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PATTERNS OF OAK WILT MORTALITY IN MIDWESTERN OAK FORESTS: STATISTICAL ANALYSES AND MODEL SIMULATIONS

BY

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ABSTRACT

Plant communities are shaped by disturbance and succession. The heterogeneities of both these phenomena are now thought to be important in community dynamics. Disease-caused disturbances have received little study as ecological entities. Spatial and temporal patterns of mortality caused by the oak wilt fungus (Ceratocystis fagacearum), an important cause of oak mortality in the Midwest and East, were examined to answer questions of an ecological nature.

Data previously collected from Sinnissippi Forest in northern Illinois and from four sites in southern Wisconsin were analyzed. The former data set summarizes 17 years of monitoring and oak wilt control in 1600 acres of oak forest. The latter data was collected over a shorter interval and included complete mappings of the sites.

Descriptive Sinnissippi data on total mortality and pocket dynamics were analyzed after considering the effects of selective cutting and oak wilt control. Mortality was highest in trees of the red oak group, in stands dominated by the red oak group, in young stands, and in sandier areas. Pocket birth rates were fairly constant in Sinnissippi as a whole, but showed a higher variation and distinct tempo-

ral patterns in smaller blocks. Expansion of oak wilt pockets approached a constant rate after several years.

Pocket expansion is accomplished mainly by root graft transmission of fungal spores between adjacent trees of the red oak group. This local spread was quantified by modeling the probability of transmission as a simple combination of the combined diameter of the two trees (DBH) and the inter-tree distance (D). Of eight models attempted, the best overall predictor of transmission probability for both data sets was DBH/D.

In southern Wisconsin, the regression parameter estimates for prediction of root graft transmission probability showed wide site to site variability, with the driest site having the highest slope. Comparison of predictions of spread taking different length intervals at the same site were more similar to each other. Analogous results were obtained from Sinnissippi data. The time for spread between two trees was independent of tree size and inter-tree distance and was fitted to a Poisson distribution.

Longer distance spread of the fungus occurs by insect vector. High levels of vector-mediated mortality showed a seven year return time for subsets of Sinnissippi. In any area of the forest, the vector mortality in a given year could be predicted from the previous

year's mortality and a weighting for forest-wide favorableness for vector transmission. The weighting was not highly correlated to available weather information.

Vector transmission was shown to occur preferentially over short distances, and an empirical distribution of nearest neighbors for this spread was derived. The evidence suggests that vector transmission is an important component of local oak wilt spread.

The quantitative relationships found for root graft transmission and vector spread of oak wilt were used to formulate a stochastic, empirically-based simulation model that predicts patterns of oak wilt mortality. Fourteen parameters were used to specify run conditions. When parameters representative of Sinnissippi Forest were used, model simulations were in close agreement to mortality data when outside introductions to a 10 acre plot occurred at an average rate of six in 20 years.

The stochastic nature of the simulation model produced wide variation in results even in multiple runs with identical parameters. The greatest variation was encountered when disease damage was low. Graphical arguments were made that suggested the actual rate and timing of background infection (from outside the plot) were critical in specifying resultant mortality. Mortality results from many runs with the same parameters tended to be clumped

in groups (i.e. discontinuous in output space) that were most distinct when outside introductions were most limiting.

Three sensitivity analyses probed the differences in 12 types of output resulting from changes of one model parameter at a time. Overall, the most important parameter was the average efficacy of vector spread, which directly influences both local and background infection. Other parameters that affect vector spread from outside the plot were also quite important. Factors influencing root graft transmission were most significant when the relative amount of red oak group trees was high. A detailed analysis of model output sensitivity to changes in the relative amount of red oak group trees is presented.

The simulation results lead to a characterization of oak wilt as a frequency-controlled system. The low rate of vector-mediated introduction from outside the woodlot constrains further development by the higher-frequency local overland spread and root graft transmission. The latter two types of spread differ less markedly in frequency. This produces the typically complex and unpredictable behavior of disease progress.

Combining the oak wilt mortality information with the knowledge of the recovery of oak forests from the disturbance is difficult for several reasons. Among

these are the complexity of post-settlement oak forests, the unpredictability of species replacement patterns, and the lack of information on the importance of different size gaps for regeneration of particular species. Nevertheless, simulation model results were combined with simple assumptions concerning ingrowth to predict the effect of oak wilt on specific stands in southern Wisconsin. In xeric stands, there should be fairly moderate damage, but only a slight shift toward members of the white oak group. In dry-mesic forests, damage should vary. When oak wilt mortality is serious, black cherry will probably be the major gap phase species benefiting. In mesic stands, the model predicts slight damage and little effect of oak wilt on species replacement. Overall, although oak wilt adds to the problems of fire-adapted oaks in a system without fire, oak forests should equilibrate at a lower, but still substantial, density of red oak group trees.

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I. OBJECTIVES

This study seeks to analyze oak wilt as an ecological disturbance. In particular, the patterns of oak wilt mortality over space and time, as well as rates and characteristics of the various types of spread, will be examined. The reactions of species in the oak forest to oak wilt disturbances will be treated in general terms only. There is a certain advantage to this approach, for far more study has been devoted to species replacement in oak forests than to oak wilt dynamics.

The objectives of this study are basically fivefold:

1) to describe typical patterns of oak wilt mortality on different sites, in terms of number of dead trees and characteristics of patches of infection.

2) to quantify the spatial and temporal components of the two types of oak wilt spread, and to show how these rates may vary in different situations.

3) to use the equations referred to in objective 2 to erect a simulation model that will predict patterns of oak wilt mortality using a manageable number of biologically defined input parameters. The variance associated with the simulation will also be of interest. The model predictions will be compared to the results of data analyses on mortality patterns (objective 1).

4) to use the simulations to describe the sensitivity of different types of output, or the system response, to changes in model parameters.

5) to use all of the results described above to attempt answers to questions concerning:
   a) the nature of disease caused disturbances.
   b) the importance of oak wilt in the dynamics of midwestern oak forests.

The outline of this thesis follows these objectives. In order to set the stage, there are three sections of review. Part II places this study in the general context of disturbance and succession, as well as describing some characteristics of disease caused disturbances. Part III contains a fairly detailed description of the oak wilt organism and disease. The fourth section outlines a classification of some types of plant disease models and reviews some notable examples, in an attempt to place this modeling effort in perspective.

Part V is a methods section for the data analyses. These results are presented in section VI, first for patterns of mortality and patch dynamics (objective 1), and secondly for the dynamics of the two types of disease spread (objective 2). This section is followed by the formulation of a simulation model for predicting patterns of oak wilt mortality, including some additional manipulations based on the results of Part VI. Section VIII reports the results of nominal simulations (objective 3) and crude sensitivity analyses (objective 4). The final section discusses the general questions outlined in objective 5.

These biological questions that I am attempting to elucidate by this statistical/modeling route are listed below. They become clearer in the context of subsequent discussions of disturbance (Part II), oak wilt (Part III),

and the nature of the oak forest (Part IX B). Despite all the efforts expended, many of the proposed answers to these questions will remain speculative.

The questions are:

1) What are the typical patterns of oak wilt damage in space and time? Are the phenomena discontinuous? Can the system be described as constrained by events occurring at different frequencies? What are the implications of these patterns to community dynamics in the forest?

2) Is the host-pathogen system described here globally stable; i.e. what are the boundary conditions for persistence of oaks? the fungus?

3) What properties of local stability does the system have? That is, are there typical levels of damage that are insensitive to small changes in parameter values? What causes large changes in mortality? How does this relate to the oak forest?

4) To what extent did vegetation changes in the midwest resulting from fire cessation and clearing of land for farming provide an opportunity for oak wilt to become a serious perturbation?

5) Can differences in damage due to oak wilt in different geographical areas be ascribed merely to different species compositions?

6) Given what is known about species properties in oak forests, how would the predicted patterns of oak wilt damage be expected to influence species replacement patterns?

7) What is the future of midwestern oak forests? What properties of the various oak species would enable persistence? continued domination?

II. ECOLOGICAL DISTURBANCE

A. The Meaning of Disturbance

The term "disturbance" and its companion "perturbation" are commonly used in ecology. Perhaps the most fundamental meaning of disturbance comes from the formal study of systems, where a disturbance represents a movement away from equilibrium conditions. The use of this term has also been common in traditional community ecology. For ecologists who viewed species replacement as an orderly succession proceding to a climatically controlled steady state (e.g. Clements 1936, Braun 1956, Daubenmire 1966, Margalef 1968, Odum 1969), it has been convenient to relegate disturbance to a position outside the normal functioning of the community.

However, disturbance has often been included as an integral part of community dynamics. The removal of the connotation that perturbations are unnatural forcing functions focuses on a more biologically based working definition. I define disturbance (and perturbation), similarly to Grime (1977), as an episodic phenomenon resulting in unusually high mortality to some segment of a plant community.

The explicit treatment of disturbance in more recent ecological literature has led to what could be character-

ized as a general disturbance-succession paradigm (sensu Kuhn 1962). Briefly stated, disturbance will reset the successional clock. The state of the community can be specified by a still deterministic successional sequence operating in the time since the disturbance reinitiated it. This generalization has spawned many theoretical treatments. For example, Levin and Paine (1974, 1975) erected models describing the relation between disturbance and community structure by focusing on the spatial patterning generated by disturbance.

Perhaps the clearest exposition of this paradigm for a specific plant community was given by Loucks (1970) for Wisconsin forests. He argued that the state of these forests (i.e. diversity, composition, structure, productivity) was dependent on the time since the last fire. The frequency of the fire regime was great enough so that the terminal steady state, or climax, predicted by classical succession models was rarely reached.

While the traditional successional models predict well in some situations, it is evident that there are often many possible routes for succession even in the absence of additional disturbance (McIntosh 1957, Monk 1965, Olson 1958). The addition of disturbance as an important factor eliminates the equilibrium implied by classical models (see Caswell 1978). Thus, the conformity of communities to the

predicted structural and functional changes over successional time (Odum 1969) has been challenged by several authors (Drury and Nisbet 1973, Horn 1974, 1976, Egler 1954, Cooper 1926, Connell and Slatyer 1977). Drury and Nisbet (1973) mounted the broadest attack on the generalizations of succession, arguing that explanations for species replacement patterns must be sought in adaptive strategies of populations, especially in relation to stress and dispersal (see also Pickett 1976).

Indeed, many specific communities fit the succession models poorly. These include bogs (Heinselman 1963), shrublands (Niering and Egler 1955, Pound and Egler 1953), chapparel (Parsons 1976, Vogl and Schorr 1972), fouling communities (Sutherland 1974), the rocky intertidal (Dayton 1971, Menge 1976), prairies (Curtis 1959), and coniferous forests (Dix and Swan 1971, Rowe and Scotter 1973).

Just as succession has been recognized by many as heterogenous, so too has disturbance (Grime 1977). Some important perturbations in forests include fire (Maissurow 1941, Olson 1958, Heinselman 1973, Swain 1973, Rowe and Scotter 1973, Cwynar 1977, Lorimer 1977, Oliver and Stephens 1977, Wein and Moore 1977), various types of wind related mortality (Stearns 1949, 1951, Spurr 1956, Westman 1968, Henry and Swan 1974, Sprugel 1976, Lorimer 1977, Oliver and Stephens 1977, Brewer and Merritt 1978, Canham 1978), forest

disease and insect damage (Wood 1953, Keever 1953, Blais 1965, Klinkowski 1970, Auclair 1975, Richardson and Cares 1976), flooding (Hosner 1958, Franz and Bazzaz 1977), landslides (Flaccus 1959), and lightning caused gaps (Brunig 1964). These perturbations often differ markedly in the amounts and patterns of mortality generated and in their effects on subsequent regrowth. Interactions among disturbances of different types and frequencies deserve more study, but some obvious examples include predisposition of windthrow areas to fire, the dependence of many diseases on weakening factors caused by severe weather, insects, or parasites (e.g. Stevens and Hawksworth 1970), and the increased susceptibility of even-aged conifer stands (perhaps dating from fire or windthrow) to spruce budworm damage.

I argue that the effect of disturbance regimes depends not only on the frequency of disturbance, but also on 1) the type, severity, and spatial scale of the disturbance, 2) the state of the community on which a disturbance acts (Kimmerer 1978), and 3) the specific adaptations (e.g. life history) of the populations making up the mosaic upon which a disturbance acts (Platt 1975, Hutchinson 1965, p.61ff, Grime 1977). As a further complication, note that the severity and scale of disturbance may depend in part on the state of the community, which in turn reflects the time since the

last disturbance. A good example is the chapparel-fire system (Parsons 1976).

Disturbance often creates opportunities for species that may be outcompeted in the absence of perturbation. This may allow persistence of fugitive species that are adapted to typical disturbance caused conditions (Hutchinson 1951, Platt and Weis 1977, Grime 1977). The characteristics of the disturbance regime also specify the refuges to which species may retreat, either to avoid disturbance caused mortality, or that resulting from competition (Woodin 1978). In fact, the species complement of a community may depend on the frequency, magnitude, and types of disturbance common over evolutionary time (Denslow 1979, Pickett 1976).

B. Disease Caused Disturbances

If one accepts the hypothesis that the cause of a disturbance is of fundamental importance in specifying community dynamics, it is interesting to consider the amount of ecological interpretation disease and insect caused disturbances have received. Widespread mortality of balsam fir caused by spruce budworm has led to much investigation of fir forest recovery, including discussions of the cyclic nature of the epidemic (Blais 1965, Morris 1963. See section IV F). On the other hand,

chestnut blight, caused by the fungus Endothia parasitica, has all but eliminated one of the eastern deciduous forest codominants, while receiving only descriptive ecological treatments (Woods and Shanks 1949, Keever 1953, Nelson 1955, Diller 1965, Mackey and Sivec 1972).

In fact, most work on forest diseases has emphasized physiological aspects, with the goal of effective control. Forest pathogens do cause more growth impact (mortality plus growth losses) than either fire or insects (USDA 1973). What the emphasis on control may obscure is the complex evolutionary and ecological interactions of host, pathogen, vector, and environment (van der Plank 1963, Hoff and McDonald 1972, Murdoch 1975, Robinson 1976), and the total impact on the forest system. Such considerations provide the context for this study of oak wilt mortality patterns.

Disease caused perturbations have several attributes that make them unique and interesting. They are usually more or less species specific, removing one species selectively, and thus will have contrasting effects on stands with differing compositions. Thus, as disturbances go, they may be unusually efficient in changing the species complement (e.g. chestnut blight). At the same time, diseases will cause less significant changes in soil properties than fire or windthrow.

Finally, many diseases leave unique spatial patterning

in their wake. The importance of the size of light gaps is frequently recognized, even if poorly known (Richards and Williams 1975, Watt 1947, Bray 1956). Current models of patch dynamics (Levin and Paine 1974, 1975, Horn and MacArthur 1972) require descriptions of at least rudimentary properties of patches before being used to quantify the pattern. Once this is accomplished, knowledge of the effects of these unique perturbations can be utilized. Dynamic gaps that are being converted by ingrowth to the background mosaic, while simultaneously expanding at their margins, may have interesting effects on community and population dynamics. It is this explicit view of disease disturbances in terms of patches that shifts the emphasis of this study away from a more static, descriptive account of states of the system to a consideration of the rates that drive its dynamics.

III. OAK WILT - A SHORT DESCRIPTION

The causal organism of oak wilt, Ceratocystis fagacearum (Bretz) Hunt, was first isolated in Wisconsin in the 1940's (Henry 1944, Henry et al. 1944). It is an Ascomycete, a member of the Pyrenomycetes, and is in the same genus as the causal organism of dutch elm disease. Despite the apparent recent origin, characteristic damage was reported as early as the late nineteenth century (Warder 1881, Harrington 1943). After Henry's isolation, Ceratocystis was implicated in widespread death of oaks throughout the midwest and northeast (Craighead and Nelson 1960). It seems probable that the disease had been widespread prior to 1944, rather than spreading rapidly (Fowler 1952, Boyce 1957b). Oak wilt showed some apparent geographical spread up until the mid 1950's, particularly to the south and west (Dietz and Barnett 1946, Fowler 1952). It has not spread to substantial new ranges, for the most part, since then (Verrall et al. 1959, Craighead and Nelson 1960, Astin and Quilline 1965, Bretz and Jones 1958, Peterson et al. 1968, Peacher et al. 1975), although there are some exceptions (Witcher 1969, Grand and Doggett 1973, Landis 1977). While the spread of oak wilt is slow geographically, its efficiency in individual stands and the prevalence of susceptible hosts have led several authors to consider oak wilt to be the greatest potential threat

to hardwood forests in the eastern U.S. (Nair and Kuntz 1975).

Oak wilt is a systemic disease. As with other wilt diseases (Dimond 1970), trees are thought to succumb mainly due to blockage of xylem vessels with tyloses formed by xylem parenchyma (Beckman et al. 1952). Other contributing substances include gels, spores, mycelia, and various polysaccharides (Parmeter et al. 1956, Struckmeyer and Kuntz 1958, Dimond 1970). The physiological mechanism for tylosis is not understood fully, but may involve auxin production (Lacasse 1966). In addition to blockage, toxin production by the fungus is involved in production of some leaf symptoms (White 1955, Dimond 1970).

Conidia (asexual spores) distribute the fungus widely throughout the water conducting system. Mycelia grow in the wood and bark, causing a soft rot (Sachs et al. 1970), but are outcompeted by secondary invaders once a tree dies (Amos and True 1960, Roncadori and True 1960, Tainter and Gubler 1973). While inside the host tree, C. fagacearum produces conidia and ascospores (sexual spores produced in perithecia) on the fruiting fungal mats that are formed between the wood and bark in recently killed oaks (Curl 1955).

Although all Quercus species are susceptible, trees of the white oak group (Leucobalanus) are less severely

infected, commonly isolating the wilt in a single branch and vertical trunk sector. New growth may cover and isolate the fungus (Parmeter et al. 1956, Nair and Kuntz 1962, Worf and Kuntz 1972). Even when they do die, white oaks may not succumb for several years after the initial infection, while trees of the red oak group (Erythrobalanus) typically die within weeks of infection (Jones 1958). Many authors have stated that there are no cases known where trees of the red oak group recovered after showing symptoms. Drake (1956) found no northern pin oaks (Quercus ellipsoidalis) immune to infection by inoculation, although some required a large amount of inoculum.

Oak wilt results in a drop in water flow, typically accompanied by a more negative water potential and tylose formation, about four days before visible symptoms appear (Beckman et al. 1953, Struckmeyer et al. 1958, Pengelly et al. 1977). There are other physiological and anatomical indicators of incipient wilt (Kozlowski et al. 1962, Nair et al. 1967, Sachs et al. 1970, TeBeest et al. 1976, Pengelly et al. 1977).

External symptoms are diagnostic and include curling, browning, and other discoloration of leaves from the tip and outer margins to the midrib and base. Many leaves fall from the tree as symptoms progress from the top and outside of the crown downward and inward. Death follows

in short order. Symptoms in white oaks are considerably more variable (Boyce 1961, Jones 1958).

Oak trees of the red oak group, especially in the midwest, are commonly connected by intraspecific root grafts (Beckman and Kuntz 1951, Kuntz and Riker 1950, 1956). Grafts between members of the white oak group are rare, as are interspecific grafts. This phenomenom is often cited as an evolutionary advantage in alleviating water stress, transferring nutrients, and resisting windthrow (Bormann 1966, Graham and Bormann 1966, Kozlowski 1971), but it is this adaptation that C. fagacearum takes advantage of. When one tree dies, conidia are carried from the xylem of the dead tree through root grafts into the grafted tree by the transpiration stream (Kuntz and Riker 1956). Tyloses do not form in roots of oak wilt-killed trees, thus allowing the tree to tree spread (Struckmeyer 1954). C. fagacearum may survive several years in the root systems of killed trees (Yount 1955, Skelly and Wood 1974), but not usually in stems (Tainter and Gubler 1973). Tree to tree transmission is often termed local spread and results in the characteristic oak wilt foci or pockets that are especially notable in the midwest. Other areas such as the northeast (True et al. 1960, USDA 1957, Rexrode and Frame 1977), and Missouri (Jones and Partridge 1961, USDA 1958, 1959) have less root

graft spread.

New pockets are initiated by vector mediated overland transmission. The most common vectors in many areas are Nitidulidae beetles, which feed on the pungent oak wilt mats formed most often in the spring beneath the bark of oak wilt-killed trees (Curl et al. 1952). Pressure cushions associated with the mat crack the bark and allow entry to the beetles (Leach et al. 1952). Nitidulid beetles have been shown to spread both conidia and ascospores from mats to healthy trees (Bretz 1951, Norris 1953, Dorsey et al. 1953, McMullen et al. 1954, Morris 1955, Dorsey and Leach 1956, Jewell 1956). They also act as agents of spermatization, transmitting spores of different mating types between trees (Barnett et al. 1952, Dorsey and Leach 1956). Wounding of trees is necessary to provide entry points for the Nitidulids, and oak wilt has been a particular problem where construction or logging in the spring has wounded trees (Norris 1955, Jewell 1956, Jeffrey 1953, Drake et al. 1957, Craighead and Nelson 1960).

Oak bark beetles, which feed and breed in healthy and infected oaks, also transmit the fungus and are especially important in parts of the south, where mat formation is rare (Buchanan 1958, Rexrode and Jones 1970, Rexrode and Frame 1973). Other agents that have been implicated in spread include other insects, mites, and squirrels (Hime-

lick et al. 1953, Himelick and Curl 1956).

Vector transmission by Nitidulids is inefficient and highly variable, occurring mostly in the spring (Curl 1955). It is highly dependent on suitable weather conditions, especially winter precipitation (Boyce 1957a, Drake 1956, Houston and Kuntz 1960, Craighead and Nelson 1960). Still, much of the "local spread" that is usually attributed to root graft transmission may be a result of vector activity (see Jones and Bretz 1958).

The patterns generated by oak wilt are less diffuse than those of many other diseases, especially in areas where root graft transmission is more important (e.g. the midwest). Nevertheless, local expansion of a patch or pocket is fairly slow, around 5-10 feet per year radially in active stands in Wisconsin (Anderson and Anderson 1958), with the highest figures reported being about 30 feet per year in Minnesota (French and Bergdahl 1972). Overall amounts of damage vary widely, and interactions of oak wilt with other agents, such as the two lined chestnut borer, may be important (Nair and Kuntz 1975, Stambaugh et al. 1955). However, oak wilt does not require any klendusic (predisposing) factors to cause mortality.

A variety of conditions have been deemed important in favoring local spread in some areas; among them shallow soils (Gillespie and True 1959), northeast aspect (Gilles-

pie and True 1955), heavily watered soils (Houston et al. 1965), and moderate temperatures (Houston and Kuntz 1960). However, the importance of these factors may vary (True et al. 1960).

Ingrowth into oak wilt pockets has rarely been described, but varies with the site. In Central Wisconsin, sandy areas may not support tree regrowth, while other similar areas may be re-invaded by oak seedlings (sprouts from wilt-killed trees will inevitably succumb). Auclair (1975) describes the importance of openings presumably caused by oak wilt to Prunus serotina release in dry-mesic southern Wisconsin oak forests. Where oaks are being replaced by more tolerant species on mesic sites, oak wilt mortality will probably have little effect on species composition.

IV. BACKGROUND ON MODELS OF PLANT DISEASES

A model can be defined as a conceptualization, often in formal logical or mathematical form. It is an abstraction, a "homomorphism" (many models possible for one phenomenon) of some real system it represents (Ashby 1956, Patton 1971). The term "model" has a diversity of meanings, and models have a plethora of forms and uses, often being formulated with a particular use in mind (Reddingius 1974).

Kowal (1971) states that formulation of mathematical models requires that one operationally define the important real world variables, and precisely state the hypothetical relationships among these variables. The guidelines he establishes for choosing models are (1) naturalness with which the mathematical theory represents the real world phenomenon, (2) ability to generate predictions, (3) comprehensiveness and esthetics, (4) tractability of mathematics, and (5) consistency with other models (Kowal 1971). Other discussions of modeling may be found in Kranz (1974a), Patton (1971), Levins (1966), Watt (1961), and Pielou (1975).

A model is a description of a system; the latter has been defined as "a limited section of the real world ... (whose) ... limits are chosen in such a way that the environment of the system does not materially influence the

system (Zadoks 1971). By this definition, one could call many things systems and descriptions of them models. In the biological sciences, "model" is most often used to denote an abstraction expressed in terms of quantitative relationships. These are the models that will be reviewed in this section.

Robinson (1976) has provided an elegant formalization of the systems viewpoint in plant disease modeling. He defined a pathosystem as a subset of an ecosystem defined by parasitism. Just as ecosystems may be defined by whatever geographic and conceptual boundaries are convenient, so, said Robinson, may pathosystems.

