Analysis of a Maple Canker Epidemic in Pennsylvania

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ABSTRACT

A sugar maple canker epidemic in Pennsylvania was analyzed in 1967 using van der Plank's techniques. The disease is induced by several species of *Fusarium*, primarily *F. solani*. Data were collected from 30 trees having more than 3,100 cankers. The frequency of cankering for each tree was recorded by tree face, height of occurrence on the stem, and by year of initiation. There was an inverse relationship between frequency of cankering and height of occurrence, but no difference in frequency of cankering among tree faces. Peak periods of cankering were 1950, 1952, 1957, and 1961-63. The frequency of disease began to increase logarithmically in the late 1930's and to decrease in the early 1960's. During the period of most rapid rate increase of the epidemic, disease incidence was doubling every 4 yr.

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An annual canker of sugar maple (*Acer saccharum* Marsh.) initially was reported in Pennsylvania by Stambaugh and Nichols in 1960 (13). Since then, many other similar annual cankers of hardwoods have been reported (2, 3, 4, 8, 11, 14, 15, 16, 17) and considerable effort has been made to determine their cause or causes and to relate the diseases to various climatic and edaphic factors. Skelly and Wood (12) determined that cankers of sugar maples originated during the dormant season of the host. The frequency of cankering varied inversely with height aboveground, and the cankers were more numerous on the north and south faces of trees.

Before control of any plant disease becomes feasible, it is imperative that as much as possible be learned about its etiology and epidemiology. Epidemics of several diseases have been analyzed by van der Plank (18). The techniques used by van der Plank are of relatively recent origin, although earlier thought had been given to the mathematical approach to studying epidemics (4, 6, 7). His method is based partially on the premise that the rate of disease increases logarithmically early in an epidemic. Merrill (9, 10) was among the first to employ the techniques of van der Plank in critically analyzing disease epidemics of forest and shade trees, although van der Plank had calculated r_1 values for oak wilt (18).

This study was begun in June of 1967 to determine the rate of development of a sugar maple canker epidemic in Pennsylvania.

MATERIALS AND METHODS.—Our study was conducted between June and December, 1967 in three widely separated Pennsylvania counties. Two stands were sampled in Bedford and Pike Counties and one in Potter County. Six severely-cankered trees, as determined from outward appearance, were selected in each stand. They ranged from 25.4 to 38.1 cm diam at breast height (dbh). The trees were felled and 5-cm-thick disks were removed every 0.61 m up the bole to a height of 15.2 m. The cardinal compass headings were marked on each disk along with the height from which it was removed, implementing much the same technique used by Skelly and Wood (12). Each disk surface was examined under a dissecting microscope and all cankers were dated. The frequency of cankering was recorded by face, height of occurrence on the stem, and year of initiation for each tree. These data were used in analyzing the epidemic.

Following van der Plank (18), the basic formula applied in this study was:

$$r_1 = \frac{2.3}{t_2 - t_1} \log_{10} \frac{x_2}{x_1}$$

The term r_1 is defined as the rate at which the population of the pathogen or amount of disease increases during the logarithmic phase of the epidemic; t_2-t_1 is the time interval between observations of a given amount of disease (x_1) at t_1, and (x_2) at t_2. In this study, time is expressed in years

and the amount of diseased tissue (x) as the cumulative number of cankers (all stands combined) during a given year (t). An underestimate of the rate of development of the epidemic can result when more than 5% of the tissue available has become infected as r_l decreases rapidly when more tissue becomes infected. A correction factor was applied to compensate for the decrease in the remaining noninfected tissue. The amount of diseased tissue is represented by x, and the remaining susceptible tissue by (1-x). It was obvious to us from field observations, that x was smaller than 5% and in such cases, according to van der Plank (18), $\log_{10} 1/(1-x)$ can be set equal to $\log_{10}x$. Our suscept population was considered infinite and r_l was expressed as "per unit per year".

Values of r_l were calculated from year to year from 1922 to 1966, and for various time periods throughout the epidemic. The course of the epidemic was graphed and its sigmoid-shaped curve is presented in Fig. 2-C. By logit transformation the course of the epidemic also was plotted as a straight line, plotting $\log_{10}x$ against time.

RESULTS.—A total of 3,115 cankers were studied on 30 trees in the five stands examined in Bedford, Pike, and Potter Counties. There was an inverse relationship between frequency of cankering and height of occurrence (Fig. 1). Peak periods of cankering occurred in 1950, 1952, 1957, and 1961-1963 (Fig. 2-A). There was no significant difference in frequency of cankering among tree faces (Table 1) as determined by an analysis of variance.

