Seasonal Periodicity and Distribution of Bacterial Blight of Coffee in Kenya

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ABSTRACT

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The inherent growth and flowering rhythm of *Coffea arabica* trees, governed by the annual rainfall pattern, strongly influenced seasonal periodicity of bacterial blight, caused by *Pseudomonas syringae* pv. *garcae*. During extension growth and flowering, abundant avenues of infection and rainfall (duration and intensity) provided conditions for epiphytic growth, dispersal, entry, and internal spread of the pathogen. During reduced growth, avenues of infection and epiphytic inoculum became limiting, resulting in low disease incidence. Based on these findings, disease development in coffee-growing areas east and west of the Rift Valley of Africa is discussed.

Outbreaks of bacterial blight (Elgon-Solai dieback) of *Coffea arabica* L., caused by *Pseudomonas syringae* pv. *garcae* (4), show marked seasonal periodicity. The disease assumes epidemic proportions at Bahati/Solai in the Great Rift Valley of Africa and on the slopes of Mount Elgon, where annual rainfall is fairly uniformly distributed from March to September (1). East of the Rift Valley, the disease causes mild or insignificant damage to coffee in Mweiga, Nyeri, Thika, Ruiru, and Wundanyi, where rainfall is bimodally distributed (1), with the wettest periods from March to June (long rains) and October to December (short rains). We attempted to determine the potential for serious outbreaks of bacterial blight of coffee in areas east of the Rift Valley.

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MATERIALS AND METHODS

Inoculation experiments. Inoculation experiments were conducted in a field of young coffee plants (cultivar S.L. 28) at the National Agricultural Laboratories, Nairobi (lat 1°22′ S, long 36°49′ E), during the April 1979 rains. On each of 20 plants, the ventral, exposed surfaces of pairs of immature, expanding leaves enclosing the terminal buds of two opposite primary twigs with green, unopened flowers were gently rubbed with an aqueous bacterial suspension (10^6 viable cells per milliliter) of a leaf surface isolate of *P. syringae* pv. *garcae* (4).

Pathogen infestation was determined weekly on 10 randomly selected pairs of immature leaves and flower clusters on uninoculated primary twigs. Surface water trapped between immature leaves and in flower clusters was sucked by capillary action into tips of sterile Pasteur pipettes and streaked on nutrient 5% sucrose agar plates (4).

Field observations. Data were taken from four plots, each containing 20 unsprayed S.L. 28 trees, on an estate at

