Oak Wilt Foci Related to Ridge Bearing and Aspect in Pennsylvania

K. L. BOWEN, Undergraduate Student, and W. MERRILL, Professor, Department of Plant Pathology, Pennsylvania State University, University Park 16802

ABSTRACT

Bowen, K. L., and Merrill, W. 1982. Oak wilt foci related to ridge bearing and aspect in Pennsylvania. Plant Disease 66:137-139.

In the ridge and valley area of south central Pennsylvania, most oak wilt infection centers were on the upper slopes and tops of ridges bearing less than 045° (ridges more or less perpendicular to prevailing west-southwest winds) and were equally distributed on northwest and southeast sides of ridges bearing more than 045° (ridges more or less parallel to prevailing winds). All data on establishment of new infection centers resulting from long-range dissemination were consistent with the behavior of a windborne vector.

Oak wilt (OW), caused by *Ceratocystis fagacearum* (Bretz) Hunt, was first found in Pennsylvania in 1950. At that time and through the early 1960s, OW was considered a serious threat to oak forests (7,10). However, beginning in 1967, disease incidence in Pennsylvania rapidly subsided and the statewide control program was terminated in 1972.

Although various types of insects may be involved in short-range dissemination of the pathogen and in the increase in size of existing infection centers (16), oak bark beetles (*Pseudopityophthorus* sp.) appear to be the major vectors involved in long-range dissemination and in the creation of new infection foci (13). These beetles breed in and acquire the pathogen from diseased trees and can inoculate healthy trees while feeding (13). They occur above the forest canopy (11) and thus could be windborne for a considerable distance.

Information on patterns of long-range spread of OW is conflicting. Henry et al (5) found that initially infected trees frequently occurred at the edges of woodlands in Wisconsin, Minnesota, Iowa, and Illinois. In contrast, Brinkman (2) in Iowa and Riker (14) in Wisconsin found that initial infections often occurred in dominant trees within stands. In Pennsylvania, Hutnik et al (6) and Wilhour (17) found more OW infection centers on westerly aspects and on the upper slopes and tops of ridges. However, although Popp (12) in Pennsylvania and Cones (3) in West Virginia found more infection centers on the upper slopes and ridgetops, they found no relationship to aspect. Nevertheless, distribution of OW infection centers in south central Pennsylvania suggested that disease incidence was, indeed, related to aspect. The following studies were done to explore this relationship further. A preliminary report has been published (1).

Contribution 2272, Department of Plant Pathology, Pennsylvania Agricultural Experiment Station. Approved for publication as Journal Series Paper 6049.

Accepted for publication 5 May 1981.

0191-2917/82/02013703/$03.00/0

MATERIALS AND METHODS

As part of a joint state and federal Oak Wilt Post-Control Appraisal Study, the Pennsylvania Department of Agriculture made regular OW surveys in south central Pennsylvania (Bedford, Franklin, Fulton, Huntingdon, and Juniata counties). Between 1964 and 1970, more than 350 OW infection centers were found in three study blocks in these counties. The topography of this area consists of a series of nearly parallel mountain ridges and intervening valleys of various widths. At the southern border of the state, the ridges bear nearly north-south; they turn gradually east-northeast to bear about 060° near the center of the state. Records on each of these sites include, among other items, latitude, longitude, elevation, aspect of slope, ridge bearing, and date of discovery and treatment, plus a general site description and a sketch of the site.

From these records, each site was plotted on a U.S. Geological Survey 7.5-minute topographic map. Measurements of these mapped locations were made to determine the ridge bearing (degrees of deviation from north as determined by the ridge line), the bearing of the site to the ridge (counterclockwise deviation from north of a perpendicular from the ridge line to the site), and the exposure of the site.

Initially, computer correlations were made to find possible relationships that would imply a pattern of spread of OW. Graphs of some of the data were also made by the computer. Variables studied individually and in various combinations were latitude, longitude, month and year of infection, bearing of the site to the ridge, exposure, elevation, and the numbers of dead and dying trees.

