Similarity of Annual Anthracnose Epidemics in Young *Juglans nigra* Plantations from 1978 Through 1982

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ABSTRACT

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During 5 yr of observations (1978 through 1982) of walnut anthracnose development in young black walnut plantations, initial lesions arising from infections by ascospores always appeared in May. Lesion numbers increased through early summer as a result of secondary leaf infection by conidia. Disease development was maximal in late July and early August, when most defoliation of previously infected leaflets occurred. Leaf wetness periods of 12 hr or more, suitable for leaf infection, occurred with enough consistency each summer to perpetuate yearly anthracnose epidemics in similar fashions.

Additional key words: *Gnomonia leptostyla*

In the eastern United States, millions of seedlings of black walnut (*Juglans nigra* L.) have been produced and distributed for planting in the last two decades (1). Many of these seedlings have been planted at close spacing in pure plantations that provide a microclimate favorable for leaf diseases such as anthracnose caused by the fungus *Gnomonia leptostyla* (Fr.) Ces. & de Not. Premature defoliation by anthracnose often results in reduced tree growth and development of poorly filled, low-quality nuts (2).

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Although epidemics of walnut anthracnose have been associated with wet growing seasons (3,6,11), in southern Illinois, there was no relation between times of infection and rainfall occurrences. Each year, a regular pattern of disease development occurred, culminating in defoliation in late July or early August. These anthracnose defoliations were maximal when only 60% of the walnut growing season had passed.

Black (4) and Black and Neely (5) found that leaf infection by conidia could take place if leaves were wet for at least 12 hr at temperatures of 10 and 21 C. Little conidial infection occurred at high temperature (32 C), no matter how long leaves remained wet. In this study, Black and Neely's period of 12 hr and a somewhat arbitrarily selected temperature range of 10–27 C are used to explore the relation between leaf wetness periods and epidemic development.

MATERIALS AND METHODS

Durations of leaf wetness were determined with Belfort recording dew balances (Hiltner type) in conjunction with occasional hand checking of foliage for surface wetness. Concurrent temperatures, relative humidities, and rainfall were measured with hygrothermographs and rain gauges situated close to the dew balances.

A 7-yr-old black walnut plantation near Carbondale, IL, with a history of annual anthracnose defoliation was selected in 1978 for detailed observations. The trees in this plantation were composed of five "wild" provenances originating in Kansas, Oklahoma, Illinois, North Carolina, and Virginia. An account of this plantation is provided by Funk et al (7). Three other anthracnose-affected plantations in southern Illinois were observed in less detail to verify conclusions derived from the principal study plantation. In all of these plantations, anthracnose was the only defoliating disease present. Defoliation by insects was of little consequence in these plantations and did not interfere with anthracnose measurements. One hundred twenty leaf clusters (one per tree) throughout the primary plantation were selected early in the growing season before appearance of the first anthracnose lesions. Leaves and leaflets in each cluster were counted. At weekly intervals, the marked leaf clusters were reexamined and lesions and numbers of defoliated leaves and leaflets recorded.

RESULTS AND DISCUSSION

Appearance of first lesions. Primary lesions caused by ascospores (2,10) were first seen about the time the earliest leaves had reached maximum size. In the 5 yr of the study, this ranged from 13 to 22 May (Table 1). Primary lesions were generally well distributed throughout the crown

and provided numerous foci for secondary lesion development by conidial infections. There was a latent period of 1–2 wk before conidia were disseminated from acervuli produced on the primary lesions. Thus, secondary infection cycles initiated by conidia could begin as soon as late May or early June under leaf wetness conditions favorable for spore dissemination and leaf infection.

Table 1. Dates when first lesions appeared on young walnut leaves and when crown was 50% defoliated

Year	First lesions	50% Defoliation
1982	13 May	1 August
1981	20 May	31 July
1980	19 May	30 July
1979	21 May	1 August
1978	22 May	Not recorded

Secondary cycles of leaf infection. Under optimal conditions, lesion numbers can escalate rapidly (Fig. 1), but they are not reliable for following disease development; the total number of lesions on attached leaves may decline as heavily infected leaflets abscise (Fig. 1). Although some infected leaflets abscised early in the growing season, most abscised in midseason, typically late July and early August (Table 1). For example, in 1982, 45% of the leaflets abscised between 29 July and 6 August (Fig. 1).

When duration of leaf wetness (probably the key factor, in conjunction with temperature, to be considered in walnut anthracnose epidemiology [4]) was evaluated weekly, there were few weeks without at least one leaf wetness period of 12 hr, which is sufficient for infection to take place (Fig. 2). Of the five growing seasons from 1978 through 1982, there were three (1979, 1980, and 1981) when every week had at least one 12-hr leaf wetness interval. In 1978, there were two weeks with maximum leaf wetness periods of only 11 hr, whereas in 1982, there was one week with a maximum leaf wetness period of 11 hr and another with a maximum leaf wetness period of 10 hr. None of these maximal times occurred in consecutive weeks. In the 5-yr study (71 wk of data collection), the longest leaf wetness periods were caused by dew (24 wk) and rain (47 wk) (Fig. 2). Thus, for about one-third of the weekly intervals, dew deposition was responsible for the longest wetness periods. This was especially true with the low-rainfall 1980 season, when maximum wetness periods associated with dew occurred in 10 of 14 wk (Fig. 2). Table 2 shows some instances of increase in lesion numbers probably attributable to infections during periods of dew-caused leaf wetness in 1980. Most dew-associated increase in lesions occurred in leaflets already possessing one or more lesions. Apparently, conidial dissemination took place within dew moisture films on leaflets.