The rate of the epidemic began to increase in the late 1930's and to decrease in the early 1960's (Fig. 2-A). This can be seen also in Fig. 2-C which shows a rapid increase in disease severity between 1940 and 1957. The logarithmic increase may be visualized better by plotting $\log_{10}x$ against time (Fig. 2-B). The curve represents the average rate of the epidemic; the slope of the curve at any given point is equal to r_l. Until 1957, the epidemic increased in intensity at an increasing rate; the rate began

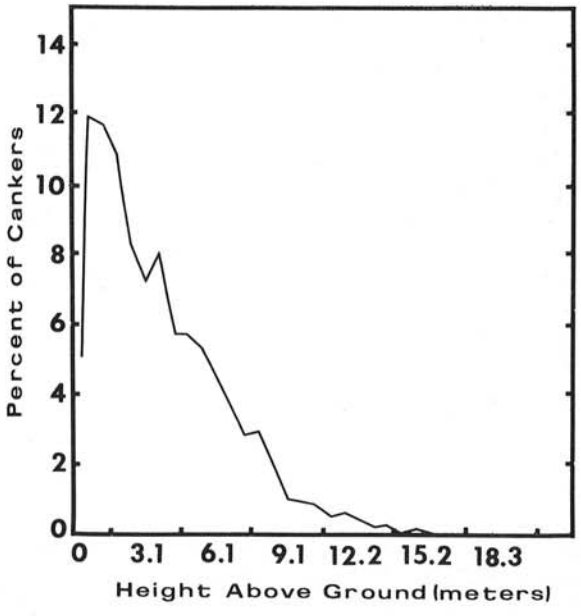

Fig. 1. Relationship between frequency of cankering in maple and height aboveground.

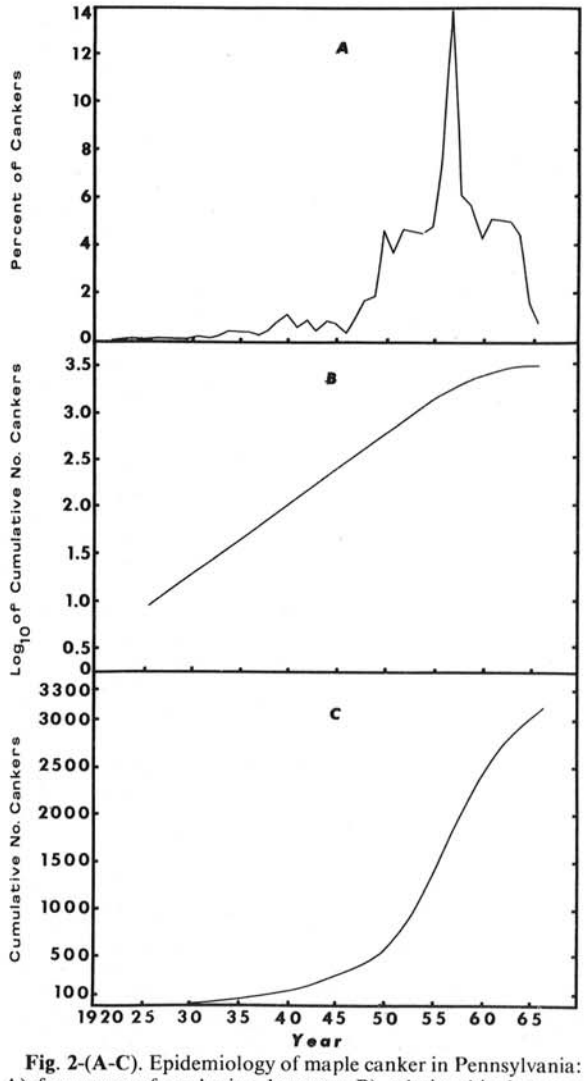

Fig. 2-(A-C). Epidemiology of maple canker in Pennsylvania: **A)** frequency of cankering by year; **B)** relationship between logarithmic increase in disease and time; and **C)** relationship of canker frequency to year.

TABLE 1. Frequency of occurrence of maple cankers by tree face

Stand location	No. of cankers on side of tree facing				
	North	East	South	West	Total
	number				
Pike County, No. 1	160	138	122	156	576
Pike County, No. 2	178	167	223	190	758
Potter County	119	97	156	133	505
Bedford County No. 1	200	189	177	143	709
Bedford County No. 2	141	121	156	149	567
Totals	798	712	834	771	3,115