The use of models and a systems viewpoint have been widespread in plant disease investigations. In order to fit the modeling approach used later in this paper into its proper context, a brief description of some of the general approaches of plant disease modeling is given here. This review does not intend to be a complete treatment of the diversity of models that have been used, nor is it intended to serve as a primer for certain types of models. The reader is referred to appropriate references, especially reviews, whenever possible.

A. Forecasting

Disease forecasting models are commonly used for

economically important crop diseases. Krause and Massie (1975), in a definitive review, distinguish two types: disease prediction and infection prediction. Both types attempt to predict the future and thus use different and more limited information than other types of models. Disease predictions can be made on the basis of infection information; hence infection prediction is a far more difficult endeavor.

Disease forecasting models commonly use the techniques of statistical models (next subsection). They are not usually mechanistic and are often empirical and deductive. This is in large part due to the nature of forecasting the future. Often, the key information is the current infection percentage or current magnitude of inoculum. Disease predictions are often made on a geographical basis, but can be modified to account for local conditions (Miller and O'Brien 1957).

Simple forecasting models utilizing phenological data often express output in terms of "negative forecasts" (see Bourke 1970). Examples of more complex models include those that predict epidemics of potato blight (Miller and O'Brien 1957, Schrödter and Ullrich 1966, Krause et al. 1975) and wheat stem rust (Calpouzos et al. 1976).

Forecast models are limited by the lack of good long term weather forecasting. Another problem is that huge

amounts of data are often recuired. Kanemasu et al. (1974) described the use of satellites to gather data from remote sites for use in the stepwise multiple regression models developed by Burleigh et al. (1972a,b, Eversmeyer and Burleigh 1970).

In summary, forecasting models have an important role to play in disease prevention (see Waggoner 1970), but their empirical nature precludes much use in providing fundamental information on disease dynamics.

B. Statistical Models

Forecasting models can be considered a subset of statistical models. In statistical models, the most common techniques used are single and multiple linear regressions that relate dependent variables such as mortality or the number of lesions to an array of independent variables, often meteorological or phenological. These models are not mechanistic per se, since a high correlation does not necessarily imply causality. They have been described as inductive, and not deductive (Watt 1961); rather demonstrative (DeWit 1968). The amount of explanation is low, and good fits may be accidental (van der Plank 1963). Their weaknesses are summarized by Butt and Royle (1974). They state that "the models make no provision for the effects of unexplained factors," although such flexibility would

only be earned at the cost of extremely high detail.

Still, such models can be very useful in prediction, and the regression techniques are commonly used on model subsystems, whose interactions within a larger system are defined more conceptually. Some examples of models that are statistically based include Massie (1973), Massie and Nelson (1973), Kerr and Rodrigo (1967), Watt (1961), Schrödter and Ullrich (1966), Royle (1973), Burleigh et al. (1972a,b), and Eyal and Zir (1974).

C. Mathematical (Conceptual, Functional) Models

Models that attempt to describe phenomena through equations based on realistic conceptual constructs make up this category. These functional models are usually based on explicit statements of biological processes and may often be explicative (DeWit 1968). The building of such equations usually follows a deductive pathway (see Watt 1961). Conclusions are dependent upon relationships and assumptions built into the model structure (Levins 1966).

Disease progress curves are the most empirical of the group. They describe the progress of an epidemic over time, usually as a percentage of host tissue affected. The basic form of the relationship follows certain assumptions related to the form of the equation used (Kranz

1974b). Some forms of disease progress curves are summarized in Jowett et al. (1974). Many other models are based on disease progress curves.

A tradition of epidemiologically oriented models owes much to the work of J. E. van der Plank (1960, 1963, 1965, 1968, 1975). His models are, for the most part, continuous and deterministic, consisting of differential equations that describe disease progress over time in terms of changes in populations of lesions of infected tissue (1963). The use of differential equations has the disadvantage of requiring constant values for epidemiological parameters (Zadoks 1971), but they are certainly more flexible than those derived from linear regressions (Caswell et al. 1972). Most of the equations are in the form of simple exponential and logistic relationships.

Van der Plank's models are biologically oriented, with explicit terms for latent period and infectious period, and adjustments for multiple infection. They also incorporate more theoretical terms that have since received wide attention in plant epidemiology. These terms include R, the basic infection rate (per unit of infectious tissue), and r, the apparent infection rate (per unit of infective tissue) (van der Plank 1975). Ultimately, it was van der Plank's ability to parameterize his models in meaningful ways, and his insistence on real world examples and appli-

cations, that helped his ideas gain acceptance in a science that was far from quantitative when his first book was published in 1963. The models have been widely modified for individual cases and uses (Kranz 1974b, Leonard 1969). Applications to native communities include a recent attempt to analyze the epidemiology of leaf spot disease in a native prairie in Saskatchewan (Howard and Morrall 1975).

Van der Plank (1959) notes that systemic diseases, such as oak wilt, behave somewhat differently than other diseases in terms of the epidemiological models. The rates of multiplication are slow, but this is balanced by severe damage resulting from each individual act of infection. Van der Plank's conceptualization (1975) of systemic diseases includes the analogy that infections are large lesions, i.e. the whole tree.

Kranz (1974a) has described epidemiology as the study of populations of pathogens in populations of host and the disease resulting from this interaction under the influence of environment and human interference. He concluded that this was a system (using the definition of Watt 1966). This approach focuses on populations of plants and parasites with an emphasis on looking at changes in numbers over time. Parasite-host models have long received attention as specialized types of predator-prey

models (see discussions in Poole 1974 and Pielou 1977).

The simplest class of such mathematical models are referred to as Lotka-Volterra models (Lotka 1925) and describe changes in predator and prey numbers, where there is no density dependence on population growth. Leslie and Gower (1960) added an upper limit to the predator population and density dependence in the prey population. A plethora of ecological models have been derived from the Lotka-Volterra formulation and include stochastic versions, upper limits to parasite attacks, time lags, and non-linear forms (Holling 1965, Pielou 1977, May 1973, Watt 1959, McDonald and Cheng 1970). For example, Hassell and May (1973) have constructed host-parasite models that showed how some degree of spatial or temporal asynchrony contributed to stability of the system, and these results have been borne out by the experiments of Pimentel and associates (Pimentel et al. 1963, Pimentel and Stone 1968, Pimentel 1977). With similar models, Chilvers and Brittain (1972) showed that plant competition can be mediated by host-specific parasites. The same model run without parasites did not reach equilibrium.

Not all ecological host-parasite models lead to a single equilibrium. May (1977) and Ludwig et al. (1978) have constructed models that result in multiple stable configurations and applied them to host-parasite systems.

The system will tend toward an equilibrium point or neighborhood unless perturbed sufficiently to enter a new neighborhood with a different equilibrium (see also Holling 1973). Some such systems may show a predictable oscillation between alternate stable states.

Geneticists have constructed host-parasite models analogous to the ecological ones, but they will not be touched on here. For further information see Flor (1955), Mode (1958), Person (1966), and Stern (1972).

A somewhat different approach uses life tables either to summarize information on the kinds of mortality affecting a host population or to follow a pathogen population. In forests, examples include work on the white pine weevil (Waters 1969), spruce budworm (Morris and Miller 1954, Morris 1963), and gypsy moth (Campbell 1967).

Many of the models mentioned in this section combine the deductive approach with statistical treatments of model components (see Analytis 1973). The divisions made here are somewhat artificial, and the best modeling route will traverse many territories (see Watt 1961).

D. Simulation Models

When a model or series of models is constructed and solutions are found for state variables over time, one could say that simulation has occurred. In fact, Waggoner

(1974) conceived of simulation as a logical assembly of knowledge of interrelationships among components. However, when a model is simple and deterministic, the "simulation" is merely the solution(s) to the equation(s).

In an attempt to introduce more realism into their models, some biologists have constructed stochastic models. The introduction of these "dice throws" into the process makes analytical solution of equations less meaningful, since results can vary even with identical parameters. Even solution of many complex deterministic models may be unreasonably difficult (Watt 1961). One solution is to experiment with the model by looking at the statistics of a number of "typical results." This simulation also has advantages if experimentation in the field or laboratory is not feasible (Kranz 1974a). With the advent of modern computers, simulation modeling has become quite accessible to biologists. This technique can have a feedback effect, since the increase in the use of computers has modified approaches to experimental work necessary to obtain data (Watt 1966, Waggoner and Horsfall 1969, Mogk 1974).

Simulation models in epidemiology have been based on statistical approaches and more mathematical formulations and include many forecasting models. Often a combination of the two approaches is used, with prudent use of statistics bridging the gulfs between peaks in conceptual know-

ledge. Simulation models have the considerable advantage of allowing changes in parameters during simulation, although such changes must usually be implemented in discrete time (Zadoks 1971).

Zadoks (1971) gives examples of simulation models based on van der Plank equations whose parameters are allowed to vary during simulation. His units are infection sites that have various conditions representing operationally defined segments of the infection process. Changing values of the variables modified properties of the generated epidemic progress descriptions.

One of the most detailed deterministic simulation models available was developed by Waggoner and Horsfall (1969) to forecast losses due to Alternaria solani, the early blight of tomato and potato. EPIDEM, as the computer program was called, was built from the bottom up on results of physiological laboratory experiments. The simulator integrated over 3 hour intervals and incorporated many climatic variables. These variables had different effects on the eight stages of the life history of Alternaria that were included in the model conceptualization. The authors get good results with the model and proceed to perform factor analyses in an attempt to pick out the most important biological factors. This extremely detailed, albeit reductionist, approach showed how the specific

modeling procedure led the authors to perform particular experiments that had not been previously considered. A similar model for southern corn leaf blight was written by Waggoner et al. (1972) with excellent results, according to these authors. These results have been challenged by van der Plank (1975), who lists four recommendations for simulation. Among them is a recommendation that more attention be given to initial inoculum levels, a recommendation that would make these models more empirical than they now are. A similar simulation model that was stochastic was formulated by Massie (1973).

Watt (1963) used a deterministic simulation model to predict results of different levels of control of insect pests. He found that large scale short term eradication resulted in higher long term (60 years) defoliation than strategies for control that were less severe.

E. Models Treating Spatial Pattern

Models that consider spatial pattern explicitly are especially appropriate when considering non-mobile organisms, such as plants. The work of Gregory (1948, Gregory and Read 1949) is noteworthy. He interpreted the flattened gradients of infection vs. distance seen in systemic diseases, as compared to non-systemics, as being due to multiple infections. He advanced multiple infection transfor-

mations as an explanation for the highly variable parameters in the mass of empirical dispersal equations developed by Wolfenberger (1946, 1958). Cammack (1958) looked at these considerations for a non-systemic disease. Later work by Gregory (1968) consisted of manipulations of the various dispersal-distance equations. The amount of localness in the source of infection, the type of source (point or diffuse), the strength of the source, the amount of secondary spread, and the amount of background contamination were some of the factors that affected the terms in Gregory's equations.

Waggoner (1952) fit these equations to the number of leaflets infected by distance from a point source for potato blight. Further work (1962) considered spore deposition explicitly, but the highly variable nature of meteorologic phenomena limits the precision of these and similar models.

Van der Plank (1948, 1960) considered patterns of spread in relation to distance. Widely separated fields of plants were more likely to show discontinuous spread and more slowly developing epidemics. He recognized that spatial effects on rates of spread can modify results obtained from his and others' non-spatial models. He also stated that spatial effects were more important in epidemic than endemic diseases (van der Plank 1975), but this prob-

ably holds best for large inter-field scales. At smaller scales, distance effects are important in systemic disease dynamics (Zentmeyer et al. 1944).

Pielou (1965) looked at patchiness in disease spread in forest trees. Estimation of two important parameters — the increase in the proportion of total area occupied by patches and the increase in proportion of within patch trees that are diseased — were made by analyzing results of transects through douglas fir (Pseudotsuga menzezii) plantations infected with Armillaria mellea, a root rot. Statistics based on the probabilities of "runs" of various lengths of diseased trees enabled Pielou to test several hypotheses concerning spatial disease spread.

F. Forest Disease Models — Some Examples

Being of generally less economic importance than their agricultural counterparts, forest pathosystems have received less attention by modelers. There are, however, several notable examples of recent application of quantitative techniques to forest systems.

In studies derivative of the work of van der Plank, Merrill (1967a) calculated values for r_1, the rate of disease increase, for oak wilt epidemics in Pennsylvania and West Virginia. From these values, he predicted the severity of damage in the future. Use of the van der

Plank equations, in this case, required many simplifying assumptions. It should also be noted that control efforts that were statewide in nature took place during the time in which data for the model were collected.

Merrill (1967b) also analyzed many other epidemics of forest diseases in a similar way. He found that high values of r were consistently associated with the introduction of an exotic pathogen to the system. Van der Plank (1959) points out that r values for systemic diseases are usually low, although oak wilt in Merrill's study (1967b) is an exception.

May (1977) looked at a model of spruce budworm dynamics which incorporated the budworm, its parasite, and the vegetation fed on by the insect. Many stable solutions were found, the number depending on the level of parasitism of the insect. May used this information to explain the cyclic budworm epidemics, as well as the failure of control, which tends to hold the system on the brink of a major change (but not forever).

Ludwig et al. (1978) provided further analysis of this system. Their analysis included division of state variables into fast and slow categories. Changes in fast variables were examined with slow variables held constant, and then slow variables were modeled using the equilibrium solutions of the differential equations for fast variables.

The analysis included solution with both qualitative and quantitative data, and the results were similar to those obtained by Morris' (1963) more detailed simulation model.

The work of Pielou (1965) with models considering spatial pattern has been mentioned. Older work by Zentmeyer et al. (1944) on distance relationships in dutch elm disease showed that the probability of infection decreased with the log of distance from the disease source. A few other forest pests have been considered with a spatial emphasis, among them dwarf mistletoe (Gill and Hawksworth 1961).

Campbell and associates (Campbell and Valentine 1972, Valentine and Campbell 1975, Campbell and Sloan 1977) have studied gypsy moth and have erected a stand level simulation model that describes losses due to gypsy moth infestation. It was based on data collected between 1911 and 1931. Important sections of the model included empirical defoliation estimates, estimations of tree conditions, and mortality, which was calculated using transition probability matrices (Campbell and Valentine 1972) and regression derived egg mass density estimates. The authors considered the model's main use to be as an aid to gypsy moth suppression, especially over short time periods.

A more oblique approach, from the point of view of

this review, is exemplified by the forest succession model (FORET) of Shugart and West (1977). The FORET model was run for forests representing pre- and post-chestnut blight forests; the latter lacking the species entirely. Despite the fact that the removal was implied to be instantaneous, the model results fitted known data well and predicted long term community changes in the absence of chestnut.

G. Characteristics of OAKWLT

The simulation model that is described in subsequent sections of this thesis is called OAKWLT. It predicts patterns of oak wilt mortality in space in time. The model combines many features of models mentioned in this review.

The model is empirically based, utilizing data from a variety of sources. However, the basic functional steps in oak wilt spread were modeled separately and combined based on a conceptual understanding of the disease. Data were found for these components of the oak wilt dynamic, and predictive equations for these submodels were formed from regressions and other techniques.

OAKWLT is a stochastic model; the results vary even with same sets of input parameters. The choice of a stochastic form reflects the belief that the exact patterns

of spread are highly variable, but that the overall results are predictable within reasonable limits. In addition, several key parts of the model are most directly expressed in terms of probabilities, making a stochastic model most appropriate.

This simulation model uses a discrete time step of one year. This allows use of a large amount of data which does not describe within year dynamics. It also provides for comparison with much of the descriptive data on total mortality and pocket size that will serve as a partial validation of the model. A detailed description of the model is found in section VII, with simulation results following in section VIII. Before this stage is reached, an analysis of oak wilt data is presented in the next two sections.

V. METHODS OF DATA ANALYSIS

There were two general goals of the data analysis. The first was to provide a fundamental description of the oak wilt system. The second was to quantify functional information on oak wilt spread, providing the basis for a computer model that would simulate patterns of oak wilt mortality.

In this analysis, three types of data were used. Two are particular data sets that were analyzed by the author; one termed Sinnissippi data and the other southern Wisconsin data. A considerable amount of information has also been gathered from literature sources.

A. Sinnissippi Data

1. Description of Site

Sinnissippi Forest is located along the Rock River in Ogle County near Oregon, in northwestern Illinois, 40 miles south of the Wisconsin border. This portion of Illinois is adjacent to the Driftless Area of Wisconsin and three other states (Hartley 1966, Cahayla-Wynne and Glenn-Lewin 1978). It is in the region of Illinoian drift, west of the Bloomington Moraine left by the Wisconsin drift (Martin 1965). Sandy loams make up about half the soils, with most of the rest being sands and silt loams (USDA, Soil Conservation

Service, J. Conroy, personal communication).

Native hardwood forest, mostly dominated by oak, cover 1648 of the 2300 acres of forested land (Himelick and Fox 1961). Species of oak found include Quercus velutina Lam. (black oak), Q. rubra L. (red oak), Q. ellipsoidalis Hill (northern pin oak or Hill's oak), Q. alba L. (white oak), and Q. macrocarpa Mich. (bur oak). See Appendix A for a listing of tree species names used. The area has been intensively managed since 1938, first by the Dept. of Forestry, University of Illinois, and more recently by private concerns. Almost all parts of the forest have been selectively cut at one time or another, and selective cutting is now proceeding on a 10 year rotation (D. Stenger, personal communication).

The forest is divided into 40 acre compartments for management and research purposes. Surveys, using permanent one fifth acre plots, have been undertaken since 1949, and most recently in 1972, to inventory the stands. Contiguous areas were assigned to twenty year age classes, and categorized by productivity and composition.

2. Description of the Data Set

Starting in 1954 and continuing through 1971, E. B. Himelick and associates collected data on oak wilt mortality in Sinnissippi forest. Ground and air surveys were

undertaken up to six times a year, to identify dead and dying trees. Laboratory tests elucidated the cause of death in trees with atypical symptoms. In addition to this survey, Himelick and Fox (1961) used a variety of oak wilt control methods, although some areas of the forest were left untreated in some years as check plots. Control methods included sanitation, girdling, and poisoning of wilt-killed trees and adjacent trees of the red oak group. The distances from wilt-killed trees over which control was attempted varied in different years. Surveys and control activities were halted in 1971. For more information, see Himelick and Fox (1961).

The following data (if appropriate) were taken for each tree that died from oak wilt: species, size, compartment, pocket, year of death, timber type, method of infection, size and species of presumed donor (or source) tree in root graft spread, and the distance over which root graft transmission had taken place. Some of the data were incomplete. Method of transmission refers to whether the tree was believed to have been infected by root graft transmission, vector, or an unknown source. The frequency of surveys makes this data set noteworthy, for it often allowed differentiation by symptoms between the two types of infection. Vector infection will often show initial symptoms on one branch only, while root graft transmission

will usually show symptoms throughout the upper part of the crown, or perhaps on one side. Since the rapidity of spread of Ceratocystis through the vascular system is notable, infrequent surveys are less successful in determining method of infection.

The approximate locations of oak wilt-killed trees were marked on detailed individual 40 acre compartment maps. The pocket to which a tree belonged was noted; a pocket including roughly any tree within 50 feet of a previously wilting tree. Data were also taken on the type of control measures taken, the species and size of trees affected by the control, and the board foot yield of the harvested material.

Himelick and Fox (1961) published results from their analyses of data taken prior to 1961, with emphasis on the success of oak wilt control measures. In 1977, the data set was turned over to this investigator for compilation and summarization. Results bearing on oak wilt control will be published by Himelick and Fox. The following methods describe work done by the author pursuant to the goals of this thesis.

Some data summarizations were made before the data were entered onto magnetic tape. Except for counts of pockets, trees were ignored that did not have a dbh listed. This most often happened in areas of reproduction or 'brush

areas.' Therefore, the data base was less accurate for smaller trees. Different control treatments were lumped into the category "control," although control measures varied in different years. Red, black, and northern pin oaks were lumped under the flag "red oak group." Sprouts from oak wilt-killed trees were ignored, since they invariably succumb to the disease. Finally, double-stemmed trees were entered as a single tree with a dbh equal to that of the largest stem.

3. Data Analyses — An Overview

The data from this set (and other sets also) were divided into four parts: total mortality, pocket dynamics, root graft transmission, and vector spread. The first two types of data are descriptions of mortality patterns that are results of both root graft transmission and vector spread, and their interactions with characteristics of the site. These data are not used to generate simulation model parameters, and thus can serve the function of a limited validation (limited because a close fit only implies the model can mimic conditions at Sinnissippi and the other specific sites). For these data, the goals are description of the phenomena and statistical testing of differences resulting from various conditions of composition, site, and age.

This type of testing also is a part of the analysis of root graft transmission and vector spread. However, these two types of spread are also functional categories, and quantification will allow simulation of oak wilt dynamics. As such, emphasis is placed on the formulation of predictive equations and tests of significance related to these equations.

B. Southern Wisconsin Data

1. Description of Sites

J. E. Kuntz and associates made complete mappings of plots at four sites in southern Wisconsin in the late 1940's. Two sites were in Sauk Co. and two were in Dane Co.

The Devil's Lake site, in Sauk Co., was situated in mature mesic oak woods at the southern end of Devil's Lake. The area is part of the complex outwash and moraine system described in Martin (1965). Soils are silt loams, moderately drained with a moderate water holding capacity.

The other Sauk Co. site, Miller's Farm, was located west of Devil's Lake on a droughty ridge in the unglaciated Driftless Area. The silt loam soils are shallow, with much exposed rock, and have a fairly low water holding capacity.

One of the Dane Co. sites was located in the Univ. of Wisconsin-Madison Arboretum in Gallistel Woods. The dry-

mesic oak woods is on silt loams of moderate permeability, water holding capacity, and depth. The fourth site was in the Nakoma area and was referred to as West Hill. It supports dry-mesic oak woods, and its silt loams are somewhat drier than the Arboretum's. Both Dane Co. sites were covered by the Wisconsin drift.

2. Description of the Data Set

Patterns of oak wilt mortality were followed in these four areas for up to seven years. No differentiation was made between root graft and vector spread. Species, size, and year of death were recorded.

The mapped data were turned over to this investigator in 1978 for summarization and analysis. Lines connecting 'most likely' paths of root graft spread were drawn from the temporal patterns of mortality. The criteria used in deciding 'most likely' were shortest distance, shortest time interval for spread, and same species, in that order. There is no guarantee that more circuitous routes of local transmission did not occur, but this is the same problem that field investigators have to deal with. Their criteria for identifying specific routes of transmission are undoubtedly similar. In any event, ability to mimic the resulting patterns depends on finding consistent correlations.

For each presumed transmission, the species involved, their dbh's, the inter-tree distance, and time to spread were noted. This was the record of successful transmissions.

Since the areas were completely mapped, the occasions when there was no spread were also apparent. This is an item that the Sinnissippi data lacked. Distances, species and sizes of all trees within 35 feet of wilting trees were noted for instances of non-transmission. Since the studies were short term, the length of time over which spread could have occurred, and did not, was noted.

C. Literature Data

A thorough search of all available oak wilt literature was completed, with representative data being garnered from several geographical areas. Most of the numbers concerned mortality and were on a regional basis. It was the lack of functional data needed as a basis for the simulation model that led to the more detailed analysis of the two data sets described above.

D. Data Analyses - Total Mortality

The number of dead trees was summarized by the following attributes for Sinnissippi Forest data: compartment (adjusted for % oak forest), species, method of

infection, the presence or absence of control measures in the pocket prior to infection, year of death, composition class, site productivity type, soil type, and size of tree.

To detect relationships of the above attributes to oak wilt mortality, Chi square tests were used to detect differences in mortality as each attribute was varied, using observed and expected values. Observed data usually consisted of the number of dead trees found in the 17 year record of oak wilt mortality at Sinnissippi. The expected values were derived from 1972 inventory data. The assumption was that the 1972 timber inventory represented typical conditions between 1955 and 1971. Because of this approximation, only large areas of the forest were compared. Since all comparisons use a common ground, the test is reasonable in a relative sense.

The number of degrees of freedom was always the number involved in that particular test (always ≤ 3), despite the fact that a piece of raw data might be used in several tests. This liberal determination of degrees of freedom means that only very significant differences ($p < .01$) should be accepted as significant.

