

DESCRIBING THE SPREAD OF OAK WILT USING A GEOGRAPHIC INFORMATION SYSTEM

by Dixon Shelstad¹, Lloyd Queen², David French³ and Daniel Fitzpatrick⁴

Abstract. The oak wilt fungus (*C. fagacearum*) spreads both through root grafts to adjacent trees and via insects over longer distances. Effective control of the disease requires a better understanding of the spatial and temporal components of both types of spread. Towards that end, color infrared aerial photography covering a ten-year interval of time was interpreted and then analyzed using a GIS for purposes of describing spread rates and areas affected. The GIS allowed rapid and thorough assessment of both overland and local disease spread. New infection centers were found to occur at greater distances than previously reported, and these new centers accounted in total for the most significant component of increase in total area affected by the fungus.

Oak wilt is believed to be native to the eastern United States and has not been identified anywhere else. It is found from Minnesota to Texas and east to Pennsylvania and South Carolina. The causal pathogen was first correctly identified in Wisconsin (13). Evidence suggests that infections existed before this date but were attributed to other causes (9). The apparent rate of spread of oak wilt in the forties and early fifties is now often described as the rate of spread of the recognition of oak wilt; and since 1951, the range of oak wilt has changed little (11).

While several members of the Fagaceae family have demonstrated potential susceptibility to oak wilt (7), the disease is only known to infect members of the *Quercus* (oak) genus. Infection by the oak wilt fungus, *Ceratocystis fagacearum*, stimulates the formation of tyloses and gummy materials in the water-conducting tissues of the host. This plugging of vessels in the tree results in wilted foliage, the primary macroscopic symptom of the disease. Members of the red oak subgenus *erythrobalanus*, usually die within weeks of symptom expression. Death may take up to several years for members of the white oak subgenus, *lepidobalanus*.

Spread of the disease occurs when *C. fagacearum* spores produced on oaks recently

killed by oak wilt are transferred to the vascular system of a healthy oak. The disease is most commonly spread from diseased trees to adjoining healthy oaks of the same species through grafted root systems and most individual oaks contract oak wilt in this manner. Many forest stands with a high percentage of oak have consistent short-distance or local oak wilt spread rates. Pure northern pin oak (*Quercus ellipsoidalis*) stands for example, have a very high incidence of root graft connections and most trees are interconnected through a common root system (15). Characteristic infection centers, which expand in size at their perimeters each year, result from this rather predictable local spread.

The other method of spread of oak wilt is by insect transmission of spores. Conidia and ascospores are produced on mycelial mats which form under the bark of recently wilted red oaks. Pressure pads formed at the center of these mats rupture the bark, and inoculum then becomes available for dispersal by outside agents (primarily insects). In Minnesota, sap-feeding beetles in the Nitidulidae family have been found to be responsible for long-distance or overland transmission of oak wilt (11).

Several specific conditions must coexist in a well-timed series of events before insect transmission can occur however. First, inoculum on fresh mycelial mats (less than a few days old) must be present. Secondly, nitidulid beetles must be present in the stand, and their activities must lend themselves to dispersal at some distance. Thirdly, the host tree must contain a xylem-exposing wound. This wound must be fresh when nitidulid beetles transfer the fungus from mycelial mats. Fourthly, the above conditions must coincide when the host tree is susceptible to infection during the spring. Finally, biological deterrents to the

1. Vermillion Community College, Ely, Minnesota 55731

2. Remote Sensing Laboratory, University of Minnesota, St. Paul, Minnesota 55108

3. Department of Plant Pathology, University of Minnesota

4. Department of Forest Resources, University of Minnesota

infection process, such as competing fungi, must not prevent infection. When all requirements are met, a successful overland transmission and infection can occur. That all conditions must be satisfied within a restricted time frame accounts for the observation that new overland infections are rare. Nevertheless, all infection centers, no matter how large, were originally started when at least one tree was infected by insect-borne spores.

Overland spread of oak wilt via insect vectors is less effective, less frequent and less predictable than the local root-graft spread of the disease. Insect transmissions have been found to cover distances of up to 0.40 km (12). Since overland spread often involves trees at some distance from existing infection centers, a new overland infection represents the initiation of a new infection center. If the newly infected tree is surrounded by oaks of the same species, the new infection center will continue to expand and infect local trees via root graft transmission. Thus, the general pattern of oak wilt spread is a function of both local and overland transmission of the fungus. A series of patch-like, rather uniformly expanding infection centers dominate the pattern, with new infection centers originating on a somewhat random basis, in a manner that is difficult to predict.