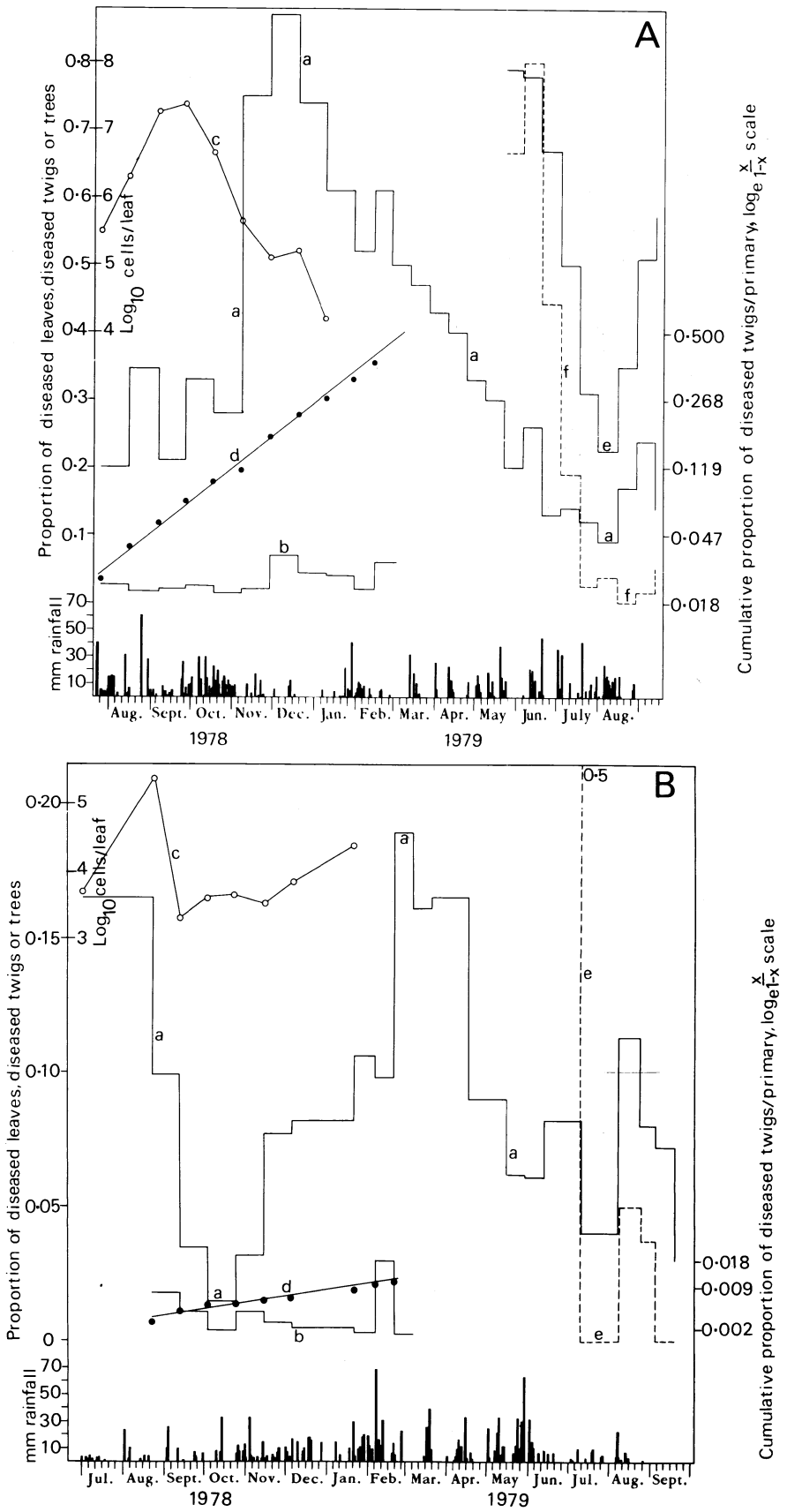

Fig. 1. Development of bacterial blight of coffee, caused by *Pseudomonas syringae* pv. *garcae*, in relation to tree flowering and rainfall. At Bahati, in the Rift Valley **(A)**, trees flowered in September–October 1978 and in July–August 1979. At Mweiga, east of the Rift Valley **(B)**, trees flowered in December–January. Trends in proportions of diseased leaves (a), numbers of newly diseased twigs per primary branch (b), $\log_{10}$ of pathogen cells per leaf (c), and proportions of trees with newly cankered or blighted twigs (e and f, respectively) are shown. Rate of increase of cumulative proportions of diseased twigs per primary branch on the $\log_e(x/(1-x))$scale (d) was 0.0989 per unit per week at Bahati and 0.0484 per unit per week at Mweiga. Incubation period was 3 wk and infectious period was 5 wk.

Bahati/Solai (lat 11′ S, long 36°8′ E) in the Rift Valley and an estate at Mweiga (lat 19′ S, long 36° 55′ E) east of the Rift Valley. Excess growth and diseased twigs were removed from these trees in June 1978 and June 1979. Every 2–3 wk, we recorded 1) numbers of newly diseased twigs on one marked primary branch per tree counted and tagged from June 1978 to February 1979; 2) numbers of newly diseased twigs per tree from May to September 1979; 3) numbers of spots per leaf from June 1978 to September 1979, on a minimum of 100 mature leaves per plot, picked randomly from the top and middle of the canopy; and 4) leaf surface populations of the pathogen, determined from June 1978 to February 1979 as described previously (4).

RESULTS

Inoculation experiments. Inoculated immature leaves at subapical (0) nodes on the 40 primary twigs developed water-soaked spots in 4–10 days. On four twigs, the infected immature leaves abscised before the 0 nodes became water-soaked. On only one of these four twigs did a canker develop (at the third node in September). This was precisely the 0 node on which immature leaves were inoculated 6 mo earlier in April. On nine twigs, such leaves grew to full size and developed necrotic spots with chlorotic halos (leaf spots) without water-soaking of the 0 nodes.