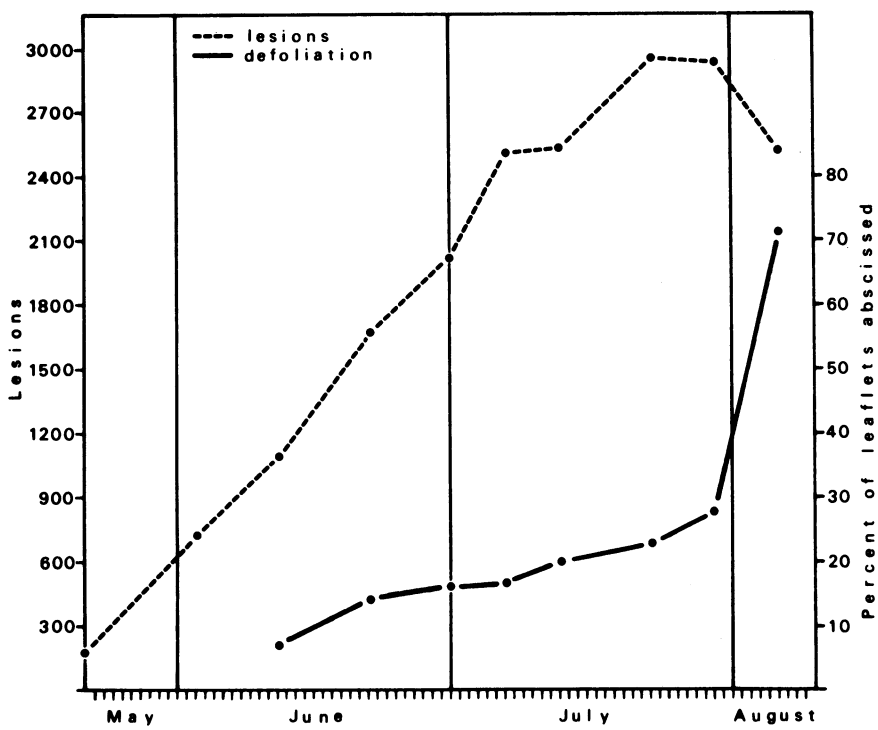

Fig. 1. Number of lesions and amount of crown defoliation during 1982.

Although leaf wetness caused by dew may allow conidial germination and infection to take place, conidial dissemination from leaflet to leaflet during these periods was uncertain. Black (4) noted that spores were liberated from acervuli into stationary water droplets placed on lesions during in vitro studies (4). A 30-km/hr wind failed to dislodge the droplets from horizontal leaf surfaces. In nature, where many leaflets grow at varying angles, dew tends to collect at edges and tips of walnut leaflets. Occasional nighttime and early morning wind gusts can cause droplets of dew to fall from leaflets; anthracnose conidia were found in these. Some within-tree-crown spread of conidia may occur through falling or windblown dew droplets as occurs with conidia of *Mycosphaerella musicola* on banana (8) and *Colletotrichum gloeosporioides* on rubber (12).

Many infected leaflets harbored populations of aphids and mites. Whether Arthropod vectors could also play a role in secondary conidial spread within tree crowns seems to warrant further study.

The hydrophilic properties of honeydew excreted by aphids and other sucking insects onto leaves influences moisture condensation on them. On alfalfa leaves, presence of aphid honeydew increased duration of leaf wetness by 25% or more (13). During drought, honeydew accumulates on leaves because it is not removed by rainfall. At these times, the effect of honeydew on the length of dew-caused leaf wetness periods probably is maximal.

For improved predictions of probability of leaf infection, Black and Neely's (5) study of the effects of temperature and duration of leaf wetness on infection needs to be expanded beyond the three

Table 2. Examples of anthracnose increase (lesion numbers) during the dry summer of 1980 attributable to dew-caused leaf wetness

	Ensuing period without rainfall			Lesions		
Rainfall occurrence	Date	Number of days	Number of nights with dew	Date[a]	Number/ leaflet	Increase attributable to dew
7 June				23 June	2.64	
	8–22 June	15	8	30 June	3.46	+0.82
2–3 July				21 July	4.16	
	4–20 July	17	14	28 July	5.19	+1.03
1 August				15 August	3.23	
	2–14 August	13	7	22 August	3.90	+0.67
15–16 August				1 September	3.16	
	17–28 August	12	6	8 September	3.63	+0.47

[a] Dates selected to allow for lag between inoculation opportunity provided by leaf wetness and subsequent appearance of new lesions.