Cutting and oak wilt control have certainly had an effect on characteristics of the forest, most notably on tree size, so comparisons are made cautiously. Table 1 shows the cutting regime at Sinnissippi, which has been

TABLE 1. Cutting Regime in Sinnissippi Forest, by Compartment (1951-1972)

	YEAR(S) OF SELECTION CUT																						
	5	5	5	5	5	5	5	5	5	6	6	6	6	6	6	6	6	6	7	7	7
COMPARTMENT(S)	1	2	3	4	5	6	7	8	9	0	1	2	3	4	5	6	7	8	9	0	1	2
90,124,155,157		X								X												
119,120,127,128	X										X											
189	P	*P									X											
190		X									X											
96,123,191,213,218	X											X										
160	X									X												
125,126			X							X												P
91,92,121,122			X									X										
93			X										X									
94			P	P										X								
82,83,95,97				X										X								
156			X												X							P
84				P	P										X							
79,80,81				X											X							
85					X											X						
86					P	P										X						
87						X										X						
57,58,59						X											X					
64							X										X					
60							X											P	P			
61							X												X			
62,63							X													P	P	
132							X														X	
116,117,130,131								X														
111,135,136,158									X													
185,186,187									X													
159										X												
212												X										
49,76,102,103													X									
50													P	P								
100,101															X							
35,36																X						
66,77,78																		X				
52,154																						X

* partial cut

Compartments 45, 46, 51, and 67 were not cut between 1951 and 1972.

fairly constant and has aimed at a sustained yield on a 10 year partial cutting rotation. This cutting has taken place since the 1940's (Fox 1972). Table 2 shows the size class distribution of the red oak group in 1972, subdivided into such distributions based on time since the last cut. The size class distributions, which were not significantly different from normal under the Kolmogorov-Smirnov test at its most stringent alpha level (Sokal and Rohlf 1969), are related to time since last cut. The cuts were partial, not eliminating all the larger trees, but shifting the average size downward. Knowledge of the cutting regime can help interpret results based on the assumption that the 1972 size class distribution is representative. It does not appear that species composition has undergone any major shifts during the period of the oak wilt study.

Literature values for mortality are also presented. No southern Wisconsin data is included in this section.

E. Data Analyses — Pocket Dynamics

Pocket, or patch, dynamics refers to the formation, initiation, or birth of new pockets; the change in size ('growth') or other characteristics of a pocket with time, and the 'death' of the pocket, defined here as the cessation of expansion.

Rates of pocket birth are presented for blocks of

TABLE 2. Size Class Distribution of Trees of the Red Oak Group, Sinnissippi Forest, 1972, by Time Since Last Cut

LAST CUT	#1/5 acre plots	DENSITY/ACRE IN SIZE CLASS (inches) 3.5-8.4	8.5-9.9	10.0-12.4	12.5-14.9	15.0-17.4	17.5-19.9	20.0-22.4	22.5-24.9	25.0-29.9	Mean Size	Stand Dev.	Total Density
1956 or before	35	8.86	5.43	9.29	7.29	5.71	3.71	3.00	0.71	0.00	12.48	4.80	44.00
1957-1959	105	18.71	0.86	1.86	2.71	3.52	2.48	1.90	0.90	0.48	10.62	6.06	33.42
1960-1961	76	25.19	4.54	8.22	4.21	2.83	1.91	0.39	0.33	0.14	9.08	4.65	47.76
1962-1963	83	20.66	3.86	6.20	5.60	3.07	1.88	0.72	0.18	0.12	9.75	4.47	42.29
1964-1965	62	15.89	3.15	3.31	3.06	2.02	2.26	1.37	0.32	0.24	10.15	5.14	31.62
1966-1967	41	31.58	1.34	3.29	6.71	4.15	2.32	1.71	0.42	0.00	8.52	6.38	51.59
1968-1971	45	29.56	0.22	1.22	1.67	2.33	0.89	0.22	0.00	0.77	7.99	4.58	36.88
ALL	447	21.30	2.70	4.56	4.08	3.15	2.16	1.25	0.46	0.27	9.88	7.78	39.93

Note: All distributions not sign. diff. from normal (see text)

various sizes in Sinnissippi and compared to literature values. Spatial patterning of new pocket initiation was examined by following changes in numbers of new pockets in local areas. Run tests (Sokal and Rohlf 1969) were used in an attempt to look for temporal patterns. Alternative methods of identifying return times for high pocket formation were also explored. Pocket growth and mortality as a function of pocket size and age were summarized from Sinnissippi data and sparse literature sources. Other literature data presented include radial expansion rates of pockets and average patch size. Southern Wisconsin data were not used for patch dynamics.

F. Data Analyses — Root Graft Transmission

1. Prediction of Root Graft Transmission Probability — Southern Wisconsin

The numbers of trees that died from root graft transmission of Ceratocystis fagacearum are summarized under total mortality. What is of more interest, especially for use in the functional model, are the factors that influence the probability of root graft transmission between two trees.

The first goal is the formation of predictive equations for root graft transmission between two trees.

Southern Wisconsin data included instances of transmission and non-transmission between two trees, where the sizes, species, inter-tree distances, and time taken by spread (or non-spread) were known. Site specific root graft transmission probability tables were formed directly from such data. These were time specific. For example, the two year table for a species combination at a site shows the probabilities of transmission in the second year. This equals the observed transmissions that took two years divided by the total number of possible spreads that could have been observed in two years' time. This would not include transmissions that took less than two years or cases where there was not two years of observation after the first death. The two year table shows these probabilities for each combination of combined diameter class (five inch intervals) and distance class (five foot intervals).

Time independent probabilities of root graft transmission were also calculated for southern Wisconsin data. These were overall probabilities of spread, by species, combined dbh, and inter-tree distance, regardless of how long. They are calculated by the following formula:

$$P_{ti} = P_n(1-P_{n-1}(1-P_{n-2}(\ldots 1-P_0)\ldots)) +$$

$$P_{n-1}(1-P_{n-2}(1-P_{n-3}(\ldots 1-P_0)\ldots))+\ldots+P_1(1-P_0)+P_0.$$

(1) where P_{ti} = time independent probability of transmission in n years
P_n = time specific probability of transmission in nth year
$0 \leq n \leq 4$, n = 0 is same year.

The formula merely calculates what percentage die in the same (=0) year as death of the donor tree, multiplies the remainder alive by the proportion dying in year one, and so on through all the years. This was done for each species, combined dbh, and distance for which there was minimum data.

Single linear regressions, fit by least squares, were carried out on the following models of combined dbh ('DBH') and inter-tree distance ('D'), which are used to predict probability of root graft transmission ('p'):

(2)
(1) $p=m(DBH/D)+b$
(2) $p=m(DBH-D)+b$
(3) $p=m(DBH)+b$
(4) $p=m(1/D)+b$
(5) $p=m(DBH/D^2)+b$
(6) $p=m(DBH^2/D)+b$
(7) $p=m(0)+b$ (1:1)
(8) $p=m(0)+b$ (2:1)

where m is the slope of the regression line and b is the Y intercept.

The first two cases consider situations where a linear combination of the two variables is important. The level of root graft transmission is directly proportional to the sizes of the two trees and inversely

proportional to the distance between them. Models 3 and 4 are null hypotheses of sorts that consider one factor alone to be an adequate predictor. Models 5 and 6 consider one of the factors to be more important by a power of two.

The last two models in Equations 2 are overlap models, based on an area overlap of two circles with radii proportional to tree size and inter-center distance proportional to inter-tree distance. Similar measures have been used in competition models: equations predicting growth or mortality based on the spatial pattern and properties of neighboring trees (Arney 1973, Martin 1978). The overlap model was calibrated to yield zero probability of root graft transmission at distances corresponding to maximum inter-tree distances listed in Himelick and Fox (1961). Individual tree diameters were not used, however, since this would have diluted the probability data. Instead, combined dbh was used, and the overlap model considered with diameters of individual trees assumed either to be equal (#7), or in a two:one ratio (#8).

2. Prediction of Root Graft Transmission Probability — Sinnissippi

The number of transmissions, by species and classes of combined dbh and distance, was calculated for Sinnissippi Forest. However, trees that did not die of oak

wilt were not mapped; thus there was no record of instances of non-transmission. To directly calculate probabilities of root graft transmission, one must know when it did not occur. This was approximated by indirect methods.

If one assumes that oaks are randomly distributed, a reasonable assumption for mature midwestern forests, one can estimate from inventory records the sizes and densities of stems expected in an area of a given size. Data from the 1972 inventory was used to this end, with the assumption that 1972 conditions were representative of the forest integrated over 1955-1971.

The 1972 size class distributions were multiplied by themselves to form matrices, or tables, of densities of two tree size combinations. The results for the red oak group in the whole forest are shown in Table 3. These matrices may be termed expected combined dbh distributions.

Expected combined dbh distributions were initially calculated in terms of densities per acre. These were then converted to densities in a five foot circle, densities between five and ten feet, between 10 and 15 feet, etc. Table 4 shows this result for the data in Table 3. The results are matrices whose rows represent different combined diameter classes, and whose columns represent distance intervals. The elements of the matrices are the number of trees of the combined dbh class specified by

TABLE 3. Illustration of Methods Used in Obtaining Expected Combined DBH-Distance Distributions.
I. Calculation of Number of dbh Combinations per Acre.

Mean Size Class		5.95	9.20	11.20	13.70	16.20	18.70	21.20	23.70	26.20	28.70
	Density	21.3	2.70	4.56	4.08	3.15	2.16	1.25	0.46	0.19	0.08
Mean Size Class	Density										
5.95	21.3	453.7*A**									
9.20	2.70	57.5B	7.29B								
11.20	4.56	97.1B	12.3C	20.8C							
13.70	4.08	86.9B	11.0C	18.6C	16.6D						
16.20	3.15	67.1C	8.50D	14.4D	12.9D	9.92E					
18.70	2.16	46.0C	5.83D	9.85D	8.81E	6.80E	4.67F				
21.20	1.25	26.6D	3.38E	5.70E	5.10E	3.94F	2.70F	1.56G			
23.70	0.46	9.81D	1.24E	2.10E	1.88F	1.45F	0.99G	0.58G	0.21G		
26.20	0.19	4.05E	0.51F	0.87F	0.77F	0.60G	0.41G	0.24G	0.08G	0.04H	
28.70	0.08	1.70E	0.22F	0.36F	0.32G	0.25G	0.17G	0.10G	0.04H	0.02H	0.01H

*expected number of combinations per acre

**combined dbh class, see Table 4.

TABLE 4. Illustration of Methods Used in Obtaining Expected Combined DBH--Distance Distributions. II. Expected Combined DBH's at Various Distances.*

CDBH Class	Expected No. Trees Per Acre	Expected No. Trees Within (ft): 0-5	6-10	11-15	16-20	21-25	26-30	31-35	36-40	41-45	46-50
11-15(A)	453.7	0.81	2.45	4.09	5.72	7.35	8.98	10.62	12.25	13.88	15.56
16-20(B)	248.8	0.45	1.35	2.24	3.13	4.03	4.93	5.82	6.72	7.61	8.53
21-25(C)	175.8	0.32	0.95	1.58	2.22	2.85	3.48	4.11	4.75	5.38	6.03
26-30(D)	104.5	0.19	0.57	0.94	1.32	1.69	2.07	2.45	2.82	3.20	3.58
31-35(E)	48.8	0.09	0.26	0.44	0.61	0.79	0.97	1.14	1.32	1.49	1.67
36-40(F)	17.4	0.03	0.09	0.16	0.22	0.28	0.34	0.41	0.47	0.53	0.60
41-50(G)	5.51	0.01	0.03	0.05	0.07	0.09	0.11	0.13	0.15	0.17	0.19
51+ (H)	0.11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	Conversion Factor from Acres	0.00180	0.00541	0.00901	0.0126	0.0162	0.0198	0.0234	0.0270	0.0306	0.0343

* This matrix is termed the $\tilde{E}$ matrix. See text.

the row that might be expected within the distance class given by the column. Such matrices are termed the expected combined dbh-distance matrices ($\tilde{E}$). Each element $e_{i,j}$ represents the expected number of trees of combined dbh i in distance class j.

Expected combined dbh-distance matrices were calculated for the following large areas of Sinnissippi:

1) Whole forest
2) Combined Timber Type Oak Poor
3) Combined Timber Type Oak Medium
4) Combined Timber Type Oak Good
5) Combined Timber Type Reproduction

While the $\tilde{E}$ matrices, which summarize the expected characteristics of neighboring trees, were based on an inventory sample, Sinnissippi Forest data were complete for instances of successful root graft transmission. The number of transmissions were arranged by combined diameter and distance classes for the following categories:

1) Whole forest - all mortality
2) Whole forest - mortality in previously controlled pockets
3) Whole forest - mortality in previously uncontrolled pockets
4) Whole forest - elapsed time for spread, same year
5) Whole forest - elapsed time for spread, one year
6) Whole forest - mortality in two years
7) Whole forest - mortality in three years
8) Whole forest - mortality in four or more years
9) All mortality in Oak Poor
10) All mortality in Oak Medium
11) All mortality in Oak Good
12) All mortality in Reproduction

The results are mortality matrices ($\tilde{M}$) of actual deaths in various combined dbh and distance classes; the classes being identical to the classes used in the $\tilde{E}$ matrix (see Table 5). Each element $m_{i,j}$ represents the number of dead trees in combined dbh class i and distance class j. Categories 1-8 (above) consider mortality in the entire forest, with the corresponding expected size-distance matrices ($\tilde{E}$) derived from the size distribution as a whole. Categories 9-12 consider mortality in areas of smaller size, and the corresponding $\tilde{E}$ matrices are derived from inventory data in those forest subsets. Categories 4-8 are time specific, the remainder are time independent.

A measure of the relative efficacy of root graft transmission for a given combined dbh-distance class is the number of successful transmissions relative to the number of possible transmissions, or:

$$t_{ij}=m_{ij}/e_{ij} \tag{3}$$

where t_{ij} is an element in a new matrix of relative efficacy of root graft transmission ($\tilde{T}$)
m_{ij} is an element in the mortality matrix ($\tilde{M}$)
e_{ij} is an element in the expected combined dbh-distance matrix ($\tilde{E}$)
and i is a combined diameter class
j is a distance class

Each element in the matrices on the right hand side of the equal sign will have a corresponding element in the $\tilde{T}$ matrices (see Table 6). This latter set of matrices is

TABLE 5. Number of Trees Dying by Root Graft Spread, by Combined DBH and Distance Classes, Sinnissippi Forest 1955-1971.*

Combined DBH Class (inches)	DISTANCE CLASS (ft) 0-5	6-10	11-15	16-20	21-25	26-30	31-35	36-40	41-45	46-50
11-15	4	9	5	5	0	1	0	0	0	0
16-20	4	17	23	12	6	3	1	0	0	0
21-25	3	22	32	20	19	11	3	3	0	0
26-30	4	34	17	20	16	11	4	7	1	0
31-35	2	15	17	12	5	7	5	6	0	0
36-40	0	5	5	3	2	1	0	2	1	4
41-50	0	2	0	4	2	3	1	0	0	0
51 +	0	0	0	0	0	0	0	0	0	0

* This matrix is termed the $\tilde{M}$ matrix. See text.

TABLE 6. Relative Efficacy of Root Graft Transmission, Sinnissippi Forest*

Combined DBH	DISTANCE 6-10	11-15	16-20	21-25	26-30	31-35	36-40**
11-15	3.67	2.23	0.874	0	0.111	0	0
16-20	12.6	10.3	3.84	1.49	0.609	0.172	0
21-25	23.2	20.3	9.01	6.67	3.16	0.730	1.06
26-30	59.6	18.1	15.2	9.47	5.31	1.63	2.48
31-35	57.7	38.6	19.7	6.33	7.22	4.39	4.54
36-40	53.2	31.2	13.6	7.14	2.94	0	4.26
41-50	---***	---	58.0	22.5	27.3	7.69	0

*This matrix is termed the $\tilde{T}$ matrix. See text.

**Only distance up to 40' were used due to small data at greater distances

***Only matrix elements for which the expected value was ≥ 0.03 were used (see Table 4)

analogous to the probability matrices formed directly from southern Wisconsin data. Despite the analogy, the elements of the $\tilde{T}$ matrices are not true probabilities, since they are based on the size and distance data derived from the 1972 inventory. However, the values should be directly related to probabilities, and thus comparisons of different $\tilde{T}$ matrices are appropriate.

The elements of the $\tilde{T}$ matrices were treated as dependent variables that could be predicted by the eight models of combined dbh and distance that were described previously.

3. Comparisons of Regression Parameter Estimates

As well as providing a basis for prediction, the regression equations for local spread using the eight models can yield additional information. It is of some interest to determine which models fit the data best and to examine the consistency of their performance on the various subsets of the data.

It is also constructive to compare the values of the regression parameter estimates. In order to do this, arc sine transformations of the data were performed to homogenize the variance over the range of probability values (Y) and allow determination of confidence belts (Snedecor and Cochrane 1967). Such confidence belts (five percent)

were compared to each other, using models that fit the data well.

Different sites, within both southern Wisconsin and Sinnissippi data sets, were compared to one another in terms of regression parameter estimates, to see if such relationships were site specific. In addition, different time specific spreads at the same site were compared. If the size/distance/probability relationships do not change with differing time to spread, then the regression lines should be similar.

When data from two sites, or times to spread, are highly correlated to the same model, this implies that the underlying mechanisms for spread are consistent. When using the same model, if the intercepts are significantly different and the slopes similar, one could say that tree size and inter-tree distance are scaling the same with probability of spread, and only overall levels of transmission are different between the sites. This overall level might reflect the time oak wilt has been at a site. If two slopes are different, this implies a more fundamental difference, i.e. a given change in tree size or distance has contrasting effects on the dynamics of spread in the two sites. Examination of the regression parameters was interpreted in this light: what does a particular best fit tell us about the dynamics of oak wilt spread?

4. Other Root Graft Transmission Analyses

A direct approach to the problem of predicting time between successive tree deaths in root graft transmission was undertaken. Tree size and inter-tree distance were used as predictors of time to spread in single and multiple linear regressions. These data did not require the grouping into size or distance classes, since no calculation of probability was necessary. The observed distributions of spread times were also fitted to an empirical distribution.

Literature data on amounts of actual root grafting were also summarized and compared to results of root graft transmission and patch dynamics in different areas.

G. Data Analyses — Vector Spread

Amounts of vector induced mortality were summarized for various sized blocks in Sinnissippi. The variation in these rates over time was explored by (1) using run tests and (2) identifying return times for high levels of vector mortality based on distances between objectively determined peaks. An attempt was made to correlate these peaks to weather data. Related literature information was also summarized. Further predictive relationships were sought, at various spatial scales, between the level of mortality in one year and vector spread in the succeeding years.

The spatial nature of overland spread was approached directly (1) by examining the importance of inoculum presumably originating from outside the forest and testing for differences in vector mortality between the forest edge and interior, and (2) by measuring the minimum distances for overland spread and modeling the frequency of infections at various distances. Minimum distance was measured on the map as the distance to the nearest tree that died from oak wilt in the previous year. Curl (1955) has demonstrated that over 80% of all oak wilt mats are produced at a time that results in transfer of inoculum and subsequent wilting (in the red oak group) in one year. Plotless sampling theory was used to show that the distances found were less than would be expected if spread by vector did not occur preferentially over short distances. The frequency of spreads at various distances was fitted empirically by linear regression, and this pattern was compared to analogous literature data.

Finally, the effects of cutting regimes in Sinnissippi Forest and the importance of tree size were examined. No analyses of southern Wisconsin data relating to vector spread were undertaken, since the original data did not specify method of infection.

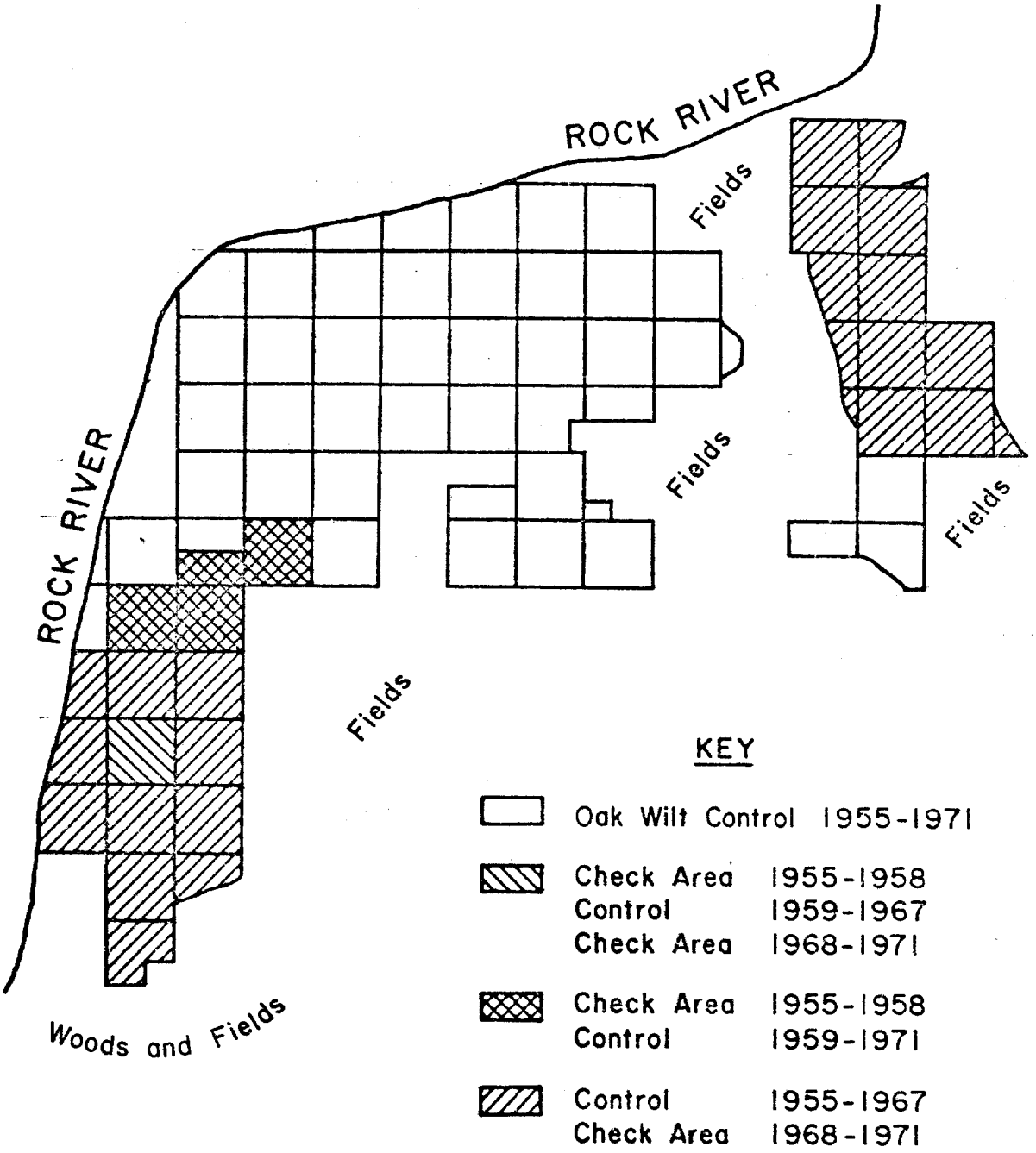
VI. RESULTS

A. Total Mortality

For an observer of an oak wilt infestation, the most readily obtained data are estimations of the amount of mortality, either in numbers of trees (in absolute numbers, relative to total stocking, or by species) or in volume. As is true of any such descriptive statistic, mortality estimates provide minimal information concerning the dynamics or mechanisms of disease spread. They are more useful when comparing data from woods with different characteristics or when making comparisons between different regions. For these reasons, and because the bulk of the literature data on oak wilt is of this nature, descriptions and analyses of total mortality are included in this section. Such data will be compared to results from model simulations.

Many results in this and other sections have been developed from data taken at Sinnissippi Forest. This forest has been heavily influenced both by cutting and oak wilt control. The effects of cutting were discussed in Section V D. The oak wilt control programs that accompanied the 17 year measurement program run by Himelick and Fox reduced total oak wilt mortality. Fig. 1 shows a map of Sinnissippi with the control regimes, by 40 acre com-

FIG. 1. Control Regimes in Sinnissippi Forest, by Compartment
(each square represents 40 acres)



partments, indicated for the period from 1955 to 1971. 'Control' refers to various oak wilt control methods, including sanitation, girdling, and poisoning of wilt-killed trees and their neighbors (detail in Himelick and Fox 1961). In many years, substantial areas were not treated in this way; these areas are referred to as check areas.

The oak wilt control treatments at Sinnissippi on a stand basis have had profound effects, the practical implications of which are not a subject of this study (Himelick and Fox 1961). However, it is important to separate the effects of control at the pocket level from the more practical questions of stand level control.

On the average, about 90% of Sinnissippi was under stand level control between 1955 and 1971, ranging between about 60% and 100%. There was less control at the individual pocket level, since much of the stand level control involved locating these pockets. Mortality had to be initiated before control could be attempted. Unless otherwise stated, control will refer to whether a tree was, at the time of its infection, in a pocket (not compartment) under oak wilt control.

The effect of control in a pocket was highly significant ($p < .01$, 1df, Chi square test) in reducing total mortality and mortality resulting from root graft spread.