Site incidence was analyzed in relation to exposure and ridge bearing using the chi-square method on number of sites fitting a given description of these parameters and assuming that the sites would be equally distributed if exposure or ridge bearing were unrelated to incidence. This analysis was done on each of the three blocks individually and on all the data combined as one set.

Infection sites were plotted (latitude vs. longitude) on a digital computer plotter and coded by color and number for the year of discovery. Visual observations for patterns of spread were made on plots for pairs of years. Because Craighead and Nelson (4) reported patterns of OW spread over a 5-yr period, one year's infection sites were plotted with the following year's sites and with the third, fourth, and fifth year's sites, where possible.

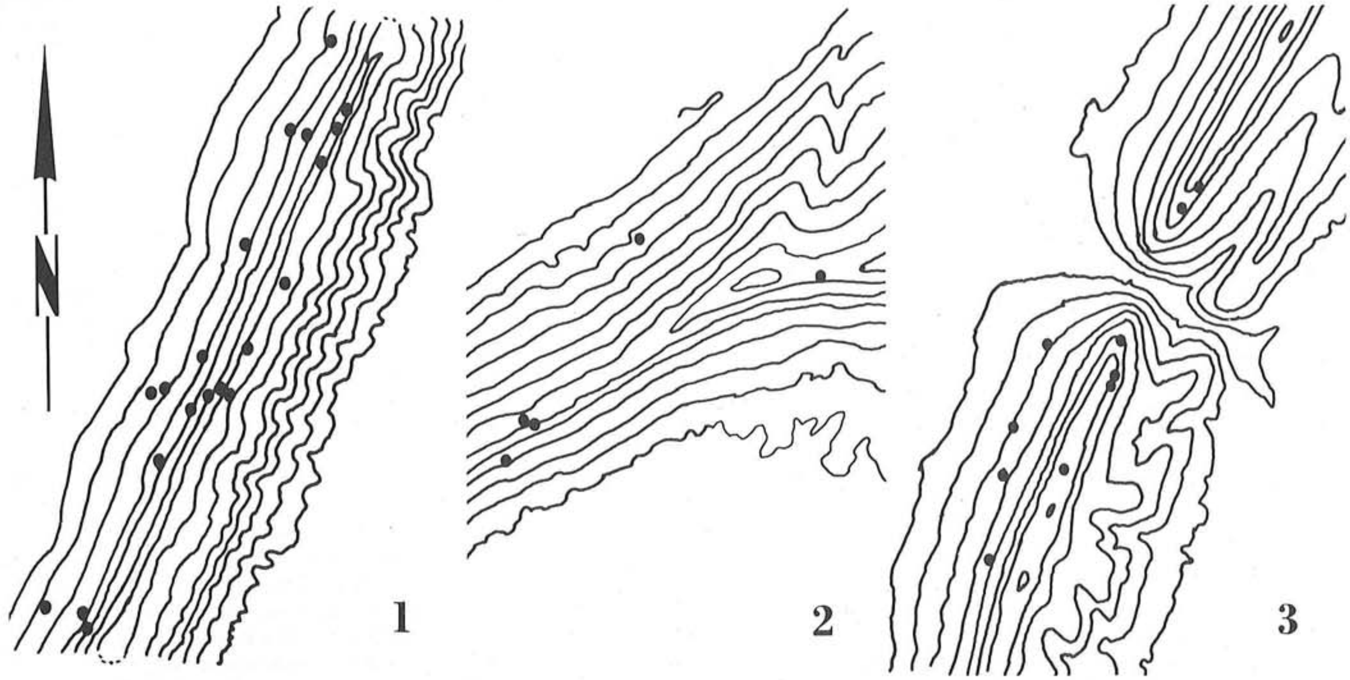
RESULTS AND DISCUSSION

Correlations between disease incidence and latitude, longitude, and month and year of infection yielded low correlation coefficients. In the few cases where the coefficient was more than 0.5, a graph of the sites, by variable involved, was made. These graphs yielded random or indiscernible patterns, indicating no pattern of spread related to latitude and longitude. The same procedure, utilizing bearing of site to the ridge, exposure, elevation, and numbers of dead and dying trees, also yielded low correlation coefficients ($r^2 = 0.037–0.0001$), indicating no pattern of spread relating to these variables.