On the remaining 27 twigs, the 0 nodes became water-soaked (blighted twigs) over a period averaging 17 days from inoculation (latent period). Water-soaking on internodes 1 to 4 followed after an average 18, 30, 41, and 47 days, respectively. These internodes blackened in 8, 6, 16, and 5 days (infectious periods), respectively. During infectious periods, bacteria oozed from invaded tissue (4).

On uninoculated primary twigs, the 10 randomly selected immature leaf pairs and flower clusters became infested 2–4 wk after symptoms appeared on the inoculated primaries. Four to 10 days later, water-soaking was evident on some of these infested leaves and flowers. Bacteria entering nodes from infected flowers and pinheads caused cankers. Cankers and dieback rarely extended beyond the junction of green and yellow green bark. Cankers on woody tissue killed the proximal portion of twigs without enlarging.

Field observations and disease periodicity. The Bahati data, pooled from the four observation plots, were analyzed separately from similarly pooled Mweiga data. Because young diseased leaves on nodes 1 and 2 invariably had one spot each, leaf spots were counted on old leaves, where the vast majority of infections occurred along surface wounds. The mean number of diseased internodes per cankered or blighted twig was similar at both places soon after

maximum disease incidence (Table 1). The cumulative proportion of infected twigs per primary branch over time followed the exponential disease progress curve of van der Plank (5). The epidemic rate was 0.0989 per unit per week at Bahati and 0.0484 per unit per week at Mweiga (Fig. 1).

At all times, frequency distributions of spots per leaf followed a negative binomial series (Table 2). Frequency distributions of newly cankered twigs per tree followed such a series poorly and did not fit a Poisson series; their variances were significantly greater than their means. Frequency distributions of newly blighted twigs per tree followed a Poisson series (Table 2).

The proportion of diseased leaves and the proportion of newly infected twigs per primary branch were correlated (Fig. 1). The correlation coefficients during 1978 were 0.76 at Bahati and 0.83 at Mweiga. The proportion of diseased leaves was also directly related to the proportion of trees with newly blighted or cankered twigs from May to September 1979 (Fig. 1). Because of these relationships, disease periodicity was analyzed using incidence of leaf disease only.

At Bahati, three distinct disease periods could be discerned (Fig. 1A). From July to October 1978, proportions of diseased leaves fluctuated around a mean value of 0.27; in October–November 1978, disease rose dramatically to a maximum of 0.88; and from December 1978 to June 1979, disease declined steadily. These three periods occurred during the maximum extension growth, flowering, and reduced growth cycles, respectively, of coffee trees in and west of the Rift Valley (3).

At Mweiga (Fig. 1B), the proportion of diseased leaves did not rise suddenly, nor was a fluctuating period clearly evident. Proportions of diseased leaves rose steadily from 0.002 in October 1978 to 0.2 in March 1979. During this period, maximum extension growth was followed by flowering in January. Disease decline coincided with the reduced growth cycle of trees, which occurs during the cool, relatively dry season (June–September) east of the Rift Valley (3,6).

During the fluctuation and increase periods, the proportion of diseased leaves at any time was correlated with the product of the number of rainy days and the total rainfall in a 21-day period 3 wk before disease assessment (r = 0.97 at Bahati and 0.79 at Mweiga). During the decline period, this relationship could not be demonstrated because of natural leaf fall enhanced by the accelerated fall of diseased leaves. Indeed, during this period, disease peaks did not occur even after long periods of intense rainfall (Fig. 1). At Bahati, a second increase in cankered twigs occurred after the August–September flowering (Fig. 1A). At Mweiga, flowering did not occur during this period and twig infection was minimal or absent (Fig. 1B). At both places, leaf surface inoculum increased during the fluctuating disease period (Fig. 1).

DISCUSSION

Correlations between numbers of diseased twigs and leaves and their relationship to leaf surface populations of bacteria show the interdependence of the different disease phases. Clustering of leaf spots and cankered twigs, suggested by the fit of their frequency distributions to a negative binomial series, implies that the most important sources of epiphytic inoculum for an individual tree are found within the tree itself. Inoculum is spread by splashing rain (4). Thus, epidemics of bacterial blight of coffee are the sum of the separate outbreaks on individual trees acting largely as independent units.