Since control could not start until a pocket was initiated by long distance spread, there was no reason to test the effect of control measures on vector spread. Table 7 shows the yearly progress of oak wilt mortality at Sinnissippi, which was strongly influenced by the various oak wilt control regimes. The relatively low variation in vector transmission, even as the amount and type of oak wilt control changed, is consistent with data presented by Himelick and Fox (1961), showing that control at the stand level had little effect on the number of new infection centers initiated at Sinnissippi.

Thus control was especially important as a factor in root graft transmission, which is important in the red oak group. In particular, control will affect comparisons made between different compartments and years. Therefore, in the following analyses, comparisons between subsets of Sinnissippi are made using large areas with similar control regimes.

As was mentioned previously, one of the most notable features of oak wilt is its preferential infection and local spread in trees of the red oak group (see Table 8). Within the white oak group, bur oak often shows greater damage, relative to stocking, than white oak. There is little evidence of interspecific differences in susceptibility within the red oak group. Between stands, absolute

TABLE 7. Yearly Progress of Oak Wilt Mortality in Sinnissippi Forest by Species and Method of Infection (1955-1971)

Year	Total Dead Trees				Root Graft Trans.		Vector Trans.		Unknown	
	All Spp	R.O.G.	W.O.	Bur	Total	%	Total	%	Total	%
1955	263	256	3	4	170	64.6	19	7.2	74	28.1
1956	239	236	3	0	163	68.2	40	16.7	36	15.1
1957	143	135	4	4	67	46.9	57	39.9	19	13.3
1958	92	84	3	5	31	33.7	40	43.5	21	22.8
1959	108	84	21	3	25	23.1	62	57.4	21	19.4
1960	85	77	8	0	18	21.2	59	69.4	8	9.4
1961	52	48	3	1	7	13.5	41	78.8	4	7.7
1962	51	43	2	6	12	23.6	29	56.9	10	19.6
1963	60	56	4	0	12	20.0	44	73.3	4	6.7
1964	59	56	2	1	2	3.4	51	86.4	6	10.2
1965	60	55	5	0	14	23.3	37	61.7	9	15.0
1966	94	87	6	1	11	11.7	75	79.8	8	8.5
1967	110	100	9	1	15	13.6	89	80.9	6	5.5
1968	145	138	6	1	72	49.7	66	45.5	7	4.8
1969	135	131	1	3	51	37.8	63	46.7	21	15.6
1970	98	94	3	1	48	49.0	44	44.9	6	6.1
1971	104	101	2	1	46	44.2	49	47.1	9	8.6

TABLE 8. Literature Values for Oak Wilt Mortality, by Species

REFERENCE	AREA	MORTALITY (per acre), (% OF TREES KILLED) All Oak Spp.	Red	Black	N. Pin	White	Bur
Himelick and Fox (1961) ***	N. Illinois	0.107§	0.026§	0.068§	0.006§	0.005§	0.002§
Brinkman (1952)	Iowa	0.73	0.42(11.3)	0.29(13.5)*	--	0.02(0.4)	--
Bergdahl and French (1973)§§§	Minnesota	--	4.4(83)** 5.8(53)**	--	--	--	--
Young (1949)	Iowa	--	(53)	--	(56)	(28)	(20)
Dietz and Young (1948)	Iowa	--	--	--	(25-51)	--	--
Jones (1965)	Kentucky	3.7**	--	--	--	--	--
	NC, TN	3.1**	--	--	--	--	--
	PA, WV	8.1**	--	--	--	--	--
Jones (1971)	PA	0.33	--	--	--	--	--
	WV (NE)	0.11	--	--	--	--	--
	WV (SW)	0.12	--	--	--	--	--
Boyce (1959)	NC, TN	(24)§§	(37)§§	--	--	--	--

*includes N. Pin
**local area of infestation
***Stand composition was 52% Red Oak Group, less than 5% Bur Oak.
§per acre oak type
§§after 4 years of activity, in terms of a 50 foot circle
§§§Two stands sampled.

mortality values may vary as much as 100 fold or more (Table 8). This may reflect such factors as composition and site, but the length of time of infestation may be most important. When mortality data are taken, it is usually not known how long oak wilt has been in the area. In areas where overall mortality from oak wilt is lowest (e.g. Sinnissippi, Himelick and Fox 1961), red oak group mortality is relatively higher. This may indicate that observed differences in mortality between groups includes effects of different spread rates, rather than merely differential susceptibility.

Lack of information on species composition renders the differences between species presented in Table 8 untestable. If one assumes the 1972 Sinnissippi inventory is a reasonable approximation of the previous 17 years' composition, Chi square tests incorporating this data may be utilized, as previously outlined.

Table 9 shows the mortality in Sinnissippi by species and method of infection (root graft transmission, vector spread or unknown). Chi square tests showed that trees of the red oak group were most susceptible to all three types of mortality, followed by bur oak and then white oak ($p < .01, 2df$). These results appear sound for two reasons. First, Himelick and Fox used laboratory tests to aid in the identification of the variable symptoms seen in white

TABLE 9. Oak Wilt Mortality at Sinnissippi, by Species and Method of Infection (1955-1971).

		MORTALITY - no. of trees, (%)		
Species	% in forest	Root Graft	Vector	Total (incl. unknown)
Red Oak Group	(50.6)	849 (98.8)	783 (90.5)	1874 (94.1)
White Oak	(46.8)	5 (0.6)	64 (7.4)	85 (4.3)
Bur Oak	(2.5)	6 (0.7)	18 (2.1)	32 (1.6)
ALL	(99.9)	860 (100.1)	865 (100.0)	1991 (100.0)

oak (see Boyce 1961). Secondly, the frequency of surveys was unusually high for oak wilt studies, and this allowed better distinction between sources of the fungus (i.e. root graft vs. vector) by symptoms.

The intermediate susceptibility of bur oak is consistent with occasional observations of local oak wilt pockets in this species in the Midwest (A. Prey, personal communication, Young 1949). Drake (1956), using artificial inoculations, found that bur oaks were less susceptible to infection than trees of the red oak group.

Composition, as expressed in the timber typing done by Sinnissippi foresters, was analyzed for its effect on oak wilt mortality by Chi square tests. Table 10 shows the numbers that were tested, with degrees of freedom as previously described. As might be expected, stands dominated by trees of the red oak group showed significantly higher mortalities than mixed stands, which had higher amounts of death than stands dominated by white oak ($p < .01$, 2df). Root graft transmission differences were more significant than those caused by mortality from vector infection.

Areas typed by Sinnissippi foresters as 'Reproduction' and 'Poles' reflect dominant ages of 0-30 and 31-50 years respectively. Both total mortality and that resulting from local root graft spread showed significantly higher

TABLE 10. Oak Wilt Mortality at Sinnissippi, by Composition of Stand (1955-1971)

Stand Type	Approx. % area	MORTALITY - no. of trees, (%) Root Graft	Vector	Total (incl. unknown)
Red or Black Oak Dominated	(21)	174 (31.7)	154 (23.0)	431 (27.3)
Mixed Oak	(59)	366 (64.7)	465 (69.5)	1058 (67.0)
White Oak Dominated	(20)	26 (4.6)	50 (7.5)	92 (5.8)

values in these young stands than in other stands ($p < .01$, 1df). Young stands may have received less diligent control, but in addition there was probably serious underestimation of mortality in smaller size classes (often termed 'brush' by original investigators) since data without individual tree dbh's were not included in these analyses, and since smaller trees were more likely to have been missed. This would have depressed mortality counts. Overland spread was not significantly different in these younger stands, as compared to that of the older stands.

Site productivity also seemed to affect oak wilt mortality. Sinnissippi foresters estimated productivity as poor, medium, or good, based upon estimates of height growth of 50, 60, and 70 feet respectively in 50 years (Schnur 1937, Jokela and Lorenz 1957). Over all compositions, sites typed as poor showed significantly higher mortalities, both root graft and vector, than medium or good sites (Chi square $p < .01$, 2df). This was true when comparing sites of mixed composition (Poor vs. Medium vs. Good). However, poor sites dominated by red oak group trees showed less of a tendency (vs. Red Oak Good, $p < .05$, 1df) for higher root graft spread, and no significant differences in vector or total mortality. Sites dominated by white oak did not show any significant differences in mortality attributable to site productivity. Thus, the

differences over all compositions may partially reflect a greater abundance of red oak group trees on poorer sites.

This may be a factor in literature reports of differential oak wilt mortality. Anderson and Skilling (1955) showed that there was more mortality in scrub oak types, and Cones and True (1967) found more oak wilt on less productive sites, consistent with the results of this study.

In Sinnissippi, since apparent differences due to productivity were greatest for root graft transmission, it is possible that differences in overland spread reflected local differences in inoculum caused by contrasting root graft transmission mortality and were not due directly to the effects of site productivity itself.

The influence of soil type on oak wilt also was examined by Chi square tests; the data used are in Table 11. Trees on sands had more mortality of all types than those on sandy loams or silt loams and loams. Sandy loams and loams showed a similar level of mortality. In these soil comparisons, the expected values were made proportional to the area occupied by these soils rather than depending on the timber inventory, since the cutting regimes were dissimilar. Control regimes also differed between soil types: loams were more often check areas, perhaps inflating root graft mortality on these soils relative to others. Results

TABLE 11. Oak Wilt Mortality at Sinnissippi, by Soil Type (1955-1971)

Soil Type	Approx. % area	MORTALITY - no. of trees (%) Root Graft	Vector	Total (incl. unknown)
Sands	(26)	310 (37.8)	375 (32.5)	685 (34.7)
Sandy Loams	(55)	349 (42.6)	600 (52.1)	949 (48.1)
Silt Loams	(19)	161 (19.6)	177 (15.4)	338 (17.1)

agree with True and Gillespie (1961, Gillespie and True 1959), who found that shallow soils favored local spread of the disease in West Virginia, with Craighead and Nelson (1960), who found more mortality on sandstones, and with observations (J. E. Kuntz, personal communication) that sandy areas in Wisconsin show most extensive damage. Anderson and Anderson (1959) also found a slower rate of patch expansion on heavier soils in Central Wisconsin.

In conclusion, descriptive statistics from Sinnissippi Forest show that susceptibilities to both root graft and vector spread of oak wilt decrease from red oak to bur oak to white oak. Stands dominated by red oak show a higher level of damage than stands dominated by white oak. Young stands may be somewhat more susceptible than mature ones, as are poorer sites, especially those on sandier soils.

Since composition, site productivity, and soil type are not independent of each other, data on total mortality can only hint at the mechanisms underlying the observed differences. Subsequent data analysis and model simulations will help explain some of these patterns.

B. Results — Patch Dynamics

Perhaps the outstanding result of an oak wilt infestation is the concentration of mortality in contiguous

areas. These areas are generally called 'foci' by workers dealing with forest diseases and often called'pockets' by oak wilt workers. More generally, oak wilt pockets may be termed 'patches:' all three terms will be used synonymously.

Patch dynamics are very much a function of vector spread, since this initiates the pocket. However, composition and the previous amount of oak wilt will affect rates of pocket initiation. Expansion of the pocket proceeds mainly by root graft transmission, but short distance vector transmission also plays a role. Cessation of expansion will be affected by the factors influencing pocket expansion. Thus, descriptions of oak wilt patch dynamics reflect many factors, as did mortality statistics. Again, such data are more common than more mechanistic data and are useful for comparative purposes. The data presented here are phenomenological (vs. fundamental) since patterns are described without reference to causality (Wilson 1969).

Description of the demography of patches requires data on pocket birth, growth, and mortality; the last abstracted for the purposes of this study as a cessation of expansion. In Sinnissippi, oak wilt control could not start in a given pocket until its initiation. Oak wilt controls were aimed partly at reducing mat production, but did not have a large effect on the number of new

patches or the levels of vector spread (Himelick and Fox 1961).

At Sinnissippi an oak wilt pocket was defined as including all wilting trees within 50 feet of previous wilt. In practice, the boundary was probably less for small trees. If a pocket had been inactive for some time, it is probable that new infections into the area would have been categorized as initiating new pockets. It is an open question as to how long 'some time' was.

Birth rates, or the number of new patches started, were summarized annually by compartment for Sinnissippi. Most compartments showed considerable variation from year to year. Representative values for areas of various sizes are shown in Table 12. The forest-wide annual value for new pocket initiation varied between 20 and 69 between 1955 and 1971 (0.48 - 1.67/40 acres) with a mean of 41.9 and a standard deviation of 12.6. Check areas (at a stand level) showed slightly higher values than areas of oak wilt control.

Typical literature values for new pocket initiation are presented in Table 13. Survey data taken over large areas and many years (e.g. Jones 1965) show considerably lower values than data reflecting outstanding damage in peak years (e.g. Wysong 1949). These differences make comparisons of geographic regions approximate, but values

TABLE 12. Numbers of New Oak Wilt Pockets in Blocks of Various Sizes, by Year, Sinnissippi Forest (1956-1971)

		NO. OF NEW PATCHES IN 19--																	
Block	Size (acres)*	56	57	58	59	60	61	62	63	64	65	66	67	68	69	70	71	X**	S**
A11	1648	29	(34)§	29	41	(48)	30	20	38	(49)	37	60	(69)	54	48	40	44	1.02	0.31
126	154	5	5	3	(9	9)	3	1	6	(12)	8	(17)	14	6	8	7	8	1.96	1.08
35	56	2	0	(4)	2	1	0	(3	3)	2	2	3	(4)	1	1	1	3	1.43	0.90
100	129	1	1	(3)	1	1	1	2	2	(4)	1	0	1	(8)	5	(9)	3	0.83	0.82
A 3	303	5	4	(7)	6	5	1	5	6	(7)	5	5	8	10	10	(12)	7	0.85	0.35
81	157	2	2	0	2	(9)	0	0	3	2	2	1	(7)	3	2	(4)	0	0.62	0.64
85	146	(4)	2	4	6	(8)	3	1	3	2	1	4	5	(7)	1	0	3	0.92	0.62
115	147	3	2	1	(4)	3	2	2	1	2	(4)	2	2	(4)	3	1	3	0.66	0.28
A21	384	(10)	5	7	7	7	(11)	4	6	11	11	11	15	(16)	14	7	11	1.00	0.37
A22	301	5	7	9	(13)	8	7	5	11	(14)	9	(24)	17	12	11	11	12	1.45	0.63
119§§	<40	2	0	(4)	0	0	(3)	0	0	0	1	2	1	0	(5)	1	1	1.25	1.57
92§§	<40	0	1	1	1	(6)	0	0	1	1	1	1	0	1	0	0	0	0.88	1.45

*approximate acreage of oak types

**per 40 acres

§circles designate peaks; see text

§§assume compartments have 40 acres oak type.

TABLE 13. Annual New Pocket Formation in Various Areas

REFERENCE	LOCATION	YEAR(s)	AMOUNTS*	COMMENTS
Himelick and Fox (1961)	N. Illinois	1955-1960	1.5-3.0	Value for non-control. Oak wilt control in surrounding area.
Anderson and Anderson (1957) (1958) (1963)	Wisconsin	1955-1959	0.3-1.2	Strip quadrats near roads
	Minnesota	1955-1959	0.35-0.71	Strip quadrats near roads
Wysong (1949)	Iowa	1949	1.20-3.96	Beginning of oak wilt in two local areas
Boyce (1959)	S. Appalachian	1958	14.5/100 mi^2	
Boyce (1957b)	Kentucky, Tennessee, North Carolina	1954-1956	26.0/yr.	Entire state
USDA (1957)	Tennessee, North Carolina	six years	41.7/yr	Entire state
Jones (1965)	Kentucky	1958-1963	24/100 mi^2	Forested land only
	Tennessee	1958-1963	23/100 mi^2	Forested land only
	Kentucky	1957-1963	2.55	Areas of high local infection
	North Carolina, Tennessee	1957-1963	3.05	Areas of high local infection
	Pennsylvania	1957-1963	6.25	Areas of high local infection
	West Virginia	1957-1963	6.25	Areas of high local infection
Jones and Bretz (1958)	Missouri	1952-1956	3-5	Areas of high local infection

*per 40 acres unless otherwise noted

from the Midwest are generally considerably higher than those in the Appalachians. The year to year variation in one area is usually small, as in Sinnissippi. One might expect this to be the case in situations where the disease has been present for some time, and the more variable estimates of Wysong (1949) might well be typical of damage resulting from recent introduction of the fungus. New pocket initiation over time in subdivisions of Sinnissippi (Table 12) showed a higher variance relative to the mean, and hence indicate that the temporal consistency in pocket formation at the forest-wide spatial scale belies considerable small scale variability.

What is the spatial relationship of patches initiated in a given year to previously formed patches? For each year at Sinnissippi, areas of between 80 and 400 contiguous acres with greater than 1.5 new pockets per 40 acres were tallied as areas of high patch birth. The number of new patches in these areas in the following year decreased by an average of 0.895/40 acres, and the number of new pockets in the subsequent year in neighboring compartments increased in 23 of 30 instances, by an average of 0.537 pockets/40 acres. Both values are significantly different from zero (t tests, $p < .01$).

Thus, the data indicate that relatively high amounts of new pocket formation 'move across' Sinnissippi at the

scale of tens and hundreds of acres annually. The picture emerging is one of dynamic change in the spatial patterning of pocket birth. However, one should realize that new pockets do not beget new pockets, but that previously wilting trees provide inoculum for infection of healthy oaks. This will be looked at more closely when considering vector spread itself.

Attempts to further examine patterns of pocket birth over time were made using run tests; nonparametric tests that can detect patterns in a time-ordered series of values. The data could not have appropriately been examined by time series analysis. Run tests for values above and below the median (Sokal and Rohlf 1969) were used on patch initiation values for various sized areas over time (Table 12). These data did not show significant monotonic trends by this test. Run tests on the same data using the sign of the differences (the 'up and down test,' Sokal and Rohlf 1969) also turned up no significant patterns. The latter test is used to detect periodicities in the run. The results from the run tests on patch birth over time indicate that if there is a regular pattern or periodicity in the data, it is more episodic than smoothly changing.

As an alternative approach, the three highest peaks in new pocket initiation in an area between 1956 and 1970 were chosen objectively on the basis of absolute magnitude

and difference from neighboring years. Peaks so chosen are circled in Table 12. Distances between peaks were determined.

The highest proportion of interpeak distances (38%) were in the six to seven year range, although all lengths were represented. Six and seven year lengths were most evident in blocks of 120 to 400 acres; results at smaller and larger block sizes were more variable. Over the period of data collection in Sinnissippi, this evidently was the return time for waves of pocket initiation. The spatial scale at which the pattern emerges is the same as that used in finding the 'movement' of areas of high patch birth. In Section VI D this analysis will be used on levels of vector spread.

The other two components of patch dynamics, patch growth and patch mortality, are more difficult to specify from Sinnissippi data. Oak wilt control measures not only affected these components in much of the forest, but few check areas were left untreated for more than five years. Table 14 shows some data on growth of uncontrolled patches that is fairly complete for the first few years of expansion. It presents the number of dead trees in a pocket at each annual time increment after initiation. This data does not consider pocket mortality, which was defined as cessation of expansion. The table is probably biased in

TABLE 14. Size of Pockets vs. Number of Years Active, Sinnissippi Forest (1952-1971)*

Number of Years Active	NUMBER OF POCKETS WITH ___ TREES								Average No. Trees	Average Per Year
	1	2	3	4	5	6-10	11-15	16-20		
1	684	106	27	17	6	5	0	0	1.32	1.32
2	91	53	22	18	4	7	0	1	2.19	1.10
3	38	48	18	15	6	8	1	0	2.65	0.88
4	16	28	9	13	6	6	2	1	3.37	0.84
5	2	15	6	7	7	6	1	0	3.96	0.79
6	0	2	3	1	1	3	0	0	4.60	0.77
7	0	0	0	0	0	0	1	0	--	--
8	0	0	0	0	0	0	1	0	--	--

*Data from pockets that were not subject to oak wilt control. This table includes data from several stages in the growth of single pockets. Thus, the numbers do not reflect the size distributions of pockets of their maximum sizes.

favor of small pockets, since these may more easily have escaped detection and control for a few years. Also, several large pockets were omitted because of incomplete data. The values indicate that most pockets began as one tree and that about half increased in size in one year. Subsequent growth was somewhat variable but appeared to approach a value of 0.8 additional trees killed per year for pockets that remained active for four or more years. The relatively constant expansion of active pockets is not intuitively expected, since older and larger pockets would presumably border increasing numbers of susceptible oaks. These results do agree with data from West Virginia (Table 15, from Boyce 1957b) which indicate that the number of new infections per year is fairly constant in centers that have been active for two to ten years. The West Virginia pockets expanded at over twice the rate of the Sinnissippi pockets studied.

Table 16 summarizes similar data from Sinnissippi expressed as transition probabilities for pockets of various sizes. Since there was not any data on pocket mortality, the matrix was not solvable for a stable pocket size distribution.

The rates of pocket expansion outlined above are in terms of numbers of trees/year and will be affected by such factors as composition and density of the stand. Many of

TABLE 15. Numbers of Infected Trees per Center and Numbers of Infected Trees per Center per Year, for Centers of Various Ages, North Carolina and Tennessee (Boyce 1957b)

AGE OF CENTER (years)	AVERAGE NO. INFECTED OAKS	AVERAGE NO. PER YEAR
10	21.2	2.1
8	6.5	0.8
7	15.0	2.1
6	10.0	1.7
5	9.8	2.0
4	7.7	1.9
3	5.9	2.0
2	3.6	1.8

TABLE 16. Transition Probabilities for Changes in the Number of Trees per Patch in a Single Year.*

PREVIOUS SIZE (no. trees)	RESULTING SIZE (no. trees)					
	1	2	3	4-10	11-29	30+
0	.84	.078	.059	.020	0	0
1	.40	.46	.10	.067	0	0
2	---	.33	.40	.27	0	0
3	---	---	0	1.00	0	0
4-10	---	---	---	.67	.33	0
11-29	---	---	---	---	.67	.33
30+	---	---	---	---	---	1.00

*Derived from Sinnissippi non-control data. Patch mortality (i.e., the cessation of patch growth) is not considered.

the estimates of pocket expansion rates have been presented in terms of radial expansion (increase in pocket diameter/year) or in percent of trees in an area infected/year. For completeness, some of those values are included in Table 17. In West Virginia, pocket expansion proceeded more rapidly on shallow soils (Gillespie and True 1959) and, in Wisconsin, heavier soils resulted in slower patch growth (Anderson and Anderson 1959). Expansion rates outside the Midwest are lower and more variable, perhaps reflecting differences in root grafting (Section VI C) or composition.

When pocket growth ceases, we can say that mortality of the patch has occurred. Since oak wilt pockets may show intermittent activity, this component of oak wilt patch demography is difficult to measure. Boyce (1959) found that 46% of centers observed were inactive for three years. None of these resumed activity in the following two years. Himelick and Fox (1961) found that 33% of all centers did not expand beyond one tree. Some of the other relevant literature data concerns the percentage of observable pockets that are active, and this is included in Table 17. Activity in a pocket is defined as mortality in a given year. In both Sinnissippi and southern Wisconsin, smaller pockets were observed to have a higher probability of ceasing expansion than large pockets, agreeing with data

TABLE 17. Literature Values for Rates of Pocket Expansion (Growth) and Growth Cessation (Pocket Mortality)

REFERENCE	LOCALE	ANNUAL POCKET EXPANSION	PERCENT ACTIVE POCKETS	COMMENTS
Anderson and Anderson (1957)	Central Wisconsin	3.5,* 6.5ft**	25-68%	
Anderson and Anderson (1958)	Central Wisconsin	5.7-11.4* ft	---	
Anderson and Anderson (1963)	Central Wisconsin and Minnesota	3.2 ft*	42%	
French and Bergdahl (1973)	Minnesota	25 ft	---	Single Pocket
Schreiber and Green (1958)	Indiana	6% available oaks	---	Within 50 foot circle. 140-310 oaks per acre
Riker (1951)	Wisconsin	1-2 Tree depths	---	Observation
Boyce (1957b)	Tennessee, North Carolina	0.8-2.1 trees	---	See Table 15
USDA (1957)	Tennessee, North Carolina	----	25%	No. of pockets remaining active for five years

*all pockets

**active pockets

presented by Himelick and Fox (1961).

One of the most notable and observable features of oak wilt is average patch size. Table 18 shows some of the values for areas in which this has been measured. Pocket sizes are larger in the Midwest than elsewhere, possibly due to higher oak density and/or the importance of root grafting. However, this size is less often measured, probably reflecting the tendency of large foci to merge (e.g. Young 1949, French and Bergdahl 1973).