Locations of OW infection centers in any given year were not related to infection centers in any of the five following years. The pattern of spread from year to year appeared to be random.

Chi-square analysis showed significantly more ($P = 0.1$) infection sites on the western sides of ridges than on other sides. Analysis of sites on the western sides showed significantly more ($P = 0.1$) sites with a northwestern exposure than with a southwestern or western exposure.

Not all ridges within the three study blocks had a northeast bearing. Therefore, a chi-square analysis was made of the number of infection sites on mountains within particular ridge bearing ranges.



Figs. 1–3. Distribution of oak wilt infection centers in Pennsylvania from 1964 to 1970. **(1)** A portion of Blacklog Mountain, Huntingdon County. Lowest plotted contour is 275 m; highest ridge point is 494 m; ridge bearing is 020°. All infection centers were on the west side or top of the ridge. **(2)** A portion of Harbor Mountain, Bedford County. Lowest plotted contour is 366 m; highest ridge point is 652 m; ridge bearing is 066°. Infection centers occurred on the ridgetop or with equal frequency on the northwest or southeast sides of the ridge. **(3)** A portion of Buffalo Mountain, in the vicinity of Buffalo Mills Gap, Bedford County. Lowest plotted contour is 427 m; highest ridge point is 640 m; ridge bearing south of the gap is 025°. Infection centers were on the west side or on top of the ridge except where eddy currents apparently impacted vectors onto the easterly sides near the ends of the ridges.

Significantly more ($P = 0.001$) OW sites occurred on ridges bearing less than 045°, and most infection centers were on the west sides of these ridges.

The prevailing winds in Pennsylvania are from the west-southwest. On major ridges with bearings less than 045°, nearly all infection centers occurred on the upper west-facing slopes and ridgetops (Fig. 1). However, as the ridge bearing changed to greater than 045°, more or less parallel to the prevailing wind, this relationship was lost; there was no significant difference between disease incidence on the northwest and southeast aspects (Fig. 2). On lower ridges and knobs within the broader valleys, disease incidence was also unrelated to aspect.

An interesting phenomenon occurred in some narrow wind and water gaps in north-bearing ridges. Eddy currents apparently carried vectors around the ends of the ridges to impact on the easterly slopes (Fig. 3).

These findings explain the data of Popp (12) and Cones (3). Popp did his study on ridges with bearings between 045° and 055°, ie, on ridges more or less parallel to the prevailing winds. Therefore he found no relationship between disease incidence and aspect. Topography in West Virginia consists of many knobs, hills, and ridges lacking the general north-south orientation characteristic of mountains in south central Pennsylvania. In such terrain, local eddy currents probably impact the vectors onto all aspects, as, indeed, Cones reported.

Our findings and all of those previously cited (2,3,5,6,12,14,17) are consistent with the behavior of a windborne vector (8). Insect vectors emerging from dead trees fly up above the canopy. When they escape their flight boundary layer, ie, when wind speed exceeds their flight speed (15) although they are still actively flying, they have little control over the direction and distance of flight (8). They are disseminated downwind until impaction, perhaps many miles from their point of origin, particularly in mountainous terrain.

Thus, OW infection centers resulting from long-range dissemination appear to occur at random and to be unrelated to previous infection centers. In mountainous terrain the insects impact onto the upper slopes and ridgetops, primarily on the western slopes of ridges more or less perpendicular to prevailing winds, and wih equal frequency on both sides of ridges more or less parallel to prevailing winds. In more broken terrain, eddy currents carry the insects onto upper slopes and tops of hills with no relationship to aspect. In flatter terrain, the insects tend to impact on trees on the edges of stands or on dominant trees protruding above the canopy within stands.