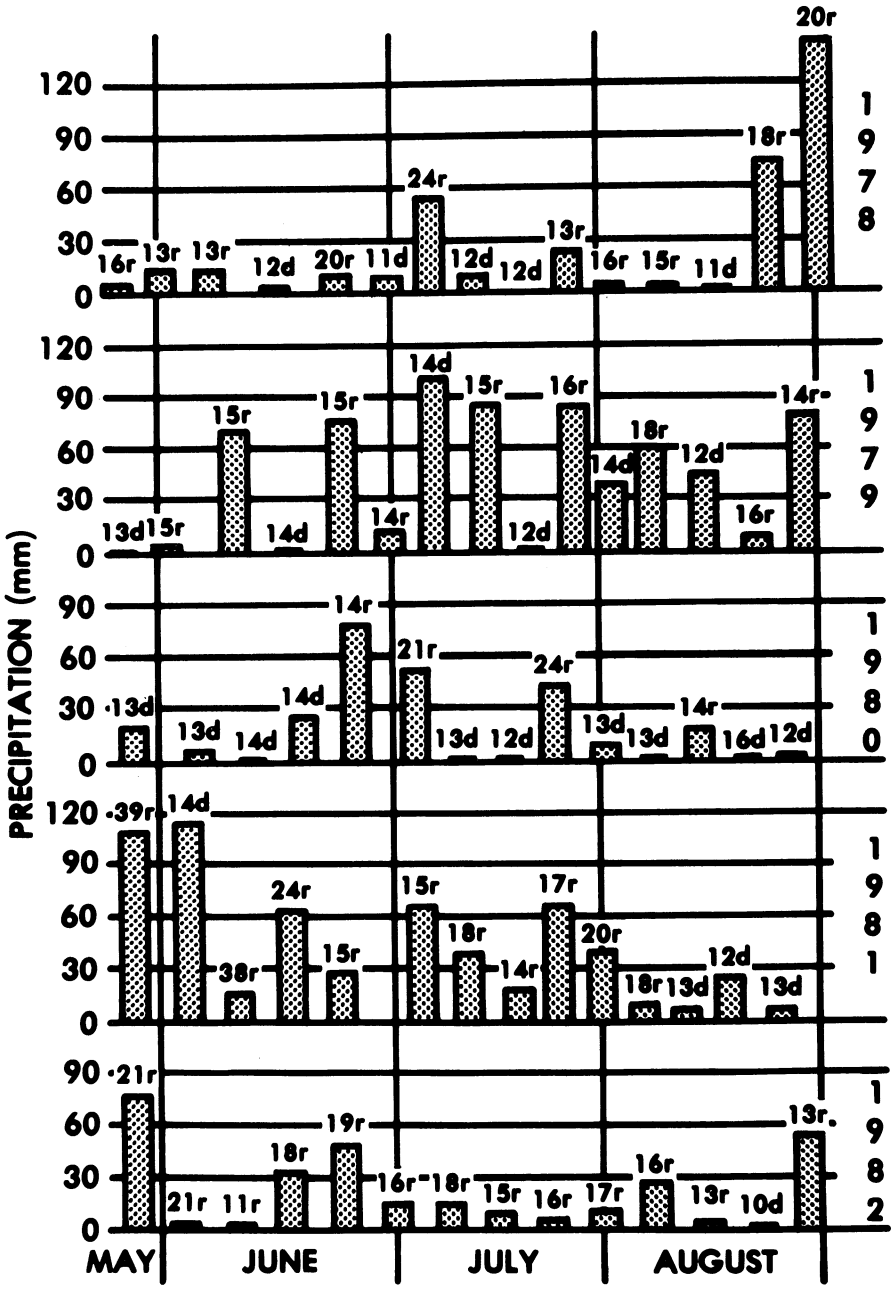

Fig. 2. Maximum consecutive hours of leaf wetness and total precipitation per week during anthracnose secondary cycles for seasons of 1978 through 1982. r = Rainfall and d = dew.

temperatures (10, 21, and 32 C) that were tested. The upper temperature limit at which significant infection levels can be achieved especially needs to be defined because Matteoni (9) has shown that growth and asexual sporulation of *G. leptostyla* are greatly reduced at 30 C.

There are probably two reasons anthracnose epidemics peaked at about the same time each year of our study. First, timing of release of ascospores, the primary inoculum to initiate the epidemics, seemed closely related to leaf morphology. From 1978 through 1982, fully enlarged leaflets susceptible to infection by ascospores occurred in mid-May of each year. Although primary lesions were always few, they were distributed evenly over the foliage in position for subsequent secondary pathogen spread within the crowns.

Second, infected leaflets bearing fertile acervuli remain on the trees early in the growing season (Fig. 1). These spore-bearing leaflets provide a continuing and enlarging inoculum supply for secondary infections to occur when leaf wetness periods caused by rain and dew last long enough for conidial germination and germ tube ingression into the leaf. Midsummer peak defoliation occurred when lesions became numerous enough to disrupt normal leaf physiology and initiate abscission.

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