Statement of Problem

Since the fungus causing oak wilt was first described and named, several studies and reports have addressed the subject of spread rate. Many of these publications have dealt with either root graft or insect transmission spread rates. Some have pursued the combined effects of both types of spread, and still others have attempted predictions based on records of past spread rates (2, 19). Recently, Menges (16, 17, 18) has derived predictive equations for both root graft and insect transmission. His study of spread dynamics has resulted in an empirically derived model that predicts tree-by-tree mortality due to oak wilt.

Our approach to the study of oak wilt involved the description of both overland and localized spread rates over a ten-year period of time. We did not extend the research into a predictive mode. Rather, the focus of this paper is a discussion of the relative significance of both types of

spread; and their cumulative impact on the total area affected by the disease. Basic descriptive statistics were determined using automated map analysis routines, which are described later in the paper.

These descriptive data provide baseline information on disease dynamics, and suggest several new insights into the two modes of spread. As described above, oak wilt spread patterns are confined to occasional scattered infection centers, which slowly expand in areal extent from their perimeters. Thus, it was thought that such a pattern of disease spread would lend itself to analysis using remote sensing and Geographic Information System (GIS) techniques. The project reported in this paper developed an approach similar to that described by Appel *et al* (5), which was used to monitor the spread of oak wilt in live oak (*Quercus fusiformis*). The current implementation is also entirely pc-based.

Specifically, color infra-red aerial photography and an automated GIS were used to perform three separate analysis tasks: 1) evaluation of linear spread rates in sample plots due to root graft transmissions, 2) an assessment of overland transmission characteristics of the disease, and 3) measurement of the total increase in oak wilt extent over a given period of time.

Experimental Procedure

Three separate test sites, located individually in Sherburne, Anoka and Chisago counties in central Minnesota, were selected for investigation. Situated within the Anoka glacial outwash plain, this area is dominated by sandy soils. Oak-type forest predominates, with *Quercus ellipsoidalis* (northern pin oak), *Q. rubrus* (northern red oak), *Q. alba* (white oak) and *Q. macrocarpa* (bur oak) commonly present. Associates include cherry, elm, basswood and aspen.

Baseline data for the project came from two sets of color-infrared (CIR) aerial photography, covering the same geographic area but separated in time by ten years. Positive CIR transparencies from both 1977 and 1987 were manually photo-interpreted onto mylar overlays at a scale of 1:9600. Interpretation delineated the extent of oak forest, the extent of oak wilt in 1977, the extent of oak wilt in 1987 as well as key cultural and

natural features. All interpretations also were field verified.

Boundaries used to tile the study area into three discrete, thematically similar strata were also entered into the GIS. Tiles were defined as homogeneous groups or micro-regions of oak forest. Selection of these three sites was based on the availability of high-quality photography that provided complete coverage of the strata, a relatively high percentage of oak type forest, presence of oak wilt in 1977, prior field experience in the study area that expedited ground truthing efforts, and geographical dispersal of study areas over the flight lines covering this area. The strata selected represent oak type forest over a relatively broad area, with sites separated by as much as 58 kilometers. Summaries of total area and area of oak forest for the three test strata are listed in Table 1 (the numbers used to name each of the study areas corresponds to the photo identification number on the original 1977 CIR photography). The results reported in the next section were gathered from a GIS-based analysis of these three strata.

The final GIS database contained seven thematic datasets. These consisted of 1977 wilted areas and extent of oak forest, 1987 wilted areas and extent of oak forest, cultural features (e.g., roads), water bodies, and study area tile boundaries. Each theme was digitized from the mylar overlays and stored in a grid-cell based GIS (Environmental Planning and Programming Language; Version 7 (EPPL7)) at a cell size of two meters by two meters.

Results and Discussion

Evaluation of root graft spread rates. An overlay of the digitized maps for both years (1977 and 1987) resulted in a single new map that showed concentric disease zones around each original infection center. Starting in the center and moving outward, these zones correspond to the area affected by wilt in 1977, then the area impacted by wilt between 1977 and 1987, and finally the unaffected area of healthy oak. This sequence of spread is shown for Area 62 in Figure 1.