In summary, pocket birth rates showed greater temporal variation in smaller areas and a distinct spatial pattern over the forest over time. In Sinnissippi, this resulted in a typical return time (six to seven years) for high levels of patch initiation. Pocket growth and pocket mortality (cessation of growth) were much affected by oak wilt control at Sinnissippi. Annual oak wilt mortality in a pocket appeared to approach a constant value after several years, despite a presumed increase in the number of root graft spread possibilities. Areal patch expansion rates and average focus size varied with site, being generally higher in the Midwest than in the Appalachians.

Characteristics of initiation, growth, cessation of expansion, activity, and size of oak wilt patches are merely descriptive. In the following sections, the two

TABLE 18. Specific Literature Values for Average Pocket Size

REFERENCE	LOCALE	NO. INFECTED AND DEAD TREES PER CENTER	COMMENTS
French and Bergdahl (1973)	Minnesota	64-168	Largest centers
Jones and Bretz (1958)	Missouri	2.31	Varied 3-fold
Staley and True (1952)	West Virginia	1.2	
Gillespie and True (1955)	West Virginia	1.05 - 1.67+	
Gillespie and True (1959)	West Virginia	2-5	
True and Gillespie (1961)	West Virginia	1.4	Actively wilting trees only
Boyce (1957b)	West Virginia	3.3	
USDA (1957)	Tennessee, North Carolina	2	
Astin and Quillan (1965)	Tennessee	~ 1.1	18 centers only
Skelly and Wood (1974)	West Virginia	2.56 - 2.76 1.96 - 2.76	All trees Actively wilting trees only

types of spread that create these patterns (root graft transmission and vector transmission) will be analyzed. The extent to which observed patterns of oak wilt mortality can be re-created based on quantification of the two different mechanisms of disease transmission will help explain their importance in various situations. To accomplish this re-creation, more fundamental data will be analyzed, and model simulations based on the results will be presented. The patch dynamics data presented in this section will serve as a baseline for comparison and further discussion.

C. Results — Root Graft Transmission

Transfer of Ceratocystis fagacearum conidiospores in the transpiration stream through intraspecific root grafts results in pockets of oak wilt mortality. Vector spread over short distances may also contribute to patch expansion. In this section, quantification of root graft spread is attempted. There are two general goals:

1) prediction of probability of root graft transmission between two trees.

2) prediction of the time between successive deaths, when the second is caused by root graft spread from the first.

In addition, the dynamics of root graft transmission will be related to different times for spread, site conditions, oak wilt control, and geographic differences in root

grafting between oaks.

The models that are proposed in this paper as predictors of root graft transmission consider the combined diameter of the two trees involved in inoculum transfer. The possibility exists, however, that individual tree diameters or, more specifically, the magnitude of diameter differences, may affect probabilities of transmission. Some possibilities:

1) If smaller trees have a seed origin, less root grafting of these to the larger trees in the stand might be expected compared to the expectation based on their combined dbh.

2) If sprouting from living oaks has been favored, more root grafting than expected between different sized trees might occur.

3) Sprouting from rootstocks after cutting or fire would result in high amounts of root grafting between similarly sized trees.

These are factors relating to diameter differences that affect root grafting. There may be others that affect the movements of spores through grafts. Table 19 shows results from the southern Wisconsin sites that bear on this problem. The percentage of root graft transmission in red oak group trees is summarized by combined dbh class and diameter difference classes, for class combinations with at least five possibilities for spread. The sparsity of data makes appropriate nonparametric tests (e.g. sign tests) ineffective in detecting differences in transmission between diameter difference classes. However, it is ob-

TABLE 19. Root Graft Transmission of Oak Wilt in Southern Wisconsin: Effects of Diameter Differences

SITE	COMBINED DBH CLASS	% TRANSMISSION BETWEEN TREES WITH DIAMETER DIFFERENCE OF (in inches)* 0-3	4-7	8-11	12-15
West Hill (WHA)	11-15	10.8	11.1	X**	X
	16-20	24.6	19.1	X	X
	21-25	29.1	40.0	X	X
	All Sizes	24.7	26.2	X	X
Miller's Farm (MF)	11-15	44.4	28.6	X	X
	16-20	47.8	50.0	X	X
	21-25	62.5	X	X	60.0
	26-30	50.0	40.0	X	X
	All Sizes	44.9	43.3	33.3	50.0
Devil's Lake (DL)	11-15	40.0	33.3	X	X
	16-20	15.8	0.0	X	X
	21-25	13.8	4.2	23.1	X
	26-30	15.6	4.8	0.0	X
	31-35	0.0	42.9	X	9.1
	36-40	0.0	80.0	X	X
	All Sizes	13.4	13.9	16.7	20.0
Arboretum (ARB)	16-20	19.2	28.6	X	X
	21-25	25.6	10.0	X	X
	26-30	23.3	25.0	11.1	X
	31-35	27.6	44.4	11.1	20.0
	36-40	27.8	16.7	14.3	X
	All Sizes	24.4	23.8	21.4	10.0

*Only instances with at least five possibilities of transmission are included

**Not enough data

vious from inspection of Table 19 that diameter differences had no effects on percent transmission at any of the four southern Wisconsin sites.

Therefore, the combined diameter approach appears justified and will be used. Results will generally deal with transmission between members of the red oak group. Discussion of other species of oak may be found in the latter part of this section.

1. Probability of Transmission between Red Oak Group Trees — Southern Wisconsin.

The results of several hundred linear regressions on southern Wisconsin data are summarized in Tables 20 and 21; they relate the eight models of combined dbh and distance to probability of root graft transmission between two trees of the red oak group. Table 20 shows correlations from 'time-specific' results, i.e. predictions of root graft transmission in a given amount of time. Data from one site (Miller's Farm) yielded no significant fits to any of the models, probably due to a small data base. The transmission probabilities were unusually high at this site. Most sites and years did show highly significant r values for several of the models, however. In general, it was more difficult to model spread taking longer times, partly due to the smaller amount of data used.

TABLE 20. Correlation Coefficients and Significance Levels from Regressions of Probability of Time Specific Root Graft Transmission on Models of Size and Inter-Tree Distance, Southern Wisconsin

SITE	NO. YEARS	MODEL*** DBH/D	DBH/D^2	DBH-D	DBH^2/D	DBH	1/D	0(1:1)	0(2:1)
ARB	0	.690**	.615**	.670**	.621**	.335	.547**	.640**	.477*
	1	.445*	.452*	.425*	.353	.187	.488*	.296	.274
	2	.634**	.628**	.543*	.595**	.196	.535*	.481*	.610**
	3	.307	.193	.351	-.135	.246	.195	.355	.332
DL	0	.473	.563*	.469	.563*	.553*	.563*	.549*	.549*
	1	.506	.494	.528*	.498	.304	.483	.473	.518*
	2	.222	.219	.287	.046	-.177	.239	.164	.002
MF	0	.441	.465	.451	.467	.127	.423	.409	.364
WHA	0	.768**	.678*	.819**	.721**	.532	.619*	.705**	.651*
	1	.588	.605*	.625*	.437	.129	.686*	.399	.395
	2	.809**	.699*	.906**	.822**	.735*	.687*	.897**	.868**
	3	.818**	.735**	.884**	.816**	.541	.742**	.868**	.821**
ALL SITES	0	.650**	.598**	.601**	.518*	.111	.681**	.313	.268
	1	.671**	.546**	.662**	.561*	.170	.607**	.422*	.359
	2	.528*	.662**	.428	.336	-.256	.720**	.214	.109
	3	.244	.213	.280	.150	.011	.327	.127	.039

*significant at .05 level
**significant at .01 level
***Best fit for a site and year is circled.
Second best fit is underlined.

TABLE 21. Correlation Coefficients and Significance Levels from Regressions of Probability of Time Independent Root Graft Transmission on Models of Size and Inter-Tree Distance, Southern Wisconsin.

	MODEL***							
SITE	DBH/D	DBH/D^2	DBH-D	DBH^2/D	DBH	1/D	0(1:1)	0(2:1)
ARB	.797**	.725**	.763**	.704**	.229	.752**	.567**	.505*
DL	.456	.589	.424	.356	-.105	.569	.188	.178
MF	.531	.490	.701	.572	-.097	.613	.457	.742*
WHA	.758**	.689**	.786**	.677*	.500	.623*	.621*	.607*
ALL SITES	.737**	.620**	.715**	.599**	.059	.723**	.408	.349

*significant at .05 level
**significant at .01 level
***Best fit for a site is circled.
Second best fit is underlined.

The models 'DBH/D' and 'DBH-D' were the best predictors overall for time specific probabilities. Both yielded similar predictions over the range of the data. The model emphasizing distance by a power of 2 ('DBH/D^2') also fitted well in many situations. In both the Arboretum and West Hill, 'DBH/D^2' generally showed improved fits when the time for spread was longer. The good performance of '1/D,' which does not consider tree size, in predicting probabilities of data pooled from all four sites may be attributed partly to the averaging of size differences among sites. The 'DBH^2/D' model was somewhat more erratic than the models emphasizing distance in fitting time specific data from southern Wisconsin.

Combined dbh alone ('DBH') was a poor predictor of root graft transmission probability. The overlap model ('O'), used here assuming both equal diameters and diameters in a ratio of 2:1, also showed variable results. Individual diameters were not used for overlap models, since the division of the data into individual diameter classes would have based probability calculations on extremely small amounts of data.

Time independent probabilities are not tied to any one duration of time; they consider root graft spread in any number of years. Table 21 shows correlation coefficients for these data, with data from the two Sauk County

sites (Miller's Farm and Devil's Lake) failing to fit most of the models. Data from the other two sites were fitted well by all models except 'DBH.' Best fits on the West Hill data were accomplished by 'DBH-D' and 'DBH/D' in that order, while Arboretum sites were fitted best by the same models in reverse order. Lumped data from all sites were fitted best by 'DBH/D.' '1/D' showed the next best prediction, which can again be explained by the dilution of the effects of diameter by combining sites with different size class distributions for the red oak group.

For all regressions with significant correlation coefficients, five percent confidence limits on both slope and intercept were calculated. Table 22 compares regressions fitting spread over different elapsed times (different intervals) at the same site. Capital letters show occasions when the five percent interval for a parameter estimate of one interval did not overlap the parameter estimate of an interval for a different elapsed time. When a five percent limit fails to overlap another parameter estimate, one can say with 95% certainty that the parameters are different. These tests were made for both slope and intercept and were made for all possible comparisons. The model that provided the best fit for the site was used; this can be expected to be most sensitive in detecting differences. Only years and sites that had data fitted signi-

TABLE 22. Comparisons of Parameter Estimates: Within--Site Comparisons--Different Times of Spread, Southern Wisconsin

LEGEND

M = regression fits significantly different in slope (.05 level)
m = regression fits not significantly different in slope (.05 level)
B = regression fits significantly different in intercept (.05 level)
b = regression fits not significantly different in intercept (.05 level)

ALL SITES (DBH/D) — Point Estimate — 5% Confidence Interval

no. years	0	1	2
0	--	mb	mb
1	mb	--	mb
2	mb	mb	--

WHA (DBH-D) — Point Estimate — 5% Confidence Interval

no. years	0	1	2	3
0	--	mb	mb	mB
1	mb	--	mB	mB
2	mb	mb	--	mB
3	mB	mB	mB	--

Intercepts: 2 > 1 > 3

ARB (DBH/D) — Point Estimate — 5% Confidence Interval

no. years	0	1	2
0	--	mb	mb
1	mb	--	mB
2	mb	mB	--

Intercepts: 1 > 2

ficantly ($p < .05$) by the same model were used.

Each site was fitted (in general) best by a particular model that was the same for all time specific regressions, and these models differed between sites (e.g. ARB vs. WHA, see Table 19). When comparing parameter estimates provided by the same model for different duration spreads at the same site, there were no cases when the slopes were significantly different. The similarity in models and slopes suggests that the relationships of tree size and inter-tree distance to probability of root graft transmission do not change with the time elapsed for transmission.

There were several significant differences in Y intercepts ($p < .05$, see Table 22) at the same site when comparing intervals of two and three years to shorter spreads. The conclusion that one could draw is that, although the form of the probability of spread dependence on size and distance (i.e. the slope) did not change with different elapsed times for spread, the absolute levels of spread (i.e. the intercept) may have been quite different. In two of three cases, the spread taking longer had a lower probability and a more negative intercept. But, since the slopes were not significantly different, this decrease did not seem to act preferentially with respect to tree size or distance. Stated another way, the probability of spread

usually decreased with time, but it scaled similarly with tree size and distance over all durations.

When the two southern Wisconsin sites that were fitted well in the time independent analysis were compared to each other, the situation was quite different. Table 23 shows the results of significance tests at the five percent level for detecting differences in regression parameters between sites for time independent data. Overall, many significant differences in both slopes and intercepts were evident. Apparently, the two sites had different size and distance relationships to local spread. Furthermore, the two models shown gave different results. They differed as predictors for individual sites; the 'DBH/D' model predicted very well for the Arboretum site, but was less precise in fitting West Hill data. These fundamental inter-site differences contrast to the similarities of different duration spreads at the same site.

Using both models, one finds that West Hill had the significantly higher slope, i.e. the highest difference in amount of root graft transmission between trees with favorable combinations of combined dbh and distance, relative to smaller trees farther apart. West Hill has drier soils than the Arboretum, the other Dane Co. site. Note that the combined data for all sites had a lower slope relative to the Y intercept than either of the individual sites.

TABLE 23. Comparisons of Parameter Estimates: Between-Site Comparisons for Time Independent Spread Using Two Different Models, Southern Wisconsin.

LEGEND--See Table 22

MODEL: DBH-D

5% Confidence Interval

	SITE	WHA	ARB	ALL SITES
Point Estimate	WHA	--	Mb	MB
	ARB	mb	--	MB
	ALL SITES	Mb	MB	--

Slopes:

WHA > ARB > ALL

Intercepts:

ALL > WHA
ALL > ARB

MODEL: DBH-D

5% Confidence Interval

	SITE	WHA	ARB	ALL SITES
Point Estimate	WHA	--	MB	Mb
	ARB	mB	--	mB
	ALL SITES	mb	mb	--

Slopes:

WHA > ARB
WHA > ALL

Intercepts:

WHA > ARB
ALL > ARB

This was due to two factors. First, Devil's Lake data had lower levels of transmission at short distances and large dbh's and were included in the lumped data. Secondly, lumping of data resulted in best fits with more conservative parameter estimates: i.e. a lower slope and a higher intercept, reflecting inclusion of diverse data.

Comparisons of time specific spread in the sites fitted by the models is shown for spread in zero, one and two years in Table 24. Spread taking place within the same year as the initial death showed inter-site differences; there were significant differences in both slope and intercept. Again, lumped data had significantly higher intercepts. There were no inter-site differences in parameter estimates, with one exception, for spread in one or two years. Model fits for spread taking one year are loose, and the lack of significant fits may be a result of wide confidence belts. However, the model fits for two year spread were close, so one must conclude that the sites did not differ for spread of this duration. If all this represents a real phenomenon, it may indicate that site differences in diameter and distance effects on root graft transmission are most significant in transfers that occur rapidly.

TABLE 24. Comparisons of Parameter Estimates: Between-Site Comparisons for Time-Specific Spread, Southern Wisconsin

LEGEND--See Table 22

5% Confidence Interval

SAME YEAR (DBH/D)

Point Estimate

SITE	WHA	ARB	ALL
WHA	--	Mb	MB
ARB	mb	--	MB
ALL	MB	mB	--

Slopes:

WHA > ARB > ALL

Intercepts:

ALL > WHA
ALL > ARB

5% Confidence Interval

ONE YEAR (DBH-D)

Point Estimate

SITE	WHA	ARB	DL	ALL
WHA	--	mb	mb	mb
ARB	mb	--	mb	mb
DL	mb	mb	--	mb
ALL	mb	mb	mb	--

5% Confidence Interval

TWO YEARS (DBH/D)

Point Estimate

SITE	WHA	ARB	ALL
WHA	--	mb	mb
ARB	mb	--	mB
ALL	mb	mb	--

Intercepts:

ALL > ARB

2. Probability of Transmission between Red Oak Group Trees — Sinnissippi

The above information applies to root graft spread in the red oak group in southern Wisconsin. Although no direct probability data could be derived from Sinnissippi data, due to a lack of complete mapping, the rather involved methods outlined in Section VF2 led to the formulation of an index ($t_{i,j}$) that summarized the relative probability of transmission for a given size and diameter class. The index related the number of observed spreads to the expected size/density characteristics of oaks within various distances, based on the 1972 inventory. The resulting indices were dependent variables in regressions, while the same eight models of 'CDBH' and 'D' (see Section VF1) were predictors. Because the indices were relative, they were used for comparisons but not in generating predictions.

Table 25 shows the performance of the models predicting time independent root graft transmission probability, in terms of correlation coefficients and significance from zero, for regressions applied as outlined above. The whole of Sinnissippi Forest was considered, as was spread in previously controlled pockets and in uncontrolled pockets.

For all models except 'DBH' in controlled areas, the r values were highly significant, indicating a very good

TABLE 25. Correlation Coefficients and Significance Levels from Regressions of Probability of Time Independent Root Graft Transmission on Models of Size and Inter-Tree Distance, Sinnissippi Forest

MODEL***	WHOLE FOREST	CONTROLLED POCKETS	NON-CONTROLLED POCKETS
DBH/D	.894**	.724**	.868**
DBH/D^2	.818**	.687**	.781**
DBH-D	.777**	.584**	.755**
DBH^2/D	.881**	.647**	.846**
DBH	.427**	.242	.437**
1/D	.650**	.591**	.596**
O(1:1)	.761**	.530**	.767**
O(2:1)	.697**	.440**	.699**

*significant at .05 level
**significant at .01 level
***Best fit for a site is circled.
Second best fit is underlined.

fit. If one were choosing a best model for prediction at Sinnissippi, it would be 'DBH/D,' also one of the best for southern Wisconsin data. The next best is 'DBH^2/D,' emphasizing tree size, perhaps reflecting the higher range of tree sizes found in Sinnissippi data relative to southern Wisconsin data. Still, 'DBH' alone was a poor predictor. The model 'DBH-D' did not provide as close a fit to Sinnissippi data as it did to southern Wisconsin data.

The Sinnissippi 'probabilities' were, as described in the Methods section, derived from expected distributions of neighbors compared to number of transmissions. When the data for control and check pockets are relativized to reflect the estimated average amount of area in each condition, one can compare regression coefficients. Table 26 shows the results of such comparisons, which were nearly identical using two different models. Check areas had higher slopes ($x^{4}5$) than areas under oak wilt control. In the former areas, small changes in diameter or distance resulted in significant changes in probability of transmission. Oak wilt control obscured this relation, and the higher intercept reflects the constancy of breakovers with respect to distance.

Time specific data (i.e. spread in a particular number of years) were also fitted to the eight models; results for the whole forest are in Table 27. The overlap model

TABLE 26. Comparisons of Parameter Estimates: Effect of Control on Time Independent Spread, Sinnissippi Forest

LEGEND--see Table 22

MODELS:
DBH/D
DBH^2/D
(same results)

		5% Confidence Interval		
	Treatment	ALL	CONTROL	NON-CONTROL
Point Estimate	ALL	--	MB	MB
	CONTROL	MB	--	MB
	NON-CONTROL	MB	MB	--

Slopes: NC > ALL > CONTROL

Intercepts: CONTROL > ALL > NC

TABLE 27. Correlation Coefficients and Significance Levels from Regressions of Time Specific Root Graft Transmission on Models of Size and Inter-Tree Distance, Sinnissippi Forest (whole forest).

	YEARS TO SPREAD				
MODEL***	0	1	2	3	4+
DBH/D	.510**	.649**	.648**	.737**	(.597)**
DBH/D^2	.402**	.586**	.505**	(.782)**	.587**
DBH-D	.490**	.543**	.664**	.496**	.462**
DBH^2/D	.547**	(.704)**	.672**	.630**	.540**
DBH	.312*	.353*	.432**	.184	.270
1/D	.314*	.401**	.054	.628**	.462**
0(1:1)	(.687)**	.613**	(.697)**	.428**	.433**
0(2:1)	.495**	.561**	.646**	.338*	.361*

*significant at .05 level
**significant at .01 level
***Best fit for year is circled.
Second best fit is underlined.

with individual diameters equal was the best predictor of short duration spread, but was more inconsistent with longer times. Perhaps the most consistent predictors were 'DBH/D,' 'DBH/D^2,' and 'DBH^2/D.' It is notable that both 'DBH/D^2' and '1/D' performed best when spread took three years or longer. This adds more indirect evidence that distance factors are more important in slow spread than combined tree diameters.

The model 'DBH/D' was used to compare regression parameters from different time specific transmission data. Table 28 shows that for Sinnissippi, unlike the southern Wisconsin data, slopes were more often different than not. Intercept differences also varied widely. Regressions for spread in one and four or more years had greater slopes than those for spread in the same year or three years, in which distance and size had less effect. Intercepts were highest in the same or three years, being most negative in transmissions taking one year. High values for intercepts were associated with low slopes and visa versa.

There are no evident patterns in Table 28 that are easily explained. The results may be somewhat obscured by the changing amounts of variance explained by a particular model (here 'DBH/D') over different durations for spread, as well as the effects of oak wilt control. Use of a different model ('DBH^2/D') yielded a few differences,

TABLE 28. Comparisons of Parameter Estimates: Comparisons of Time Specific Spread, Sinnissippi Forest

LEGEND--see Table 22

Model:
DBH/D

5% Confidence Interval

Point Estimate: no. years	0	1	2	3	4
0	--	MB	Mb	mb	Mb
1	MB	--	MB	MB	Mb
2	Mb	Mb	--	Mb	mb
3	mb	MB	Mb	--	Mb
4	MB	mb	mb	MB	--

but in general the lack of pattern was the same.

It is also worth noting that the correlations for time specific data at Sinnissippi were lower than those for time independent numbers. This fact indicates that, for Sinnissippi at least, time independent regressions offered more stable predictors.

A comparison of parameter estimates for time independent transmission in different timber types is shown in Table 29. 'DBH/D,' again the most consistent model, is used. The four types showed significant differences at the five percent level for almost all slope comparisons. The most unique were Oak Good, where the slope was lower by a factor of 10 and the intercept was much higher, and Oak Poor, which had the highest slope and the lowest intercept. In fact, those timber types with the highest slopes had the most negative intercepts, with overall levels of transmission about the same order of magnitude. Similar results accrued with the 'DBH/D^2' model. The lower slope indicates that size and distance are less important, and/or a smaller range of sizes were sampled. For Oak Good, the latter (at least) was true.

The larger slopes in the other three types suggest that root graft transmission may be rare between small trees. Perhaps these trees had not yet had a chance to form root grafts after a seed origin. On the other hand,

TABLE 29. Comparisons of Parameter Estimates: Between-Site Comparisons for Time Independent Spread, Sinnissippi Forest

LEGEND--see Table 22

Model: DBH/D

		5% Confidence Interval			
	Timber Type	Oak Good	Oak Medium	Oak Poor	Repro-duction
Point Estimate	Oak Good	--	Mb	MB	MB
	Oak Medium	MB	--	mb	Mb
	Oak Poor	MB	MB	--	MB
	Repro-duction	MB	Mb	Mb	--

Slopes: OP ≥ OM > REPRO > OG

Intercepts: OG > REPRO, OM ≥ OP

it may merely reflect the natural bias against small trees and brush areas (less common on good sites) in oak wilt surveys. Not all trees were located and fewer distances were measured in brush areas, as compared to more mature stands, by the original workers at Sinnissippi. Only a portion of the original data on root graft transmission included inter-tree distances.

The significant differences obtained in inter-site comparisons of equations predicting root graft transmission probabilities may reflect factors intrinsic to the sites, both for different site types in Sinnissippi and the various southern Wisconsin sites. In both Wisconsin and Illinois, poorer sites (drier soils) showed higher slopes, and hence more sensitivity of transmission probability to tree size and inter-tree distance. However, since size distributions varied with site, it is not clear whether the quality of the site was a primary factor. Whether the patterns seen reflected differences in stocking, edaphic effects on root growth and grafting, or factors affecting oak wilt epidemiology is an open question.

3. Transmission Probabilities in Red Oak Group Trees — Literature

There are no calculations of root graft transmission probabilities in the literature at all comparable to those

presented here. There is some treatment of distance in many publications. A maximum 50 foot inter-tree distance limit to transmission is often cited (e.g. Nair and Kuntz 1975). Maximum distances reported include 36 and 25 feet in Missouri (Jones and Partridge 1961), 20 feet in West Virginia (Rexrode and Frame 1977), 26 feet in West Virginia (True et al.1960), 28 feet in Tennessee and North Carolina (Boyce 1957b), and 50 feet in Illinois (Himelick and Fox 1961). Mean distances over which root graft spread usually occurs are usually between 8 and 15 feet, indicating that probability of transmission is highest over short distances. Himelick and Fox (1961) found that 91% of all transmission occurred over 30 feet or less; Craighead and Nelson (1960) found 90% in Pennsylvania.