Since these studies were completed, Mason and McManus (9) have developed a model of insect dispersal in a ridge and valley system. They modified an advecting Gaussian puff model by assuming an elliptic rotor of wind in a valley when wind flow was perpendicular to the ridges. The model predicts that most insects lofting off one ridge will fall out on the opposite ridge just below the ridgetop. Thus, the pattern predicted by the mathematical model coincides with the observed distribution of OW infection centers in south central Pennsylvania.

ACKNOWLEDGMENTS

We thank L. V. Madden and S. P. Pennypacker for advice and assistance in conducting the computer analysis.

LITERATURE CITED

1. Bowen, K. L., and Merrill, W. 1980. Oak wilt foci related to ridge bearing and aspect in Pennsylvania. (Abstr.) Phytopathology 70:459.
2. Brinkman, K. A. 1952. Oak wilt mortality in an unmanaged Iowa forest. J. For. 50:941-942.
3. Cones, W. L. 1968. The relationship of site productivity and some site factors to the incidence of oak wilt resulting from long distance spread in West Virginia. Ph.D. thesis. West Virginia University, Morgantown. 123 pp.
4. Craighead, F. C., and Nelson, J. C. 1960. Oak wilt in Pennsylvania. J. For. 38:872-881.
5. Henry, B. W., Moses, C. S., Richards, C. A., and Riker, A. J. 1944. Oak wilt: Its significance, symptoms and cause. Phytopathology 34:636-647.
6. Hutnik, R. J., Wood, F. A., and Grand, L. F. 1963. Oak wilt-site relationships. Pages 15-16 in: Proc. Coop. Oak Wilt Control-Res. Conf. Morgantown, WV, November 1962.
7. Jeffrey, A. R. 1968. The 1968 oak wilt program in Pennsylvania. Pa. Dep. Agric. Bur. Plant Ind. Harrisburg. 25 pp.
8. Kennedy, J. S. 1975. Insect dispersal. Pages 103-119 in: D. Pimentel, ed. Insects, Science, and Society. Academic Press, New York.
9. Mason, C. J., and McManus, M. L. 1982. Larval dispersal of the gypsy moth. In: The gypsy moth: Research towards pest management. U.S. Dep. Agric. Tech. Bull. 1584 (In press).
10. Merrill, W. 1967. The oak wilt epidemics in Pennsylvania and West Virginia: An analysis. Phytopathology 57:1206-1210.

11. Merrill, W. 1968. A summary of recent oak wilt research at The Pennsylvania State University. Pages 1-7 in: The 1968 Oak Wilt Program in Pennsylvania. Pa. Dep. Agric. Bur. Plant Ind. Harrisburg. 25 pp.
12. Popp, R. M. 1968. Relationship of oak wilt incidence to vegetation in south-central Pennsylvania. M.S. thesis. Pennsylvania State University, University Park. 108 pp.
13. Rexrode, C. O., and Jones, T. W. 1970. Oak bark beetles—important vectors of oak wilt. J. For. 68:294-297.
14. Riker, A. J. 1951. The spread of oak wilt in local areas. Phytopathology 41:30.
15. Taylor, L. R. 1974. Insect migration, flight periodicity and the boundary layer. J. Anim. Ecol. 43:225-238.
16. True, R. P., Barnett, H. L., Dorsey, C. K., and Leach, J. G. 1960. Oak wilt in West Virginia. W. Va. Agric. Exp. Stn. Bull. 448T. 119 pp.
17. Wilhour, R. G. 1968. Relationship of environmental factors to the distribution of oak wilt in Pennsylvania. M.S. thesis. Pennsylvania State University, University Park. 65 pp.