Within Area 62 the patch dynamics and increase in area affected from 1977 to 1987

around the perimeter of existing pockets of infection is obvious. Clearly this localized spread is not isotropic, yet expansion of the area affected occurs along virtually the entire oak perimeter of the patches. The 1987 coverages also show the location of new infection centers, some at quite a distance from existing centers. Notice that new centers tend to be located along road and forest edges, where tree damage was most likely to occur.

Next, an estimate of the average spread rate over the ten year period for a sample of infection centers was generated using a polygon buffer operation. Samples were drawn from all three strata. Specifically, a buffer command was used to build a layer of cells of a specified width onto the entire perimeter of nine sample plots (infection centers) depicted in the 1977 oak wilt area. The width of each buffer was then iteratively increased until the area of "buffered" cells matched the total area of the ten-year spread (as defined by the



Figure 1. Spread of Oak Wilt in Area 62. Green areas depict healthy oak forest. By 1987 the patch dynamics of spread via root graft transmission of the fungus are quite evident, as are several totally new infection centers.

Table 1. Land areas by study area (hectares)

Study area name	Total area	Oak type area	% Area in oak
62	335.7	144.7	43
32	366.1	132.0	36
11	291.1	96.7	33
Total	992.9	373.4	38

1987 extent of the patch) for each sample plot. This process replicates advancement of the disease as though spread were occurring at a constant linear rate along the entire infection center perimeter. Finally, given both the total area affected and the area of each cell, an average linear spread rate was determined for each strata by assessing the radius of each of the nine buffered infection centers.

As shown in Figure 2, the average spread rates varied from 2.42 to 4.43 meters per year, with a nine-plot mean of 3.47 meters per year. A 95% confidence interval on the mean of plot spread rates yields a range of 2.94 to 4.01 meters per year. These figures are comparable to previous estimates of spread in Minnesota; Anderson and Anderson (2, 3) estimated a radial spread rate from 1.35 to 3.46 meters per year, and French and Bergdahl (9) reported a maximum value of 7.65 meters per year.

A second component of the linear spread rate analysis measured the spread rate necessary to achieve the maximum extent of oak wilt in a sample plot in any single (raster) direction over the ten-year period. Again the nine sample plots were analyzed using the GIS by measuring the maximum distance that the disease had spread in any of eight directions within the raster. This octant-

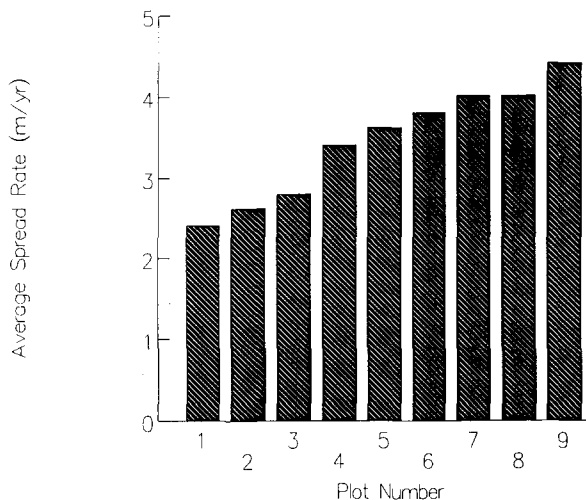


Figure 2. Average Linear Spread Rates via Root Grafts. Results show spread rates for nine random sample plots or patches of oak wilt. The overall sample mean was 3.47 meters per year. Note that plot numbers do not correspond to the sequence or order of sampling.

based direction analysis is also described in Appel *et al* (5). The results of this process, shown in Table 2, show that of the nine plots, number 9 had a maximum spread of 76.8 meters in ten years, an average maximum spread of 7.68 meters per year. The lowest maximum linear spread rate per year was 3.64 meters per year for plot number 1.

Overland spread. Overland, or insect transmission, spread was analyzed in two different ways. First, the rate of new infection center formation was determined on a per year basis. Secondly, spread distances from nearest known sources of inoculum were measured.

New infection center formation rates were determined from each of the three original study areas (Areas 11, 32, and 62). The total number of new centers and the number of new centers per study area per year on a per hectare of oak forest basis are in Table 3.