TABLE 2. Rates of disease increase (r_l) for individual years

Year	Rate of disease increase (r_l)	-Fold increase in number of cankers per year	Year	Rate of disease increase (r_l)	-Fold increase in number of cankers per year
1924	0.80	2.23	1946	0.05	1.05
1925	0.34	1.40	1947	0.11	1.12
1926	0.13	1.14	1948	0.17	1.18
1928	0.16	1.17	1949	0.16	1.17
1929	0.24	1.27	1950	0.31	1.36
1930	0.07	1.07	1951	0.19	1.21
1931	0.29	1.34	1952	0.02	1.02
1932	0.05	1.05	1953	0.16	1.17
1933	0.25	1.28	1954	0.14	1.15
1934	0.39	1.48	1955	0.13	1.14
1935	0.24	1.27	1956	0.18	1.20
1936	0.21	1.23	1957	0.26	1.30
1937	0.10	1.12	1958	0.10	1.10
1938	0.16	1.17	1959	0.08	1.08
1939	0.27	1.31	1960	0.06	1.06
1940	0.28	1.32	1961	0.06	1.06
1941	0.16	1.17	1962	0.06	1.06
1942	0.16	1.17	1963	0.06	1.05
1943	0.07	1.07	1964	0.05	1.05
1944	0.12	1.13	1965	0.02	1.02
1945	0.09	1.09	1966	0.01	1.01

TABLE 3. Values of r_l for selected time periods during the epidemic

Time period	Rate of disease increase (r_l)	-Fold increase in number of cankers per year
1922-1932	0.30	1.35
1932-1946	0.18	1.20
1946-1957	0.18	1.20
1957-1966	0.05	1.05
1928-1941	0.21	1.23
1941-1948	0.11	1.12
1948-1953	0.20	1.23
1922-1946	0.23	1.26
1922-1957	0.22	1.25
1922-1966	0.18	1.20

to decrease slightly after 1957. Table 2 is a record of the progress of the epidemic from year to year in terms of calculated r_l values. Table 3 shows the progress during certain periods of the epidemic. These periods were selected because they appeared to represent different trends in disease development. For example, during the logarithmic phase between 1922 and 1957, disease severity was increasing at the rate of 1.25-fold per year; i.e. the amount of disease was doubling approximately every 4 yr. In contrast, 18 yr would have been required to double the amount of disease at the rate of canker accumulation during the early 1960's.

DISCUSSION.—The increase of disease incidence from year to year was logarithmic. Since cankers form only during the dormant season and throughout much of this period weather conditions are unfavorable for the development of the pathogen, it is not likely that the pathogen multiplies through successive generations and that the inoculum produced on one plant infects another in the same season. Sugar maple canker is characteristic, therefore, of a simple-interest type disease (18). The r values of the Fusarium canker epidemic were generally low, ranging from 0.05 to 0.30 for various periods, suggesting that the pathogen is probably indigenous.

We believe the Fusarium canker epidemic in Pennsylvania is subsiding. This conclusion is substantiated by the decreasing r values obtained during the last nine years (1958-1966) that the epidemic was examined. Different reasons have been postulated for that phenomenon. Jensen and Rostrup (5) recognized a logarithmic increase in the production of inoculum by *Phytophthora infestans* (Mont.) de Bary and hypothesized that the amount of infected foliage would be equal to the logarithmic increase in inoculum multiplied by a constant representing the amount of inoculum before any time period. Large (6), recognizing that an epidemic could be halted by a lack of tissue available for infection, stated that this relationship holds good only as long as total foliage area remains infinitely great in relation to infected area. Van der Plank (18) suggested that epidemics usually subside due to lack of availability of susceptible tissue.

The decreasing r values during the later years of the epidemic indicate either that a high degree of non-uniformity of susceptibility exists within the stands (18), or that conditions became less favorable for infection after 1957. Obviously an epidemic will subside if little or no susceptible tissue remains to be infected, but how does one explain the subsidance of an epidemic when a high proportion of the suscept population remained free of disease? The availability of susceptible tissue does not

appear to be the limiting factor in this case; more tissue was available on a given tree during 1960 than during 1940.

Fusarium solani has been shown to occur in the bark of healthy sugar maple trees (20). Wounds in the bark, possibly to the inner phloem and cambium, are probably required for colonization by this pathogen. Since the amount of "susceptible" tissue has probably not decreased in the last 20 yr, the decline in the rate of development of the epidemic during recent years could be due to the unavailability of suitable wounds. On the other hand, changes in bark characteristics associated with age may result in decreased susceptibility of the lower stem, perhaps resulting in a reduction in the amount of "susceptible" tissue. Since moist conditions favoring infection on tree stems tend to decrease as trees grow taller and canopies become denser and higher, there could be a considerable influence exerted on the inoculum by such microclimatic changes at potential infection sites. Insects have not been associated with the disease and it is likely that factors such as wind stress or sudden drops in bark temp during late fall or early winter may be responsible for the occurrence of compatible wounds (1). Countywide precipitation was not strongly associated with the epidemic (19).

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