The closest approach to the treatment of root graft transmission probability found in this thesis is a table of maximum root grafting distances for oak wilt transmission for trees of various combined dbh, created by Himelick and Fox (1961) from Sinnissippi data prior to 1960. This table is reproduced in Table 30, along with maximum distances (i.e. minimum distance where probability=0) predicted by transmission probability models for various sites. Within the range of data collected, the results are remarkably close. Important disagreements at very small tree sizes (an extrapolation from Sinnissippi data) may result

TABLE 30. Maximum Root Graft Transmission Distance as a Function of Combined DBH; Results of Several Models.

	MAXIMUM DISTANCE (ft)			
Combined DBH (inches)	Himelick and Fox 1961	DBH/D Sinnissippi Non-Control*	DBH/D ARB*	DBH-D WHA*
2	12	3	4	10
4	13	6	7	12
6	15	9	11	14
8	16	12	14	16
10	18	15	18	18
12	21	18	21	20
14	23	22	25	22
16	26	25	28	24
18	28	28	32	26
20	31	31	35	28
22	34	34	39	30
24	36	37	42	32
26	38	40	46	34
28	41	43	49	36
30	43	46	53	38
32	45	49	56	40
34	48	52	60	42
36	50	55	63	44
38	52	58	67	46
40	54	61	70	48
42	56	65	74	50
44	58	68	77	52
46	60	71	81	54
48	62	74	85	56

*solved for p = o

from non-linearities in the modeled relationships or underestimates of transmission potential of very small trees by Himelick and Fox.

Table 31 presents data from Jones (1971) from West Virginia. They show relative amounts of spread at varying distances from source trees. When relativized for the amount of area (presumably linearly related to oak density) the results resemble those of this study. Note the steep drop in relative infection beyond 50 feet, the oft-cited limit of root grafting. Similar results from a different study (Boyce 1957b) are also included.

4. Time for Root Graft Transmission

The various regressions of spread in a specified number of years have indicated that time for root graft spread seems to have had little effect on the size/distance/probability relationship. But was the time for spread truly independent of tree size and distance? If the fungus moved gradually between trees, one might expect spread between small trees and over long distances to take longer. There is some evidence that the transmission is not gradual (Yount 1958), but the difficulty of isolating C. fagacearum from oak roots (Boyce 1960) makes it difficult to draw conclusions.

This problem was analyzed directly from Sinnissippi

TABLE 31. Relative Amount of Root Graft Transmission vs. Distance from Nearest Previously Diseased Trees

DISTANCE	CUMULATIVE PERCENTAGE*	% AT DISTANCE**	ACREAGE IN DISTANCE CLASS**	RELATIVE AMOUNT PER AREA**	%/AREA**	% AT DISTANCE***
0- 10	13	13	.00541	2403	35.2	22.8
11- 20	47	34	.0126	2698	39.5	35.3
21- 30	66	19	.0198	959.6	14.1	20.5
31- 40	81	15	.0270	555.5	8.1	8.5
41- 50	88	7	.0343	204.1	3.0	9.4
51-200	100	12	2.70	4.44	0.07	3.6

*data from Jones (1971)
**derived from Jones (1971)
***data from Boyce (1957b)

data by regressing combined diameter and distance individually on time to spread. No significant results were found. Multiple linear regressions of these two factors on time to spread were calculated for the whole of Sinnissippi Forest and for spreads that took place in controlled and uncontrolled pockets. None of the regression coefficients were significant. Time to spread appeared to be independent of tree sizes and inter-tree distances.

The empirical results for time for spread are presented for both Sinnissippi and southern Wisconsin, by control, in Table 32. The average time for spread is 2.16 years for Sinnissippi (non-control), but only 0.90 for southern Wisconsin. The shorter average time for southern Wisconsin may reflect smaller average tree sizes, but tree size was not found to be related to time for spread in Sinnissippi data. Both sets of data were fitted to a Poisson distribution, but data on the whole of Sinnissippi, including control, did not show a significant fit.

5. Local Spread outside the Red Oak Group

The data that have been presented refer to spread between members of the red oak group only. Sinnissippi mortality by root graft to white oak and bur oak was too sparse to analyze (see Section VI A). In southern Wisconsin sites, overall mortality of white and bur oaks did not

TABLE 32. Time to Spread for Root Graft Transmission Between Trees of the Red Oak Group, Sinnissippi Forest and Southern Wisconsin

	NO. OF SPREADS AT SITE		
YEARS FOR SPREAD	ALL SINNISSIPPI	NON-CONTROL SINNISSIPPI	SOUTHERN WISCONSIN
0	35	24	130
1	92	75	96
2	76	55	47
3	56	38	19
4	34	23	4
5	29	21	1
6	4	2	
7	1	0	
8	1	1	
Mean	2.23	2.16	0.90
df	6	5	4
X^2 for Poisson fit	16.2	11.3	3.8
Different from Poisson?	Yes, .05	No	No

show any statistical relationship to distance and tree size, and it may thus be assumed that such mortality was primarily the result of vector transmission.

6. Root Grafting in Oaks

The major cause of geographical differences in root graft transmission appears to be differing amounts of root grafting. Table 33 shows some of the values for root grafting in different areas. There is a large disparity between high grafting of the red oak group in the Midwest, and the lower level in the white oak group in the Midwest, and in all oaks in Missouri and the East. Some of the variation in values may reflect different methods used for detection and will certainly reflect stand properties of distance and size. However, the higher amounts of grafting in the Midwest help to explain the contrasting oak wilt patch dynamics (see Section VI B) between the Midwest and the East. Interspecific root grafts are very rare in all areas.

7. Summary

One of the major goals of this research has been to form predictive equations for root graft transmission. The probability of such spread between two trees, based on direct measures in southern Wisconsin and indirect analysis in Sinnissippi, could be predicted by combinations of com-

TABLE 33. Literature Values for Root Grafting*

REFERENCE	LOCALE	METHOD	SPECIES	AMOUNT
Kuntz and Riker (1956)	Wisconsin	Radioactive Tracer and Dyes	R.O.G.	~100%
		Radioactive Tracer and Dyes	White, Bur	rare
		Radioactive Tracer and Dyes	Interspecies	very rare
Parmeter et. al. (1956)	Wisconsin	Oak Wilt	Bur	6%
Beckman and Kuntz (1951)	Wisconsin	Tracer	N. pin	~100%
Himelick and Fox (1961)	Illinois	Oak Wilt	White, bur	rare
USDA (1958)	Missouri	Oak Wilt	White	17%
		Oak Wilt	Black	11%
Jones and Partridge (1971)	Missouri	Oak Wilt	White	16-20%
USDA (1959)	Missouri	Oak Wilt	Black	13-16%
		Observation	All Oaks	15%
Boyce (1957b)	Tennessee, N. Carolina Pennsylvania, W. Virginia	Oak Wilt	All Oaks	2%
		Poisoning	All Oaks	15%
True, et.al. (1960)	West Virginia	Poisoning	All Oaks	10%
Rexrode and Frame (1977)	West Virginia	Oak Wilt (estimate)	All Oaks	10-20%
Rexrode (in preparation)	West Virginia	Pressure injections	All Oaks	31%
		Pressure injections	All Oaks	15%
		Oak Wilt	All Oaks	35%**

* % of trees grafted to at least 1 member of the same species
** after three years

bined diameter ('DBH') and distance ('D'). Individual diameter differences did not substantially affect local spread dynamics in southern Wisconsin. Of the eight models tested, the best predictors of root graft transmission probability were 'DBH/D' and 'DBH-D.' Model results agreed with cruder analyses of previous workers.

Model fits to particular times for spread in southern Wisconsin showed no significant differences in slope, indicating that there was nothing different about the size and distance relationship to spread probability in spreads taking different amounts of time. In fact, time to spread was independent of tree size and inter-tree distance and was fitted empirically with the Poisson. In Sinnissippi, slopes for different length intervals differed, but not in any coherent fashion.

Different sites from each data set showed significant differences in intercepts and slopes for regressions predicting time independent spread at different sites. Thus, site differences affected root graft dynamics more than different durations of time for spread. Slopes (i.e. sensitivity of transmission probability to changes in 'DBH' and 'D') were generally highest on poorer sites and drier soils. Local spread characters that differ between sites may, however, merely reflect differences in densities and size classes of red oaks. Oak wilt control at Sinnissippi

depressed the slope of the predictive equations while increasing the intercept; overall levels of local spread were much lower in these controlled areas.

The above statements apply to transmission between members of the red oak group. Transmission between white oak group members in southern Wisconsin was independent of tree size and distance, indicating most was surely due to vector spread. This agreed with total mortality results at Sinnissippi and with measurements of actual root grafting by several authors. Available data also lent support to the hypothesis that differential root graft transmission primarily reflects differences in actual grafting. Grafting between red oak group trees outside the Midwest is lower and may account for the average smaller pocket size there. This level of grafting may also reflect lower oak densities.

The predictive equations and relationships derived in this section are a much more fundamental type of data than presented in the previous two sections and will be used in a simulation model that will further explore the properties of the oak wilt system.

D. Results -- Vector Transmission

Insect vectors of Ceratocystis fagacearum feed on sporulating mats of the fungus and subsequently may spread

viable spores to wounded, but otherwise healthy, oaks. This transfer is termed vector (or vector-mediated) transmission. Outside of the Midwest, other vectors may transfer fungal mycelia proper. Vector spread may occur at distances greater than possible for root graft transmission and thus initiate new pockets of infection at various distances from extant patches. Quantification of oak wilt dynamics requires understanding of this process.

In particular, answers were attempted to the following questions: How does overland spread vary over time? Can it be related to climatic factors? Over what distances does it occur? Can one predict the amount of vector spread in a local area in a given year? How does the overland spread of oak wilt vary in different species? How important is it in different geographic areas? What size trees are preferentially attacked?

Data from southern Wisconsin did not include differentiation of vector spread from root graft transmission. Sinnissippi Forest included areas under stand level oak wilt control. However, the number of pockets was not affected by control in the 1950's (Himelick and Fox 1961), and levels of overland mortality did not show any discernable relationships to the varying control regimes. This contrasts with observations from other studies on the efficacy of felling and girdling in reducing mat forma-

tion (Morris 1955, Gillespie et al. 1957), beetle infestation (Rexrode and Frame 1973), and new pocket formation (Jones 1965, 1971), as well as shifting mat production to the fall, a less favorable time (Boyce 1954). Most of the Sinnissippi data analyses considered large parts of the forest, and absolute figures should be regarded as approximations.

Literature data on amounts of oak wilt mortality from vector spread are not abundant. The Sinnissippi data presented in Table 13 for new pockets give some idea of the magnitude of vector mortality. Rates of new pocket formation are a lower bound on vector spread, since the latter may also occur into extant pockets. Craighead and Nelson (1960) present overland rates for a county in West Virginia that are much lower than Sinnissippi values (Table 7). Absolute amounts of vector spread would be expected to vary depending on the amount of oak wilt in the area. Estimates of absolute amounts are thus of limited usefulness, especially when the area over which estimates are made differs. Of more interest are patterns of vector transmission over time in the same area.

1. Patterns of Vector Spread over Time

Amounts of vector induced mortality in areas of various sizes at Sinnissippi are summarized in Table 34. The

TABLE 34. Oak Wilt Mortality by Vector Spread in Selected Blocks of Sinnissippi Forest, by Year

Block	Acres*	1956 1957	1958 1959	1960 1961	1962 1963	1964 1965	1966 1967	1968 1969	1970 1971	$\bar{X}$ **	S **
A11	1648	40 57	40 (62)§	59 41	29 44	(51) 37	75 (89)	66 63	44 49	1.28	0.38
126	154	4 5	2 5 (10)	3 2	2 13	8 (17)	14 6	(16) 7	7	1.96	1.31
35	56	4 0	(5) 3	1 0	(5) 3	2 1	3 (5)	1 2	1 (5)	1.83	1.30
100	129	(4) 2	3 2	0 1	3 2	(5) 1	1 1	(14) 7	7 3	1.09	1.08
A3	303	(11) 6	8 8	5 1	(8) 6	8 5	6 9	(16) 9	10 11	1.05	0.44
81	157	1 3	0 4 (10)	2 0	3 2	0 3	(9) 3	4 5	0	0.78	0.76
85	146	4 5	5 6 (10)	7 0	3 2	1 4	(10) 8	2 0	5	1.23	0.87
115	147	3 2	2 (5) 3	(4) 3	2 3	3 2	3 (4)	3 1	3	0.78	0.26
A21	384	9 9	8 11 9	(13) 6	7 13	11 11	(20) 13	(22) 11	14	1.22	0.45
A22	301	10 15	16 (22) 11	8 9	13 (14)	9 (29)	22 16	11 10	13	1.89	0.77
45	<40	0 0	(4) 1 1	0 (3)	0 1	1 (3)	0 0	1 1	(3)	1.19	1.33
50	<40	0 0	0 (1) 0	0 0	0 0	0 0	0 0	(1 1) 0	0	0.19	0.40
59	<40	1 (4)	1 0 0	(4) 0	3 2	1 0	0 1	2 0	1	1.19	1.42
82	<40	0 1	0 1 (3)	2 0	1 1	0 (2)	0 1	0 0	0	0.75	0.93
92	<40	0 (2)	1 0 (7)	0 0	1 1	1 0	0 1	0 0	0	0.88	1.75
101	<40	2 2	3 2 0	1 3	2 0	0 0	0 (10)	2 (6)	1	2.12	2.63
117	<40	0 0	0 (2) 0	0 0	0 0	0 0	0 0	0 0	0	0.12	0.50
119	<40	1 1	(3) 0 0	(3) 0	0 0	1 1	2 0	(3) 1	2	1.12	1.15
155	<40	1 2	1 1 (3)	1 0	1 (3)	2 (6)	5 2	0 1	0	1.81	1.72
189	<40	(4) 0	2 (3) 1	2 (3)	2 2	1 1	1 2	1 1	(3)	1.81	1.05

* approximate acreage of oak types

** per 40 acres. Blocks less than 40 acres in size assumed to be 40 acres

§ circles designate peaks

period of maximum oak wilt control, from 1959 to 1967, did not correspond with any reduction in overland spread. As with data on new pocket formation, the consistency of year to year values in the forest as a whole belied the higher variation present in smaller areas from year to year.

Run tests (Sokal and Rohlf 1969) were used with the vector data on blocks of various sizes and, as with the analysis of patch patterns, did not give significant results. Peaks (Table 34) were chosen objectively in a manner identical to that of pockets, and the results for inter-peak distances, for blocks of 120-400 acres, indicated a preponderance of seven year separations (Table 35). This return time was not evident when considering larger or smaller blocks. The seven year pattern in new pocket formation may thus be attributed to the observed pattern in vector transmission.

The fact that the peaks were abrupt (run tests did not detect smoothly changing patterns) may indicate the highly variable nature of the whole process of vector spread. Weather should be a major factor. Mat production, in particular, is sensitive to particular conditions of temperature and moisture, being favored by wet, cool weather (Morris and Fergus 1952, Curl 1955, Thompson et al. 1965). Boyce (1957a) has found that August to December

TABLE 35. Periodicities in Vector Spread, Sinnissippi Forest, Blocks of 120-400 Acres

Number of Years Between Peaks	Frequency-(Highest Peaks)	Frequency-(All Peaks)
2	3	7
3	1	7
4	1	7
5	1	5
6	1	3
7	6	8
8	0	3
9	3	6
10	0	1
11	0	0
12	1	2
13	0	0

rainfall was a good predictor of subsequent January to April mat production and that yearly rain and temperature were less important. Curl (1955), in his detailed treatise, found that not only was mat production highest in the spring, but so were numbers of conidia, percentage germination of conidia, size of mats, perithecia density, percent germination of ascospores, and insect vector activity. Conidia numbers also peaked in December. Moisture was important in determining the levels of both the spring peak and a lower fall peak. The observation that mat production occurred in Minnesota after a long drought (Campbell and French 1953) was a notable exception to data indicating mat production requires wet weather.

The noted preponderance of seven year return times for overland mortality did not occur at the scale of the whole forest, and different 120-400 acre blocks peaked at different times. If only the population dynamics of the vector were responsible for peaks in overland transmission, or if the patterns were related solely to weather, one would have expected the whole forest to show a distinct pattern. This was not the case, although 1967 was a peak year for many parts of Sinnissippi.

It appears that two waves of infection, seven years apart, passed through Sinnissippi. One hypothesis is that

local microclimatic conditions favored intensification of inoculum in one part of the forest one year and that this high inoculum level spread in subsequent years. Data on spatial patterns of new patch initiation also suggested this. The tendency for a seven year return time was probably unique to the particular history of Sinnissippi Forest.

2. Prediction of Levels of Vector Spread

Curl (1955) has shown that trees dying in a given year will most likely provide inoculum for trees that will die from vector spread in the following year (in trees of the red oak group). In his study, 82% of all mats on trees killed by oak wilt in 1952 were produced between March and July 1953. Such mats would provide inoculum leading to death of trees of the red oak group in 1953. Thus, one would expect that the number of trees dying in a given year from oak wilt would be a good predictor of overland mortality in that area in the following year. Regressions on data from blocks ranging in size from 40 acres to the entire Sinnissippi Forest did not show this relationship, however. Apparently, factors such as weather do control vector mortality levels to a major degree, at least over the mortality range found at Sinnissippi. More extreme differences in mortality (e.g. initial infestation levels)

would probably show a stronger correlation with subsequent vector spread.

An effort was made, by removing other influences, to uncover the relationship between the previous year's mortality in one area and the current year's overland spread. One approximation to the forest-wide favorableness for vector transmission would be the ratio of the current year's vector spread to last year's total red oak group mortality for the whole of Sinnissippi Forest. Thus, the current year's (i) vector spread in a subset (j) of the forest could be predicted by the following relationship:

$$V_{i,j}=m(T_{i-1,j})\ (V_{i,all}/T_{i-1,all})+b \qquad (4)$$

where $V_{i,j}$ is the vector mortality in year i, in block j.
$T_{i-1,j}$ is the total red oak group mortality in year i-1, in block j.
$V_{i,\,all}$ is the vector mortality in year i for the whole forest.
$T_{i-1,all}$ is the total red oak group mortality in year i-1 for the whole forest.

In Equation 4, the 'V/T' ratio for the whole forest represents the effects of all factors other than the previous year's mortality in block j, over the entire forest.

Regressions were used to test this relationship, for blocks of two different sizes. When the forest was divided into four parts (average 412 acres each of oak type), vector mortality ($V_{i,j}, j=1,4$) in these blocks was predicted by

Equation 4 with r=.589**. With smaller blocks of average size 137.7 acres, the correlation coefficient was .512**. Thus, previous year's mortality can be used as a predictor if some information is known about regional or forest-wide favorableness (which for these data is represented by the 'V/T' ratio). There will be more treatment of Equation 4 in Section VII.

Equation 4 is not a true predictive equation since '$V_{i,j}$' is related to the '$V_{i,all}$' term in the 'V/T' ratio. However, the 'V/T' ratio might be related to some weather factors (although variable control regimes may also be important). I attempted to relate the 'V/T' ratio to some weather data collected at Rockford, Illinois. Of the eight crude weather factors examined, none were significantly correlated to the ratio at the five percent level. The current year's temperature was negatively correlated to the 'V/T' ratio only at the 10% level. Addition of other weather factors using multiple regression did not significantly improve the fit. The 'V/T' ratio is evidently either more complexly related to weather, or some other unknown factors are important.

3. Spatial Characteristics of Vector Transmission

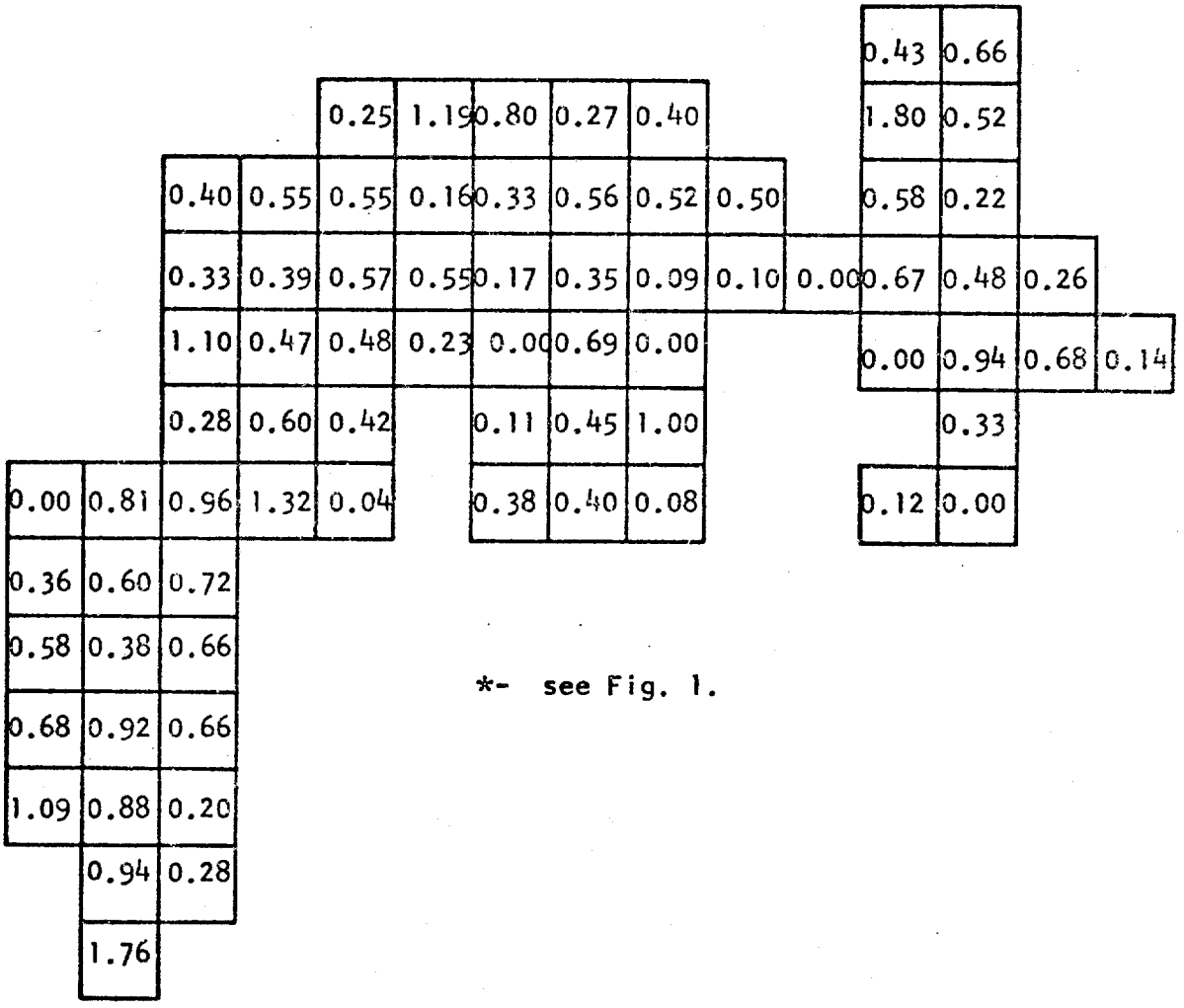
Anecdotal accounts indicate that oak wilt spread over large distances is rare. Often one woodlot shows

extensive damage with many pockets while a nearby one is essentially free of the disease. If composition and site of the two woods are similar, this observation would further support the contention that overland spread principally initiates foci near old pockets and that occasional longer distance spreads are highly stochastic.

There are few studies of oak wilt vector behavior. Morris et al. (1955), using radioactive tracers, found that nitidulids showed quite local dispersal that spread out slowly with time. As long as mats or wounds remained fresh, they would not move away. This local movement is the usual case for most insects.

One can also approach the problem statistically. In Sinnissippi, where oak wilt has been established for some time, did most of the inoculum come from within the stand, or was there an 'edge effect' commonly seen in crop diseases dispersed over long distances (van der Plank 1948)? A comparison was made of vector mortality in the interior of the forest with areas facing the Rock River, facing open fields, and facing areas of mixed fields and woods outside of the Sinnissippi boundaries. Fig. 2 shows the relative amounts of overland transmission by compartment at Sinnissippi, per acre of oak type. There were no significant differences, using t-tests for two independent samples, between compartments partly on the borders of the forest

FIG. 2. Vector Mortality per Acre Oak Type, by Compartment, Sinnissippi Forest (1955-1971) *



*- see Fig. 1.

and those in the interior. The direction that a compartment on the border of Sinnissippi faced (i.e. river vs. fields vs. mixed woods and fields) also did not affect its rate of vector mortality. Apparently, the amounts of inoculum in the forest over the 17 years have been evenly distributed. The results indicate that the movement of vectors from outside the forest was not an overriding force, and lends more support to the idea that overland spread has a strong local component.