The total number of new centers for all three study areas was 51, with an overall new center formation rate of 0.015 centers per hectare (of total oak forest area) per year. This figure compares to reported rates of 0.026-0.063 (14), 0.042 (3) and 0.002-0.006 (16). The rates in Table 3 do show variability, ranging from 0.011 to 0.025 new centers per hectare per year.

The second phase of overland spread analysis determined the distances of new infection centers from known centers of inoculum. Only existing, active infection centers (those having increased in affected area over time) were used as sources of inoculum in this study. Other possible sources of inoculum (including those at greater distances), such as piled firewood, were not considered. Distances from the new centers to the perimeter

Table 2. Maximum linear root graft spread for sample plots

Sample number	Maximum spread (meters/year)
1	3.64
2	4.43
3	4.83
4	5.05
5	5.26
6	5.66
7	5.84
8	6.45
9	7.68

of the nearest existing center for each of the three strata are listed in Table 4.

In terms of site-specific results, recall that areas 62 and 32 were both infected by oak wilt to a considerable degree prior to 1977, with 12% and 20% of the total oak areas affected, respectively. In contrast, less than 0.5% of Area 11 was affected prior to 1977. Yet, as Tables 3 and 4 show, Area 11 had many more new centers develop. These new infection centers also developed in the longer distance categories (300 to 600 meters) compared to Areas 62 and 32, which combined had only one new center develop in that distance category. Insect transmissions were found to cover distances of 300 to 600 meters in 12 of the 51 cases. One new center occurred in Area 11 at 580 meters from the nearest source of inoculum. This compares with the 400 meter distance reported by Guyton (12).

The sum total of all new centers established in all three areas is shown in Figure 3. This graphic classifies new center formation according to 150 meter intervals from existing centers. The pooled results clearly show that most new center formation occurred within 300 meters of an existing center. Yet the most startling result shows that, even though the number of new centers at long distances is small, they have a significant impact on total area affected over time (see Figure 4). This is dramatically true for Area 11.

Evaluation of combined past spread. The final descriptive data presented consist of original area occupied by oak prior to the introduction of the disease, area affected by oak wilt as of 1977,

Table 3. New infection center formation rates

Study area	No. of new centers 1977-1987	Rate (centers/yr/ha)
Area 62	15	0.012
Area 32	12	0.011
Area 11	24	0.025
Total	51	0.015

Table 4. Number of new centers by distance category

Study area	0-150m	150-300m	300-450m	450-600m	Total
Area 62	7	7	1	0	15
Area 32	8	4	0	0	12
Area 11	7	6	8	3	24
Total	22	17	9	3	51

area affected by oak wilt as of 1987 and difference in affected area over the ten-year period of study.

Areal measurements were easily obtained from the GIS base files, and a simple map differencing procedure was used to detect the total increase in area affected by oak wilt from 1977 to 1987. These results are illustrated for each of the three strata in Figure 4. Area 11, which had the largest percentage of oak forest that remained healthy in 1987 (89% versus 70% and 62% healthy for areas 62 and 32, respectively), was the most dynamic. In fact, Area 11 experienced a 1000 percent increase in area affected by oak wilt between 1977 and 1987, while Area 62 experienced a 250 percent increase and Area 32 showed only a 200 percent increase in total area affected by the disease. Again the dramatic impact of new centers and of centers a relatively long distance from existing centers is clearly evidenced for Area 11. Area 11 was relatively unaffected by oak wilt in 1977, but had nearly double the number of new centers of Areas 32 and 62 (Table 3).

Summary

Sample forest plots and demonstration areas were utilized in this study for testing and

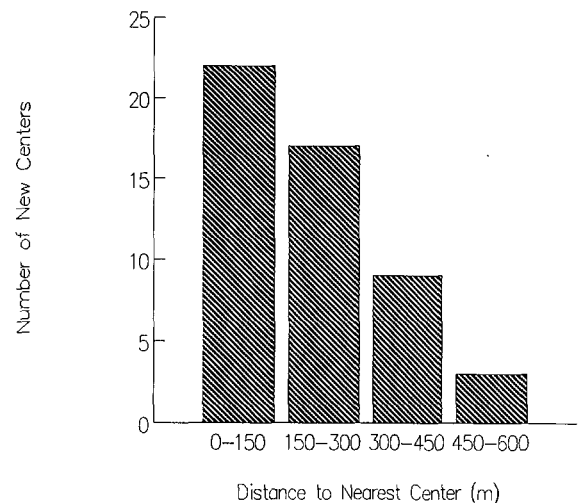


Figure 3. Distances of Overland Spread of Oak Wilt. Bars show the number of new infection centers formed within 150 meter intervals from an existing infection center. Results are from all three strata and show the common development of new centers close to existing pockets of oak wilt.