Abscission of infected leaves, the greater viability of bacteria in old infections on woody tissue than on succulent tissue, and the reactivation of bacteria following showers (4) suggest that residual cankers and lesions on woody tissue of twigs are the most important sources of epiphytic inoculum. Demonstrating this relationship is difficult because of epiphytic bacterial growth, leaching caused by rain, and death during dry periods. Crosse (2) reached a similar conclusion with *P. mors-prunorum*.

The inherent growth and flowering rhythm of trees determined by the annual rainfall distribution pattern (3,6) strongly influenced seasonal disease periodicity. Disease fluctuation, epiphytic inoculum increase, and subsequent disease increase, which occurred during maximum extension growth and flowering of trees, were determined by the intensity and duration of rainfall. The sequential relationships among inoculum increase, rainfall peaks, and disease peaks indicate the existence of dispersal periods. The lack of disease peaks after dispersal periods during disease decline could be attributed to the relative scarcity of epiphytic inoculum and avenues of infection brought about by natural leaf fall, fewer new surfaces for infestation and epiphytic growth (2,4), and scarcity of actively growing tips, flowers, and pinheads.

Differences in isolates of the pathogen cannot account for the markedly lower disease incidence and epidemic rate at Mweiga (east of the Rift Valley) compared with Bahati (in the Rift Valley) (4). Similarity in the susceptibility of trees at both places, indicated by the approximately equal numbers of invaded internodes per diseased twig, and similarity in total annual rainfall also exclude these variables as causal factors.

For disease to be sustained, passive entry of the bacterial pathogen into the main avenues for twig infection and its internal spread require a combination of large amounts of epiphytic inoculum, numerous avenues of infection, and prolonged rains. These conditions are provided at Bahati and areas west of the Rift Valley by the long maximum growth and flowering periods brought about by fairly uniformly distributed rainfall from

Table 1. Distributions of numbers of invaded internodes on coffee twigs infected by *Pseudomonas syringae* pv. *garcae* at Mweiga and Bahati, Kenya[a]

	Number of internodes invaded												
Site	1	2	3	4	5	6	7	8	9	10	11	12	Mean[b]
Mweiga	1[c]	5	4	1	5	5	3	4	4	4	1	...	6.0
Bahati	1	7	5	10	4	7	7	4	5	2	2	1	5.7

[a] Recorded in March 1979, after maximum disease incidence.
[b] Mean number of internodes invaded.
[c] Number of diseased twigs with 1, 2, 3 . . . 12 invaded internodes.

Table 2. Distributions of bacterial blight symptoms caused by *Pseudomonas syringae* pv. *garcae* on coffee trees at Bahati, Kenya[a]

		Number of leaf spots or diseased twigs										Chi-squared statistic
Symptom	Frequency	0	1	2	3	4	5	6	7	8	9	10	
Leaf spots	Observed	50	72	69	64	42	30	25	18	12	8	12	...
	Estimated	52.595	71.090	68.869	57.927	45.027	33.275	23.741	16.506	11.250	7.548	14.172	1.94
Cankered twigs	Observed	18	17	14	7	24	...	...	...	...	...	...	...
	Estimated	14.929	20.538	15.969	9.233	19.331	...	...	...	...	...	...	3.15
Blighted twigs	Observed	16	27	16	12	9	...	...	...	...	...	...	...
	Estimated	13.04	23.68	21.44	12.96	8.88	...	...	...	...	...	...	2.59

[a] Negative binomial distributions fitted to observed frequencies of leaf spots and cankered twigs on trees. Poisson distribution fitted to observed frequency of blighted twigs on trees. These distributions were followed at all times. The Mweig data were similar.

March to September (2). At Mweiga, which lies just inside the bimodal annual rainfall area, about 100 km from Bahati, maximum growth and flowering occur mainly in flushes early during the long and short rains (3,6). However, sporadic rains from June to September provide the conditions for mild outbreaks. Further southeast, pronounced bimodal annual rainfall precludes significant outbreaks.

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