How local is the pattern of overland spread within the forest from year to year? A map analysis was undertaken on Sinnissippi data. Minimum distance, in classes, was measured from each tree of the red oak group dying of overland spread to the nearest oak wilt-killed tree dying the previous year. Most inoculum takes a year, from death of the donor tree, to cause death of the recipient (Curl 1955). Linear regression provided the distribution equation for nearest neighbors of the previous year:

$$p = (0.628 \times \text{LOG } d) - 0.980$$

$$r = .999^{***} \qquad (5)$$

Where p is the proportion of trees found up to that distance,
d is the distance in feet.

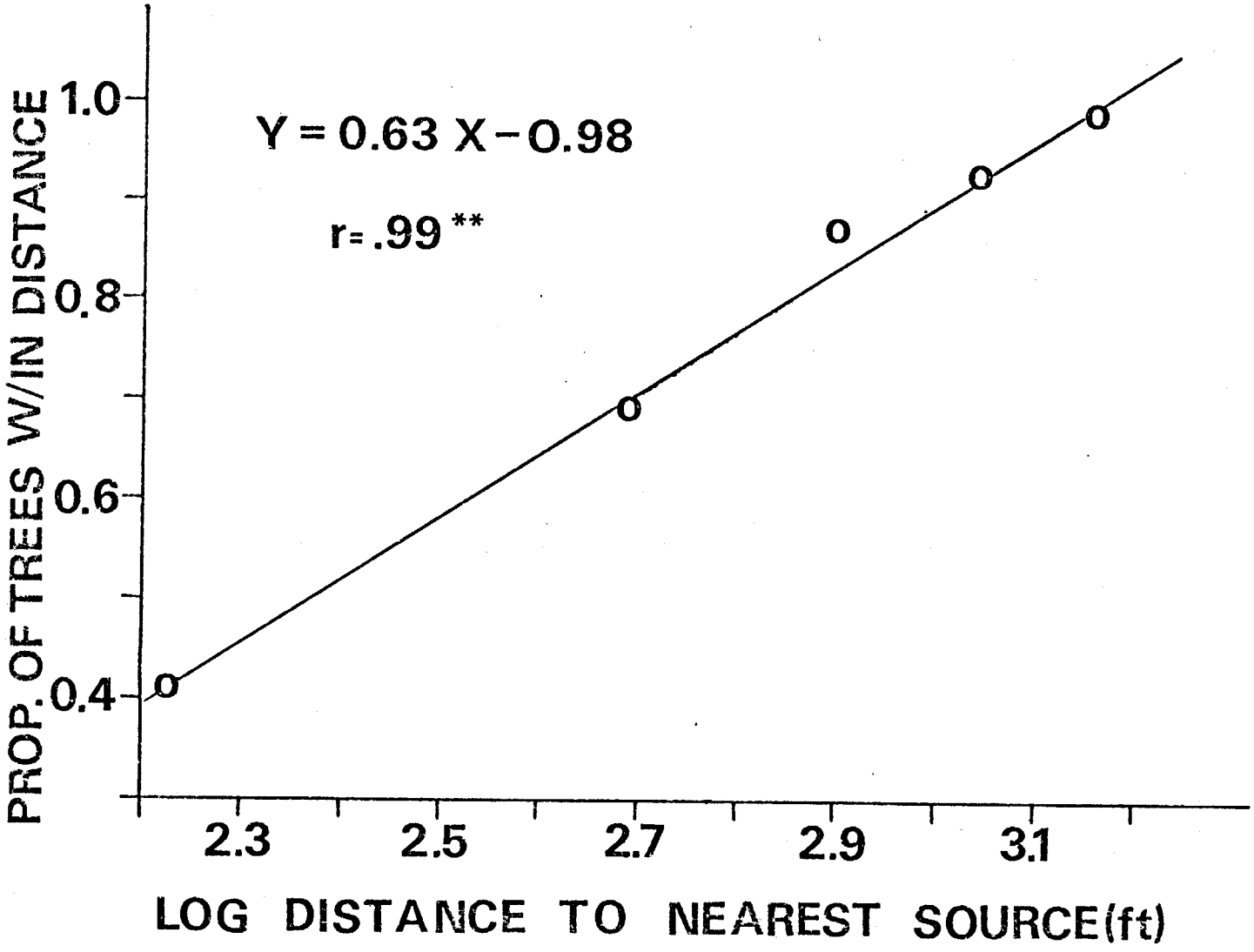
Although the fit is very good, there is no guarantee that such a fit is anything more than a statistic associated

with random nearest neighbors. However, these distances were closer than expected (Chi square test, $p < .05$, 1 df) based on considerations of plotless sampling theory (see Appendix 2).

Equation 5 is a predictive equation that will re-create patterns of overland spread, despite the fact that spread will not always occur over distances that correspond to the nearest neighbor distances measured. Various situations (e.g. different levels of infestation, contrasting compositions) may affect the constants in Equation 5. The key point for this discussion is that the resulting pattern shows significant contagion of new infections to old; this is proven in Appendix 2 and emphasized by Fig. 3.

The fact that vector spread in Sinnissippi appears to be concentrated over short distances, coupled with other evidence presented in Section VI B, supports the hypothesis that overland transmission is an important component of oak wilt spread over short distances. This is not a new idea, although the literature suggests that this process is not always appreciated. Working in Missouri, Jones and Bretz (1958) documented the high number of trees wilting at distances of 50 to 100 feet from previously wilting trees, while Jones and Partridge (1961) observed that known levels of root grafting could not account for observed patterns of pocket formation in that state. Jones (1971) found that

FIG. 3. Cumulative Proportion of Vector Mediated Oak Wilt-Killed Trees at Distances from Nearest Possible Source Tree



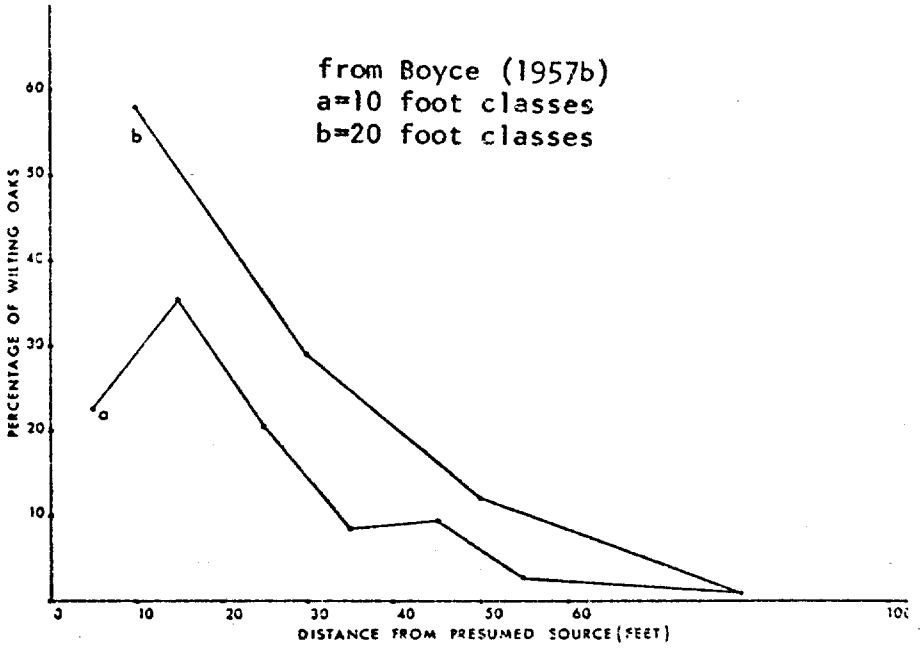
12% of 'local' spread in West Virginia occurred at distances too great for root graft transmission. Indeed, simple arithmetic on root grafting percentages found in Table 33 result in expected mean pocket sizes far below what actually occur in the same areas (Table 18). Even given problems of technique in measuring actual root grafting, it is likely that vector spread accounts for a major part of pocket expansion in areas outside of the Midwest. In the Midwest, high levels of root grafting are still undoubtedly the most dominant force in producing larger pockets, and this allows accurate prediction of root graft transmission probability (Section VI C). Still, the role of vector transmission in local spread has likely been underestimated in the Midwest.

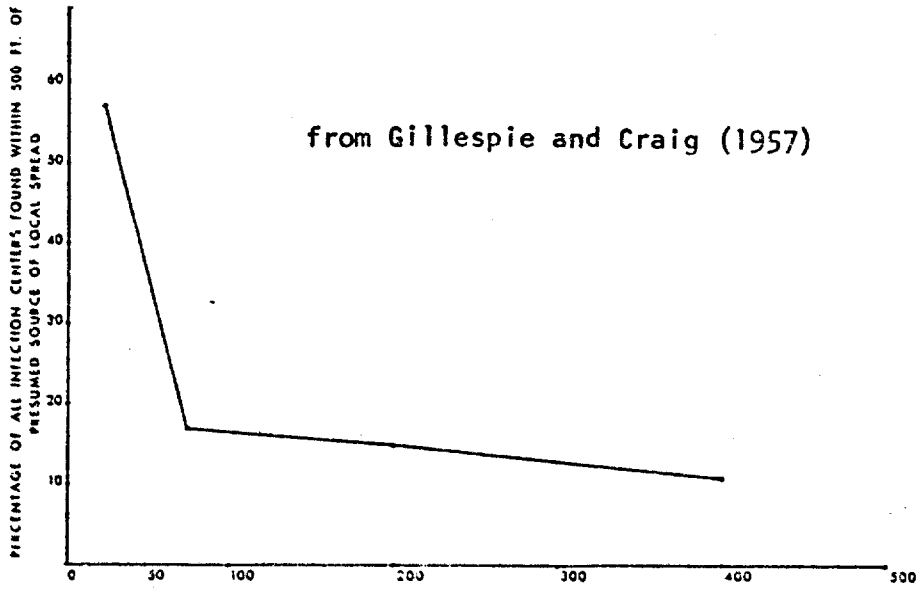
Figure 4 shows the overall results of the strongly distance-dependent root graft transmission and less dependent, but still distance-related, overland spread on patterns of spread at two sites in the Appalachians (Boyce 1957b, Gillespie and Craig 1958). The change in the slope of the line at 35 and 50 feet respectively reflects the existence of the two distinct mechanisms of oak wilt spread.

4. Other Factors Affecting Vector Spread

Wounding is a factor generally associated with increased vector mortality. Jeffrey (1953) found that 108

FIG. 4. Percentages of Wilting Oaks Found at Distances from Presumed Source of Local Spread, Appalachians (figures from True et al. 1960)





of 164 disease areas in Pennsylvania were associated with wounds, and Norris (1953) found that all natural overland infections were associated with wounds of one sort or another. The wounds are attractive to spore-bearing nitidulid beetles. Selective cutting in an area often creates wounds in the remaining trees and is responsible for tremendous losses in areas of new home building (Craighead and Nelson 1960). However, in Sinnissippi, selective cutting in a compartment did not significantly increase overland mortality in the year of cutting or in the following two years. Presumably, cutting took place after the spring season of greatest vector activity (Curl 1955).

One might hypothesize that the surface area of a tree bole would directly influence the probability of a tree becoming infected by vector spread. Larger trees would have, on the average, more area for wounds. Brinkman (1952) and Boyce (1957b) found more mortality in large oaks and Riker's (1951) observation that the first tree affected by oak wilt in an area is often a dominant has been echoed by several workers. Young (1949) and others have found no such relationship, however. Using Sinnissippi data, the size of trees infected by overland spread was compared to the size, by species, of trees in the 1972 inventory. Trees of the red oak group killed by oak wilt were significantly larger (average three inches dbh) than the over-

all population ($p < .01$, t-test), while white oaks and bur oaks had no significant differences. However, the effect of root graft transmission removal of trees over the years of the study and the inconspicuous nature of wilt on small trees are factors which could not be accounted for by any statistical test. Due to these complications, it is difficult to be sure from the Sinnissippi data that large red oaks are indeed more susceptible to vector infection.

5. Summary

In this section, it has been shown that the levels of vector mortality have similar properties to new patch initiation, namely large small-scale year to year variability compared to forest-wide values, and a seven year return time for peaks in overland spread in Sinnissippi. Vector mortality in a small area was predicted by the product of the previous year's total red oak group mortality in that area and the forest-wide ratio of vector spread relative to the previous year's mortality. The latter ratio can be thought of as a representation of the effects of environmental factors on vector spreads. It was, however, only weakly correlated to various crude weather data.

Vector spread occurred most often over fairly short distances, as evidenced by the patterns of vector killed trees and possible donor trees. An empirical distance dis-

tribution was derived from Sinnissippi data. It is hypothesized that this characteristic is indicative of the importance of vector transmission in short distance spread of oak wilt; this will be particularly important in areas of low root grafting.

With the analyses in these last two sections, we now have the basis for predictive equations from which a simulation model can be constructed. This model will be used as an experimental tool to answer questions about the oak wilt system that have not or cannot be answered by traditional field studies.

VII. FORMULATION OF A SIMULATION MODEL FOR PATTERNS OF OAK WILT MORTALITY

A. General Structure and Characteristics

The preceding sections have described and quantified many aspects of mortality resulting from infection by Ceratocystis fagacearum, the oak wilt fungus, for two areas of the Midwest. In addition, relevant data from the literature have been summarized. Predictive equations have been presented for several aspects of oak wilt spread, while statistical tests shed light on some factors important in influencing levels of mortality.

To accompany the preceding partitioning of the oak wilt system into its parts, I will now describe the reunion of these parts into a functional representation of the disease spread process. The vehicle by which this reunification is attempted is OAKWLT, a computer simulator of patterns of oak wilt mortality. By using this simulation model as an experimental tool, more questions on the oak wilt disturbance can be answered.

OAKWLT is written in modified FORTRAN and is currently operable on a Harris Datacraft computer. A copy of the program code is available from the author.

Section IV contains a general description of OAKWLT in terms of one scheme of model classification. The gen-

eral model structure is based on a conceptual organization of the functioning of the oak wilt disease. Compared to this general organization, many of the subunits of the model are less mechanistic and more statistical, based on correlations described in previous sections. Other sections are based on more qualitative descriptions. The model may be termed empirical, since its general structure, specific component sections and equations, and parameter values are based specifically on oak wilt dynamics. The model is also stochastic, meaning that successive runs with identical starting conditions will yield different results. This was thought most realistic for modeling the real system. In addition, many of the relationships described previously are explicitly probabilistic. The stochastic nature of OAKWLT means that several runs with each set of starting conditions will be necessary to provide reasonable estimates of output values. An advantage that accrues is that a measure of the variance associated with a particular set of parameter values may be obtained.

Oak wilt-caused mortality of individual trees is modeled by OAKWLT. Each tree has the attributes listed in Table 36. Most of these do not vary during simulation; these include location, dbh, and species. In addition to not incrementing tree size over time, the model does not consider ingrowth into the stand or mortality from other

TABLE 36. Attributes of Trees in OAKWLT

Attribute	Fortran Name	Value definitions and units	Comments
Location	X(I) Y(I)	X and Y coordinates, units in feet	Distribution may be regular or random; latter constrained by distance vs. size criteria.
Species	SPP(I)	0=red oak group 1=white oak 2=bur oak	May be specified for each tree or generated stochastically from composition statistics.
Size	DBH(I)	units in inches, to the nearest inch	Generated from specific values, or densities by size class, or stochastically from specified normal distributions.
Living Status/ Method of Infection	DEAD(I)	0=alive 1=dead by root graft transmission 2=dead by within-plot vector transmission 3=dead from background infection	Initial values usually zero.
Year Died	YRDEAD (I)	0=alive other integer-year died	Initial values usually zero.
Pocket ID	POCKET (I)	ID number of pocket, assigned sequentially	Initial values usually zero.

causes. The attributes that may change during simulation include living status/method of infection, the year of death, and the pocket number. Each tree has an identifying number (ID), which is used as the subscript in each of the parallel arrays of attributes.

The time step used in the model is one year. Within-year dynamics are subsumed in some of the yearly equations, but are explicit in others.

Conceptually, OAKWLT may be divided into five major parts which will be described in more detail. They are:

1) initial conditions and stocking
2) background infection (overland infection from outside the plot)
3) within-plot overland infection (local overland infection)
4) root graft transmission
5) output and bookkeeping.

There are one main program and seven subprograms that handle various aspects of the simulation. Fig. 5 is a flow chart of OAKWLT that also shows clearly the five basic sections outlined above. The following sections outline the model formulation and parameter assignments in more detail.

B. Initial Conditions and Stocking

Each simulation begins with the reading in of parameters specifying initial conditions, followed by stocking of the stand. This happens only once during the simulation.

FIG. 5. General Flow Chart for OAKWLT Model

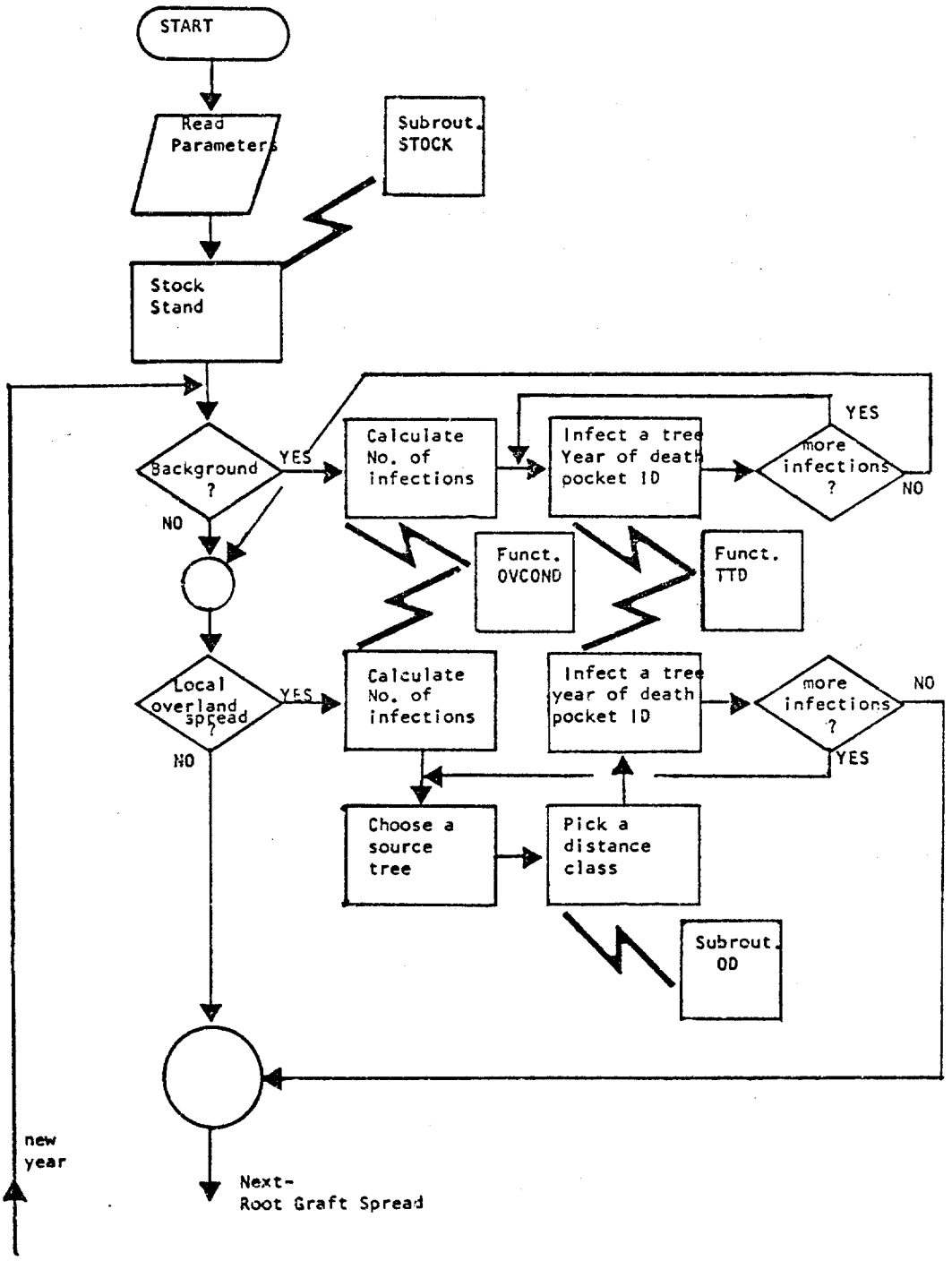
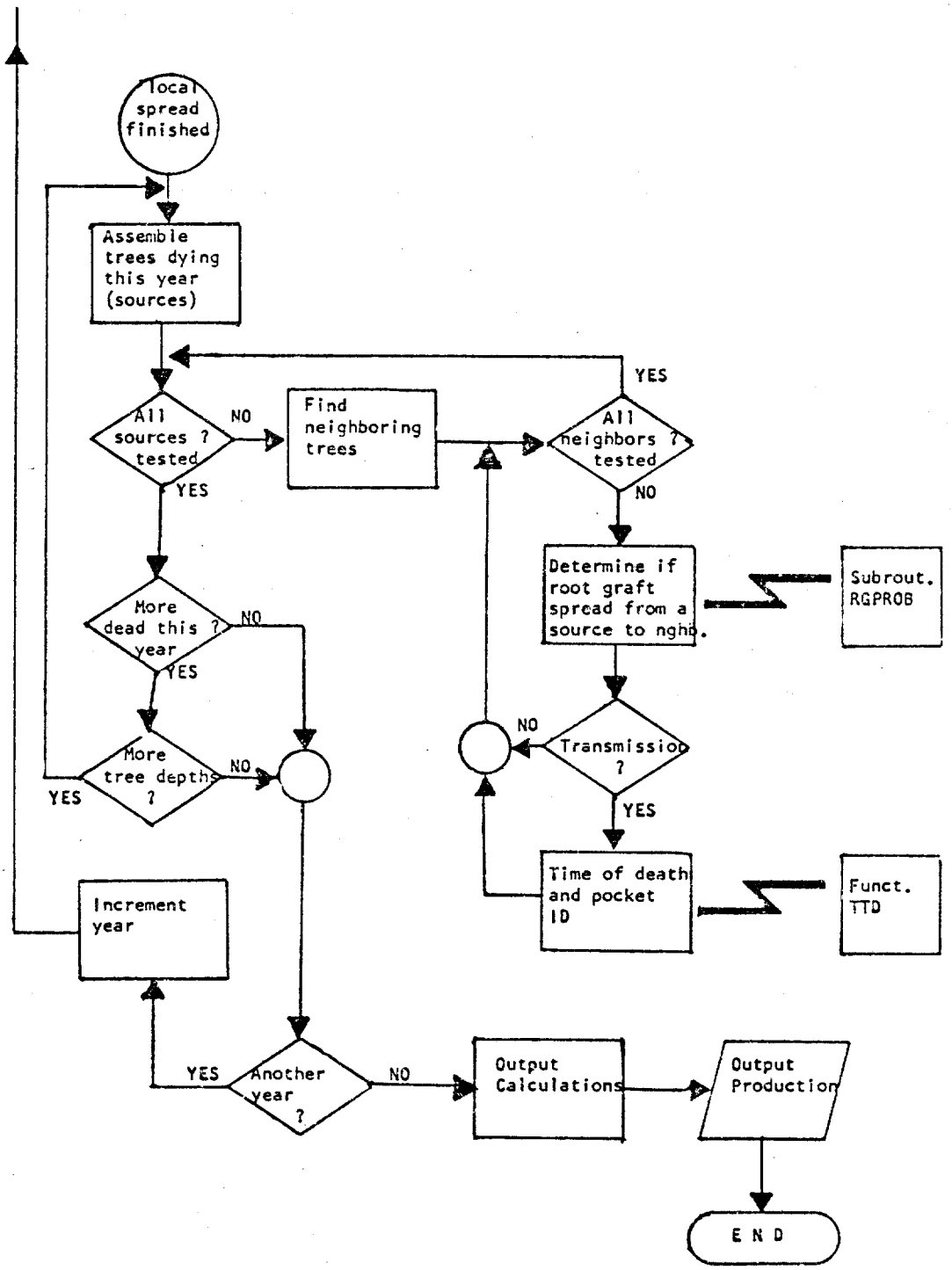


FIG. 5. (continued)



The parameters specified by input may be divided into two groups: run parameters and biological parameters.

Run parameters are used to define the simulation. They include the number of simulations in the run with all other parameters the same, the number of years of each simulation, the size of the plot, the amount of data used in stocking, and whether the plot begins with dead trees. Other run parameters are concerned with characteristics of output. Diagnostics are controlled by sense switches on the Datacraft computer which may be manipulated during the run.