demonstrating the utility of GIS-based processing for oak wilt assessment. The information derived from our project is representative of the oak wilt situation in central Minnesota, but selection of test plots was not randomized; thus extrapolation or prediction efforts were not included in this paper.

Prediction of the area affected by root graft spread at the perimeter of existing centers could be accomplished by extending the calculated average buffer zones through a given interval. However, the major obstacle to effective spread prediction involves overland (insect-based) spread. Without more detailed temporal analysis, characterization of the potential for overland spread is simply not feasible.

Although initially conceived and developed for oak wilt, the approaches presented here can be applied to any patch-like insect or disease problem where spread from infection centers occurs in comparable patterns. For example, dwarf mistletoe and *Ceratocystis wagneri* (8) may be other candidates for evaluation using these GIS approaches.

The use of linear models in this study follows the work of others (5, 16), yet in the case of local spread the degree of progression of the disease in terms of area is not a linear nor simple isotropically exponential case. It is also likely that the more established oak wilt becomes in an area, the more probable both types of spread become.

A major problem associated with securing meaningful information on overland spread in particular relates to the biology of the insect transmission process. Of the several factors necessary to bring about a successful transmission, perhaps the most critical as to whether an infection will occur is the wounding of a healthy oak. This action is in fact independent of the degree of oak wilt activity in the area, as was the case in Area 11. Thus, the consideration of external wounding sources, such as human activity, should be factored into any predictive effort; perhaps in the form of spatial stratification of spread rates by probability of wounding (i.e., proximity to human activity such as road construction).

Another condition important to the assessment of oak wilt is the prevailing oak coartype. As stated in the introduction, "pure" red oak stands on sandy soils have a high incidence of root-

grafting and are therefore highly susceptible to the root-graft spread of oak wilt. Oak stands on heavier soils are typically less pure and appear to have less incidence of root-grafting between oaks. The spread of oak wilt within these types of oak stands is both less rapid and less predictable.

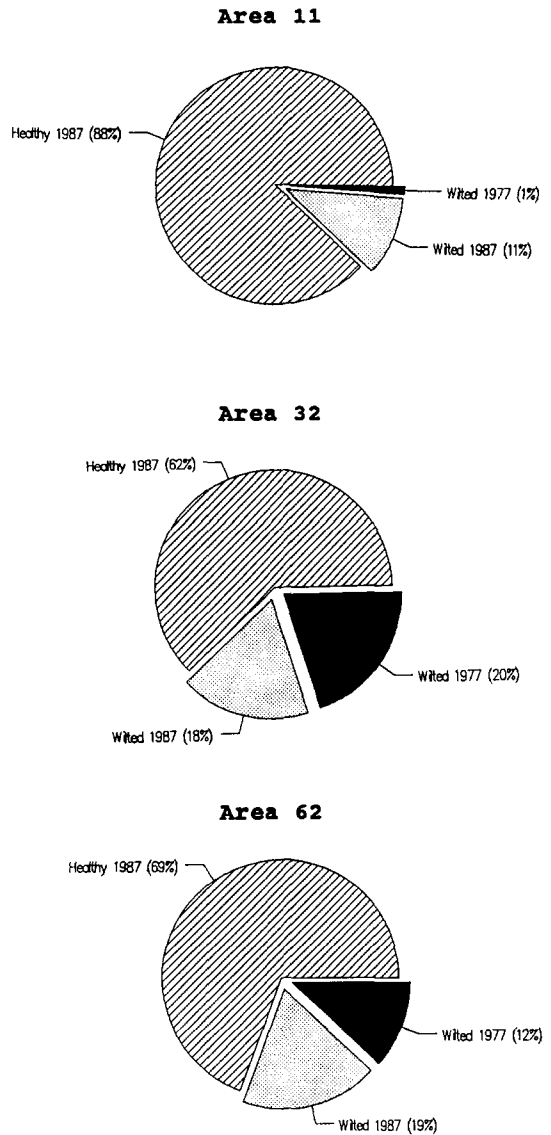


Figure 4. Percent of Oak Area Affected by Oak Wilt. Differences in initial conditions and the total area affected by oak wilt are evident between the three strata. Area 11 experienced a dramatic increase in area affected, yet shows an overall level of impact well below that of the other two strata.

