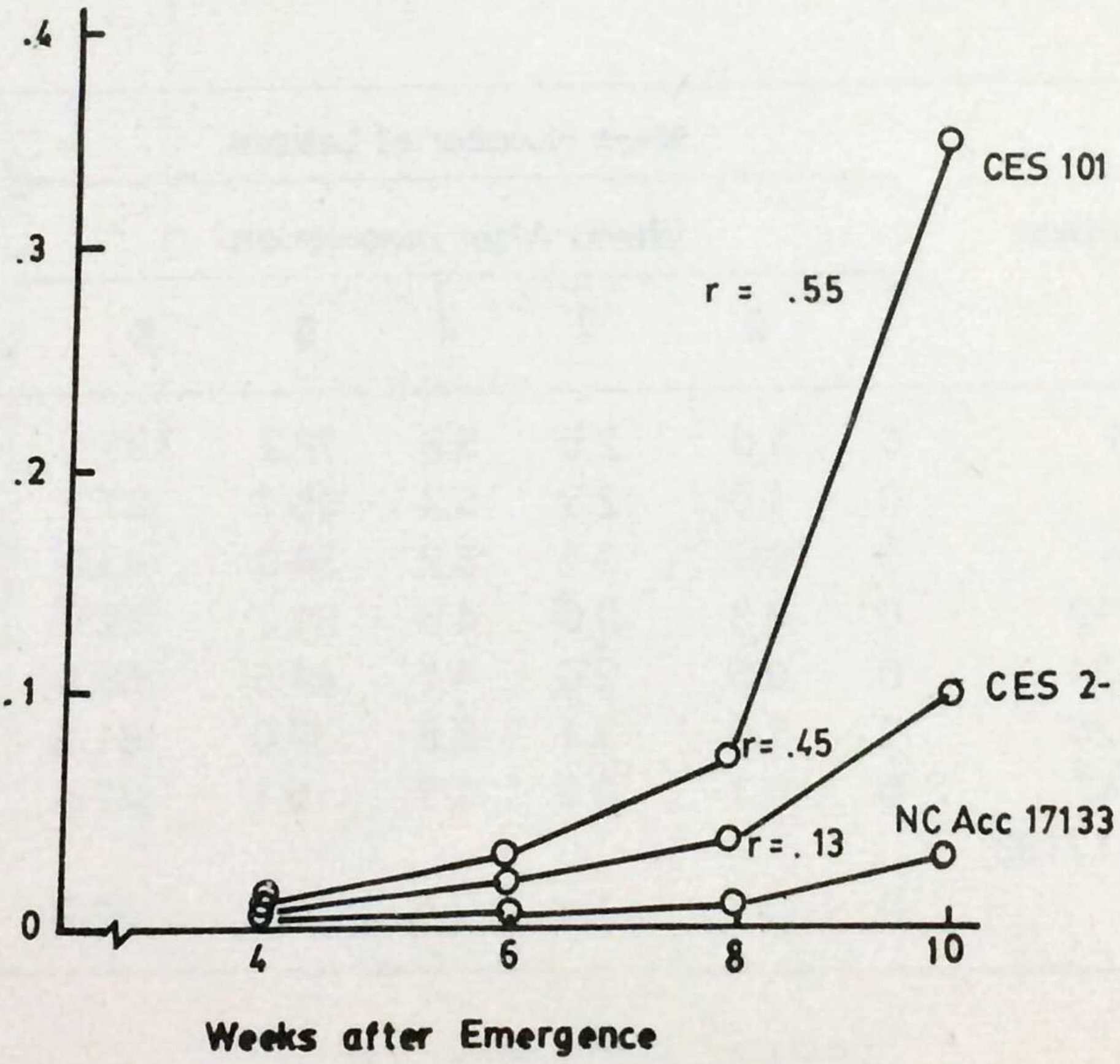

Figure 4. Comparative progress curves of *Cercospora* leaf spot as influenced by peanut cultivars grown in the field, r values are estimated rates of infection based on linear regression of logits of diseased proportion per plant with time.

could have enabled the pathogen to generate relatively more disease cycles than the resistant cultivars PI 259747, NC Acc 17133 (RF), and PI 350680. Since high inoculum levels and therefore high infection frequency shortened the period from inoculation to onset of sporulation (Paningbatan and Ilag, 1984) and with the relatively high rate of lesion formation (Table 2), the tremendous amount of spores produced by *C. personatum* in the susceptible cultivar must have generated more disease cycles during subsequent infections. These spores which were produced early should have contributed a greater positive effect on the leaf spot epidemic and might have accelerated disease development in susceptible cultivars like CES 101.

As the plot size used was virtually small, the differences in rates of disease development among varieties might even be more pronounced if plot interference was minimal. Parlevliet and van Ommeren (1977) reported that the progress of leaf rust epidemic due to *Puccinia hordei* in the most resistant and most susceptible barley cultivars differed by a factor of over 2,500. However, when the same cultivars were grown in small adjacent plots of four rows and one row wide, cultivar differences were reduced by 80-150 times.

The reduced rate of disease increase in peanut infected with *C. personatum* in the field may have resulted from the combined effects of various resistance characteristics of the cultivars on the different phases of pathogenesis. Such resistance in peanut is associated with prolonged latent period, and reduced rates of lesion enlargement, lesion formation and lesion sporulation. Since the degree of leaflet abscission is positively related to susceptibility as observed by Paningbatan and Ilag (1984), abscissed infected leaves might still sustain sporulation of the necrotrophic pathogen which further enhances the available inoculum for secondary disease cycles.

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