Given that soil mapping units are highly correlated to the root-grafting nature of oak stands and given that those data layers are currently resident in a GIS, extension of the research to include soils can and will be readily accomplished. Stratification of descriptive and predictive efforts correlated to soil characteristics is important to furthering our understanding of this disease.

In conclusion, CIR photography at a scale of 1:9600 or larger is extremely useful for oak wilt interpretation. Although the pc-based GIS provided the power and flexibility necessary for measuring and assessing root graft spread, insufficient data on overland spread precluded future overland spread projections. An assessment of the patterns of new center formation and a better temporal sequence of rate of formation is clearly needed. Breaks in oak stand continuity and adjacency are not concentric or linear, thus further work on irregularly shaped and non-linearly iterated buffers is need. This in particular could build and expand on an octant-based effort, such as that applied by Appel *et al* (5)

Both the GIS and the remote sensing techniques helped provide a simple, yet reasonably complete assessment of disease progression. These procedures permitted both the analysis of root graft spread and the characterization of overland transmission. The pc-based GIS provided for efficient data storage and for quick access and analysis. The major limitation of a GIS approach is the cost of digital database development and the need to convert interpretations into digital coverages. Once established, however, the database itself is an invaluable resource for current and historical data.

Furthermore, in addition to providing quantitative data concerning disease spread, the GIS supported the added capability of presenting visual displays of the data. These visual displays enhanced the clarity, comprehension, and impact of data presentations. Coupled with future refinements to the basic methods outlined in this paper, a GIS approach truly supports powerful capabilities for interpreting the spatial and temporal qualities of the dynamics of oak wilt spread.

Acknowledgments. This work was supported by the Col-

lege of Natural Resources and the University of Minnesota Agricultural Experiment Station under Project MN 40-16. Contributed as Paper No. 18586 of the Minnesota Agricultural Experiment Station.

Literature Cited

1. Anderson, G.W. and R.L. Anderson. 1957. The local spread of oak wilt, 1955 to 1956. Lake States Forest Experiment Station, U.S. Dept. of Agric. Technical Note No. 480.
2. Anderson, G.W. and R.L. Anderson. 1958. The local spread of oak wilt, 1955 to 1957. Lake States Forest Experiment Station, U.S. Dept. of Agric. Technical Note No. 521.
3. Anderson, G.W. and R.W. Anderson. 1963. *The rate of spread of oak wilt in the Lake States*. Forestry 61:823-825.
4. Anderson, R.L. 1958. Survey results in Wisconsin and Minnesota. Proc. Oak Wilt Conference, Morgantown, West Virginia. Compiled by the Central States Forest Experiment Station. Nov.
5. Appel, D.N., R.C. Maggio, E.L. Nelson, and M.J. Jeger. 1989. *Measurement of expanding oak wilt centers in live oak*. Ecology and Edipemiology 79(11):1318-1322.
6. Boyce, J.S. 1959. *Oak wilt spread in control-treated and untreated counties in the southern Appalachians*. J. Forestry 57:660-661.
7. Bretz, T.W. 1955. *Some additional native and exotic species of Fagaceae susceptible to oak wilt*. Plant Disease Reporter 39:495-497.
8. Cobb, F.W., Jr., G.W. Slaughter, D.L. Rowney, and C.J. DeMars. 1982. *Rate of spread of Ceratocystis wageneri in ponderosa pine stands in the central Sierra Nevada*. Ecology and Epidemiology 72(10):1359-1362.
9. French, D.W. and D. Bergdahl. 1973. Oak wilt in Minnesota 1972. University of Minnesota. Agricultural Experiment Station Miscellaneous Report 118, 8 p. St. Paul, Minnesota.
10. French, D.W. 1988. Forest and shade Tree Pathology. University of Minnesota, Department of Pathology, St. Paul, Minnesota. 271 pp.
11. Gibbs, J.N. and D.W. French. 1980. The transmission of oak wilt. USDA Forest Service Research Paper NC-185, 17 p.
12. Guyton, T.L. 1952. *An unusual occurrence of oak wilt in Pennsylvania*. Plant Disease Reporter 36:386.
13. Henry, B.W., C.S. Moses, C.A. Richards and A.J. Riker. 1944. *Oak wilt: It's significance, symptoms and cause*. Phytopathology 34:636-647.
14. Jones, T.W. and T.W. Bretz. 1958. Experimental oak wilt control in Missouri. United States Forest Service Missouri Agricultural Experiment Station Research Bulletin 657.
15. Kuntz, J.E. and A.J. Riker. 1955. *The use of radioactive isotopes to ascertain the role of root grafting in the translocation of water, nutrients, and disease-inducing organisms among forest trees*. International Conf. Peaceful Uses of Atomic Energy Proceedings 12:144-148.
16. Menges, E.S. 1978. Patterns of oak wilt mortality in midwestern oak forests. Proceedings of the Central Hardwood Forest Conference, 508-528.
17. Menges, E.S. and O.L. Loucks. 1984. *Modeling a disease-caused patch disturbance: oak wilt in the*

- midwestern United States. Ecology* 65(2):487-498.
18. Menges, E.S. and J.E. Kuntz. 1985. *Predictive equations for local spread of oak wilt in Southern Wisconsin. Forest Science* 31(1):43-51.
19. Merrill, W. 1967. *The oak wilt epidemics in Pennsylvania and West Virginia: an analysis. Phytopathology* 57:1206-1210.

University of Minnesota
St. Paul, Minnesota 55108

Résumé. Le champignon pathogène de la flétrissure du chêne (*C. fagacearum*) se dissémine, et par l'entremise de greffes racinaires aux arbres adjacents, et via des insectes sur de longues distances. Un contrôle efficace de la maladie exige une meilleure compréhension des composantes spatiales et temporelles de l'un et l'autre des types de dissémination. En vue de cette fin, une photographie aérienne infrarouge couvrant une période de dix ans était interprétée et ensuite analysée en utilisant un SIG (système d'information géographique — GIS) dans le but de décrire des taux de dissémination et les aires affectées. Le SIG permet une évaluation rapide et complète de

l'une et l'autre des propagations sur de grandes surfaces et locales. De nouveaux foyers de contamination étaient découverts qui se rencontraient à des distances supérieures à celles imaginées précédemment; ces nouveaux foyers comptaient en totalité comme la composante la plus significative d'accroissement du total d'aire affecté par le champignon pathogène.

Zusammenfassung: *C. fagacearum* (oak wilt fungus) dehnt sich aus durch Verwurzelung mit benachbarten Bäumen und über weitere Distanzen durch Insekten. Wirksame Bekämpfungen von den Krankheiten fordert ein besseres Verständnis von den räumlichen und zeitlichen Komponenten von beiden Ausdehnungsarten. Um diesen Zweck zu erreichen wurde farbe-infrarot Luftaufnahmen über eine zehn-jährige Zwischenzeit interpretiert und dann mit einem GIS analysiert um Ausdehnungsraten und infizierten Gegenden zu beschreiben. Das GIS erlaubte eine schnelle und umfangreiche Analyse von der lokalen sowohl auch weiteren Krankheitsausdehnung. Neue Infizierungsknoten wurden festgestellt, aber weiter getrennt als früher geglaubt; diese neuen Knoten sind insgesamt die wichtigsten Komponenten der Zunahme von der Gesamtfläche, die mit *C. fagacearum* infiziert ist.

ABSTRACT

PEARSON, S. 1990. **Don't get my drift.** *Grounds Maintenance* 25(1): 32,36,38.

Pesticide drift can be a major environmental problem. It also wastes chemicals and contributes to spotty pest control. You can control pesticide drift if you and your crews make it a point to read pesticide labels, check weather conditions and follow proper application instructions. Two types of drift cause chemicals to go off-target — particle drift and vapor drift. Particle drift occurs when the wind or careless application procedures scatters spray droplets off the application site to neighboring shrubs, flowers or even adjacent yards. Vapor drift occurs when chemicals evaporate into the air then the vapor moves wherever the air drifts. Applicators can control particle drift by following these basic principles during application: read the label, check weather conditions, minimize fine droplets, and select the right nozzle.