Biological parameters are those values which determine the results of OAKWLT simulation; these may be varied from run to run. Many are concerned with stocking the stand (see Table 37) and include composition, size class information, and density. Other variables affect the characteristics of spread. These are shown in Table 38 and include the level of background spread, average rates of total vector spread, and regression constants for root graft transmission predictive equations.

The stand is stocked with trees by the subroutine STOCK. The tree attributes resulting from stocking (Table 36) are held in a COMMON block, and the various stocking parameters are passed as arguments. STOCK can handle three types of data. Data type 1 is a complete specification of

TABLE 37. Input Parameters Related to Stocking in OAKWLT*

Variable Name	Type	Definition	Values
COMP(I)	REAL	% of oaks of a species	COMP(1)=% of red oak group COMP(2)=% white oak Remainder are bur oak
DEN	INTEGER	Number of oaks/acre	Maximum values constrained by dimensioning
PATTRN	INTEGER	Code for pattern	1=random 2=regular
PLOT	REAL	Number of acres in plot	Maximum values constrained by dimensioning
SIZEM(I)	REAL	Mean of normal size distribution for a species	SIZEM(1)=red oak group SIZEM(2)=white oak SIZEM(3)=bur oak
SIZEV(I)	REAL	Standard deviation of normal size distribution for a species	See SIZEM(I)

* not all parameters are used for all types of stocking.

TABLE 38. Non-Stocking Biological Input Parameters in OAKWLT

Variable Name	Type	Definition	Values
BGRND	REAL	Code for level of average background inoculum	Rate of introduction corresponding to "BGRND" percentage of all vector transmission in an average year at Sinnissippi.
OVERFN	REAL	Code for average level of favorableness to vector spread	The number of trees infected on the average, by vector spread from a wilt killed tree; Sinnissippi value = 0.578
RGPARM(1)	REAL	Slope of root graft transmission probability equation	Using CDBH/D model; nominal value = 16.4
RGPARM(2)	REAL	Y intercept of root graft transmission probability equation	Nominal value = -6.9

all tree attributes, including location. For data type 2, the composition and sizes of trees are determined by parameters representing densities of trees, by species, in particular size classes. Within a size class, random integer values are assigned for a tree's dbh. The most used type of stocking data, type 3, requires the minimum amount of input. The species of each tree is determined stochastically from composition values for the three possible species. Thus, if the composition is 40% white oak, any particular tree has a 0.4 chance of being designated a white oak. The dbh is drawn from a species specific normal distribution, with mean and variance specified by input parameters. Normal (or near normal) distributions of oak populations are common (Schnur 1937, Johnson and Bell 1974) and were found for Sinnissippi Forest inventory data (Table 2).

For data type 3, the pattern of tree placements may be random or regular. The placement occurs within the plot, the size of which is determined by inputs. The number of trees in a plot depends on plot size and oak density. Random pattern is constrained by a minimum nearest neighbor distance based on combined dbh. This relationship was derived empirically from mapped data for Noe Woods, Univ. of Wisconsin-Madison Arboretum:

$$MD = 0.154 * CDBH + 2.872 \qquad r = .851^{*} \tag{6}$$

where MD is the average of the five smallest distances (in feet) between oaks in a 4" diameter class; and
CDBH is the combined diameter in inches.

When stocking, no trees may be placed closer together than the distance MD.

Except for data type 1, all simulations begin with every tree alive, and hence in no pocket. STOCK, as well as stocking the stand, does bookkeeping on the actual composition and size characteristics of the stand. Once stocking is completed, the simulation starts at year one with vector infection.

C. Vector Infection

Vector infection is modeled by OAKWLT in two parts: background infection from outside the plot, followed by within-plot overland infection. Parameter estimation from Sinnissippi data involves both processes together, since the source of inoculum is not known for most field data.

Curl (1955) has shown that in the red oak group over 80% of the inoculum capable of infecting trees by vector transmission in a given year originated in mat production on trees dying in the previous year. Previous oak wilt mortality in local areas in Sinnissippi was shown not to be a good predictor of subsequent vector transmission (in

Section VI D), but a predictive equation (Equation 4) was formed by weighting previous mortality by the overall forest-wide (or regional) efficacy of vector spread (the V/T ratio). The latter statistic was not highly correlated to simple weather data.

Weather per se is not a part of OAKWLT. The regional efficacy of vector spread is assumed to vary randomly, leading to a restatement of Equation 4 for model purposes as:

$$V_i = m(COUNT)(OVYR) + b \qquad (7)$$

where V_i is the vector mortality in year i
COUNT is the total mortality in year i-1 (T_{i-1})
OVYR represents all conditions over the geographic area, including weather, other than the previous year's local mortality, influencing year i vector transmission; and
m and b are regression constants.

OVYR has a value drawn stochastically from a normal distribution with mean and variance of the V_i/T_{i-1} ratio (described in Equation 4) as nominal values. Equation 7 is a general model that serves as a basis for the succeeding specific equations.

1. Background Infection

Levels of background spread do not depend on within-plot inoculum levels. Equation 7 describes the dependency

of overland spread on previous mortality, and the Y intercept of this equation (i.e. the point at which $T_{i-1}=0$) approximates the overland mortality that is not explained by local inoculum considerations. Since the regression fit in Equation 4 is based on smaller blocks than the whole of Sinnissippi Forest, the intercept includes spread from other blocks, as well as being influenced by less than perfect fit. For four blocks averaging 412 acres of oak type, the Y intercept was 43.1% of the average Y value. For blocks averaging 137.3 acres, the Y intercept represents 54.0% of all spread. The rate associated with these percentages (21.384 introductions/40 acres/year) is then an estimate of background spread, assuming that all infection not explained by the slope of the regression originated from outside the block.

The local nature of overland spread would suggest that these values are high for woodlots. Analogous literature estimates are county-wide rates for new pocket formation, and range from 0.7/40 acres/year in Wisconsin (Anderson and Anderson 1958) to less than 0.0001/40 acres/year in the East (Jones 1965, Boyce 1959). Amounts of new pocket formation include all vector spread except that into extant pockets. Of greater relevance are ratios of new pocket formation in locally infested areas to the

above county-wide estimates; they are 290,000:1 in Kentucky, N. Carolina, and Tennessee (derived from Jones 1965, Boyce 1959), and 208,000:1 in West Virginia and Tennessee (derived from Jones 1965). The high rates of pocket initiation in locally hard hit oak wilt areas relative to that in large geographical areas suggests that background spread (which should be closer to the county-wide estimates) is much less important than the 43% contribution based on the Sinnissippi regression predictions. Rates of new pocket formation in newly affected areas (e.g. 3.96/40 acres/year, derived from Wysong 1949) are not particularly useful as estimates of background spread, since these are most likely exceptionally high values.

It is thus difficult to pin down the exact contribution of background spread to oak wilt dynamics, since it will vary with the region, the inter-woodlot distance, etc. Levels of background infection used in the model (entered via BGRND) will correspond to 1-43% of all infection by vector in an average year at Sinnissippi (i.e. .0124-.5346 introductions/acre/year or 0.497-21.384/40 acres/year). BGRND is expressed as a percentage, and the following equation is used to predict the actual number of background infections in a given year:

(8)

NUMINF=0.02145 * BGRND * OVYR * PLOT

where if BGRND=1.00 (1% of all infections in an average year at Sinnissippi);
if OVYR=0.578 (average $V_{i,all}/T_{i-1,all}$ for Sinnissippi);
if PLOT=1.0 acres;
and if NUMINF=0.0124/acre (1% of all infections in an average year at Sinnissippi);
then the constant=0.02145.

OVYR is stochastically determined in any one year by drawing from a normal distribution with mean OVERFN and standard deviation 0.501 * OVERFN. For nominal simulation, OVERFN=0.578; this and the corresponding standard deviation are values for the V/T ratio. In the simulation model, the function OVCOND determines each year's OVYR.

Using area (PLOT) in Equation 8, rather than the number of oaks (e.g. DEN), assumes that the number of background infections a vector is capable of initiating is not limited by the number of suitable oaks. This formulation may result in overestimates of background spread in woods with very low oak density or in the later stages of severe epidemics. This is a factor only in background spread, however, which will usually be low relative to other types.

NUMINF is subsequently adjusted to take into account the number of trees left alive. If a tree is selected that has previously been killed by oak wilt, that introduction will be ignored and a new tree will be selected. NUMINF is thus an upper limit on the actual number of background

infections in a year.

There was no evidence that indicated background infection occurred with any edge effect (Section VI D), despite the generally local behavior of vectors. The plot size used will be at the scale of a woodlot, most often 10 acres. Edge effects will be ignored and up to NUMINF introductions will be made randomly with respect to spatial position.

Red oaks showed a tendency for size influenced vector infection, while white oaks and bur oaks did not. There are reasons to be wary of the red oak group conclusions (Section VI D) and thus introduction of overland infection will be made randomly with respect to tree size.

When one (I) of the NUMINF trees is chosen, DEAD(I)=3; that being the code for background infection (see Table 36). The function TTD then determines the year of death for each tree. For overland infections, all trees of the red oak group are assumed to die in the same year as infection. In Sinnissippi, although white oaks made up nearly one half of all oaks, only 10% of vector deaths were to this species. Assuming that trees of the red oak group are totally vulnerable, about 10% of infected white oaks die. This weighting was used. Jones (1958) followed white oaks showing symptoms of infection for five years and found only a 19% mortality in that time. Members of the white

oak group may take as long as seven years to succumb if they die, although there is little quantitative data available. An arbitrary distribution of 0-7 years for time to death was used for the white oak group.

Once the year of death is determined, the pocket ID of the newly killed tree is assigned. The criteria for inclusion of a vector-killed tree in an extant pocket are:

1) Trees less than 51 feet apart,
2) One tree in the pocket died within five years of the year of simulation.

If no trees are found fulfilling these criteria, a new pocket has been formed and the tree is given a new pocket ID. If more than one pocket assignation is possible, the search algorithm will choose the pocket having the tree with the lowest ID number. Thus, as with field data, the pocket is not strictly a functional characteristic. The numbering of pockets also does not include such factors as merging of expanding pockets.

2. Within-Plot Overland Spread

Equation 4 predicts vector mortality in a local area based on total mortality in the previous year weighted by the overall efficacy of spread in a larger area. The constants in Equation 4 were estimated for blocks averaging 412 acres in size, creating the following equation:

(9) $V_{i,j}=0.5805(T_{i-1,j})(V_{i,all}/T_{i-1,all})+5.558 \quad r=.58^*$

Background spread was considered in the previous section. For within-plot spread, I would like an equation that predicts $V_{i,j}=0$ when $T_{i-1,j}=0$; i.e. no local vector spread when there is no local inoculum possible. With that constraint, the best linear fit to the data used in forming Equation 9, using model variables as in Equation 7, is:

(10)
NUMLOC=1.0190 * OVYR * COUNT
where NUMLOC=the number of local vector infections in year i ($=V_{i,j}$),
OVYR represents the overall efficacy of overland spread, represented by the V/T ratio,
and COUNT the total oak wilt mortality in year i-1($=T_{i-1,j}$).

Again, the values of OVYR are taken from a normal distribution with mean OVERFN (0.578 for the nominal simulation) and standard deviation 0.501 * OVERFN. Analogous estimates to OVERFN were made by Jones (1965) in West Virginia and Pennsylvania (0.45) and Tennessee (0.22). Note that NUMLOC is independent of area (PLOT) per se since COUNT specifies the potential inoculum level.

NUMLOC source trees (i.e. trees that can provide inoculum) are randomly picked, with replacement, from COUNT trees dying the previous year. For each source tree, a distance for within-plot vector spread is picked from the probability distribution described by Equation 5. For the model, these are picked in 165 or 330 foot classes,

corresponding to the classes used in summarizing Sinnissippi data and forming the regression equation. The subroutine OD contains this distribution. When this distance approaches half of one of the linear dimensions of the plot, plot edge bias can result; this has been a factor is succession models that consider spatial pattern explicitly (Ek and Monserud 1974). However, the plot being simulated is considered to be a woodlot with discrete boundaries, and this bias is in fact desirable. There will be less vector spread over very large distances than expected from Sinnissippi data, since this spread will often end up outside the woodlot.

Once a distance class has been chosen, trees are picked randomly until one falls within the class. After 25 unsuccessful picks (indicating a poorly represented class), a new distance class is chosen. Once an appropriate tree is picked, it is infected if it is alive. If it is dead or dying, the program considers the next instance, again effectively weighting the actual number of overland infections by the number of live trees. NUMLOC is thus an upper limit on the actual within-plot vector spread.

Once a tree (I) is newly infected, the procedure is analogous to that of background spread. For trees of the white oak group, it is determined whether the tree will

die (DEAD (I)=2). The time until death and the pocket status are determined. After up to NUMLOC trees are infected, the program moves to consider root graft transmission.

D. Root Graft Transmission

Root graft transmission from a wilt-killed tree to a healthy one is determined in the year of simulation in which the first wilt-killed tree dies. The first part of this section of the model gathers these trees into an array INFECT(J).

From each tree, all possible root graft transmission candidates are found. These are live trees within 50 feet of the wilt-killed tree. The subroutine RGPROB does most of the important calculations. Regression equations derived in section VI C are used, of the form:

(11) $p=m(CDBH/D)+b$

where m=RGPARM(1), and
b=RGPARM(2).

The parameter values are 16.40 and -6.90 for m and b respectively for the nominal simulation. This equation represents data from the Arboretum for time-independent spread. Values of 24.1 and -9.1 (time-independent spread at West Hill) were also used, with little difference. The regression parameters are input parameters that may be

varied from simulation to simulation.

These equations were derived for trees of the red oak group. There were not sufficient data to determine equations for white oak and bur oak, although transmission was low. In Sinnissippi, the amounts (weighted by composition) of root graft transmission relative to trees of the red oak group were 0.32% for white oak and 7.06% for bur oak. Therefore, the probabilities in Equation 11 are weighted by these amounts for these species. The probability of interspecific root graft transmission is assumed to be zero.

Once the probability is calculated (as a percentage), a random number from 1 to 100 is drawn. If it is less than or equal to the probability, there has been a successful root graft transmission to tree I (DEAD(I)=1). The function TTD determines time until death, based on empirical data for root graft spread between members of the red oak group (Table 32) and independent of tree size and inter-tree distance (Section VI C). This relationship is assumed to hold for the rare occurrences of local spread in white and bur oak. The pocket is the same as that of the tree from which spread occurred. Again, the pocket ID will reflect the first possible transmission; the possible transmissions are tried in descending tree ID order.

If successful root graft transmission resulted in additional mortality in the current year of simulation, additional spread in the same year is possible. This is handled in a parallel manner to the above, with the constraint that a maximum of four tree depths of mortality are allowed in any one year. This constraint was rarely invoked.

E. Bookkeeping and Output

After root graft transmission is simulated, the model begins a new year of simulation. There is considerable bookkeeping taking place during actual simulation, but much of this involves diagnostics whose expression may be suppressed by sense switches. Other bookkeeping is essential and keeps track of such values as the current year's mortality and the number of tree depths of root graft spread.

Non-diagnostic output is postponed until after simulation and is controlled by the subroutine OUTPUT. All output is taken from the final values of tree attributes and is thus somewhat distinct from during-simulation diagnostic bookkeeping. Before output is produced, trees dying after the last year of simulation are restored to life.

OUTPUT handles three levels of production. Maximum

output includes a listing of the attributes of all dead trees. At an intermediate level, detailed tables of yearly progress and individual pocket dynamics are produced. Minimal output consists of summary tables only. The input variable RAW controls the level of output.

VIII. RESULTS OF MODEL SIMULATIONS

A. The Nominal Simulation

The nominal, or baseline, simulation is the basis for further explorations of properties of the OAKWLT model. In essence, the nominal simulation is an attempt to mimic as closely as possible patterns of oak wilt mortality in Sinnissippi Forest. Some aspects of these patterns are discussed in Section VI A and B. The equations for oak wilt simulation are derived from data analyses performed in the root graft transmission and vector transmission sections of the results (VI C and VI D).

The output that is considered includes:

1) total mortality, by species and method of infection.
2) relative mortality, by species and method of infection.
3) pocket statistics, such as average pocket size.
4) temporal patterns of oak wilt mortality.

The parameter values used in the nominal simulation are discussed in the previous section (VII). The root graft transmission equation used was derived from data gathered by Kuntz and associates in southern Wisconsin. All other data are from Sinnissippi. There was one parameter that could not be fixed from a priori considerations; that was BGRND, the level of introduction of inoculum from

outside the plot. An upper limit of 43, corresponding to 0.54 introductions/acre/year, was derived.

The nominal simulation is a validation of sorts, in the sense of confirming the ability of the model to produce reasonable output, but this is limited since it is being used to mimic oak wilt damage in non-controlled compartments of Sinnissippi. BGRND was varied until the model output fit Sinnissippi data closely. A plot size of 10 acres was used. It will be shown that different plot sizes would have generated different nominal values for BGRND.

The OAKWLT model generally begins with no oak wilt in the area, and thus no local incculum or root graft transmission until they are initiated from background mortality. The period of data collection at Sinnissippi was pre-dated by many years of infestation, as is evidenced by data now housed in the Illinois Natural History Survey. It was decided to run the model for 40 years and use the results from the second twenty years to compare to Sinnissippi data. Density and composition at year one were manipulated iteratively to give values at year 20 that were, on the average, similar to the desired composition and density values.

Average Sinnissippi values for number of oaks per acre and percent of oaks that were in the red oak group were about 82 and 51 respectively. However, the effects

of oak wilt on a forest in which all areas were half red oak group, and the impacts on a forest where half the area was 80% red oak group and half 20% are likely to be quite different. The model results showed that patchier distributions of compositions had a major effect in increasing both total mortality and root graft transmission, as compared to a more homogenous forest (Table 39).

Thus, the nominal simulation is necessarily an average of many simulations on stands of differing compositions. The nominal simulation weights results from stands of differing composition by the frequency with which they occurred in Sinnissippi Forest (samples from 40 acre blocks). It was assumed that heterogeneities in other factors, such as density, would not be as important. For example, density might often be inversely correlated with tree size, and the effect of such heterogeneities on observed mortality would be lessened.

Table 39 shows observed results from data analyses of Sinnissippi check areas in the left three columns. The nominal simulation results are shown in the center column, with the final three columns showing the effects of increasing the heterogeneity of composition, holding the average composition at 50%. The best nominal simulation occurred with BGRND=2.30, or 5.68 introductions per 10 acres in 20 years.

TABLE 39. Comparison of Oak Wilt Mortality from Nominal Simulation (NS) and Sinnissippi Check Areas.

	SINNISSIPPI CHECK AREAS			OAKWLT RESULTS			
					Compositions (% R.O.G.) at		
VALUE*	Mean 1950s***	Mean 1960s§	Approx. Range of Values in ¼ blocks§§	NS§§§	All 50%	½ 65% ½ 35%	½ 80% ½ 20%
Total Dead	40.0	25.0	15-100	41.0	24.1	41.8	80.7
Red Oak Group Dead	39.7	25.0	---	39.5	22.8	39.8	79.2
White Oak Dead	0.3	0.5	---	1.6	1.4	2.0	1.5
% R.O.G.	99.2	98.1	---	96.3	94.4	95.2	98.1
Root Graft Spread**	27.5	12.0	4-85	20.2	10.0	19.9	44.5
Vector Spread**	12.5	13.6	10-22	20.8	14.1	22.0	36.2
% Root Graft	69.0	40.0	30-75	49.3	41.5	47.6	55.1

* per 10 acres in 20 years

** for Sinnissippi data, many trees had an unknown method of infection. For this table, they were apportioned to root graft and vector in proportion with which those two types of spread occurred.

*** 1955-1959

§ 1960-1971

§§ Blocks averaging 412 acres oak type

§§§ Average of many simulations of various compositions weighted by actual occurrence in Sinnissippi, BGRND=2.30.

There is generally good agreement between the nominal simulation (NS) and the results from Sinnissippi check areas. Recall that the total mortality and patch dynamics results from Sinnissippi did not enter into model formulations, with the exception of the empirical weightings for mortality in white oaks. Modeled total mortality and vector transmission are at the high end of averages from Sinnissippi for the 1950's and 1960's, but well within the range for individual treatment blocks. The percentage of deaths attributable to root graft transmission is fairly typical.

One discrepancy is the higher value for white oak deaths in simulations as compared to observed values. The previous section (VII) describes the 0.1 vector death rate and 0.0032 value for root graft transmission, for white oak, as compared to the red oak group. There were relatively more white oak deaths in oak wilt control areas at Sinnissippi, and thus the check areas show much lower relative mortality of white oaks. The observed data were taken from these check areas.

Since I have now shown that the nominal simulation approximates observed Sinnissippi values that were derived independently, it is now time to turn to more interesting matters. The next three sub-sections will consider characteristics of individual runs, multiple

runs with the same parameters, and the effects of parameter changes on various output.

B. Patterns within Single Simulations

It will be shown in succeeding sections that the simulation results from this stochastic model are highly variable, even in multiple runs with identical conditions. Since it is difficult to generalize about patterns from single simulations, only some of the most obvious trends will be briefly described here.

One of the unexpected results, given the observed temporal patterns in Sinnissippi, was the smooth rise and fall of mortality over time, with the peak generally occurring between 15 and 30 years, depending on conditions. There was no evidence of periodicity, although observed patterns in Sinnissippi took place over a greater area than 10 acres. This may indicate either that the spatial scale is important, or that a randomly varying environment is not sufficient to produce such patterns. Root graft peaks tended to occur slightly after peaks in overland transmission. Also, simulations with predominantly root graft mortality tended to show later peaks than those in which most deaths were vector mediated.

In later sections, data are presented concerning average pocket size. The frequency of pocket sizes was

always heavily skewed, with many pockets of one and two trees, and occasional large pockets (see Table 40). This tends to parallel literature reports, although such data are usually only expressed as average pocket size.

Finally, as a consequence of the distance equation for overland spread (Equation 5), new pockets tended to be clumped, especially initially. This is analogous to the fine-grained contagion expected when new species are invading communities (Greig-Smith 1957). Since the simulated woodlot was small (10 acres), most simulations eventually resulted in some oak wilt in all parts of the woodlot.

C. Variation in Multiple Simulations with the Same Parameters

As mentioned in the model description, OAKWLT is explicitly stochastic. That is, different results are obtained from multiple runs with the same parameters. Because of this, model outputs (e.g. the nominal simulation) are discussed in terms of mean values of many simulations. This section explores the variability inherent in the model.

The measure of variation employed is the ratio of the standard error to the mean. Standard error is defined as the standard deviation divided by the square

TABLE 40. Summary of Size Distribution of Pockets from 20 Runs of NS at 65% Red Oak Group Composition

RANGE OF AVERAGE POCKET SIZES		0-1.0		1.1-2.5		2.6-3.0		3.1-4.2	
NO. OF RUNS		3		8		5		4	
NO AND % OF POCKETS OF SIZE*		No.	%	No.	%	No.	%	No.	%
	1	5	100.0	94	49.2	117	50.9	91	40.1
	2	0	0	45	23.6	38	16.5	36	15.9
	3	0	0	21	11.0	24	10.4	25	11.0
	4	0	0	11	5.8	18	7.8	19	8.4
	5	0	0	6	3.2	5	2.2	14	6.2
	6	0	0	7	3.7	7	3.0	10	4.4
	7	0	0	3	1.6	5	2.2	6	2.6
	8	0	0	3	1.6	4	1.7	6	2.6
	9	0	0	0	0	1	0.1	2	0.9
	10+	0	0	1	0.1	11	4.8	18	8.0
TOTALS		5	100.0	191	99.8	230	99.7	227	100.1
AVERAGE NO. POCKETS PER RUN		1.7	--	24.0	--	46.2	--	56.8	--

* size measured in number of wilted trees

root of the number of samples; in this case the number of simulations. Most model runs consist of 30 simulations with identical parameters. If the mean equals the standard deviation, 30 runs brings the standard error to mean ratio to 18%. In ecology, an arbitrary cutoff for adequate samples is often made when this ratio is less than or equal to 10%. However, if the mean equals the standard deviation, this 10% cutoff requires 100 runs.

Table 41 shows the standard error to mean ratios for total mortality, for runs with single parameters either raised or lowered 10% from nominal values. With 10% increases in parameter values, the average total mortality increased, but the variability, as expressed in the ratio, decreased. The variability was also lowest for mortality through 40 years, as compared with the greater unpredictability for 20 year results.

Not all types of output exhibit the same degree of variability as total mortality. Table 42 shows seven types of 40 year output and their variability for simulations at +10% for individual parameters. The variation in background mortality is generated from the variation in OVCOND (with mean OVERFN), which is fairly low. It is not affected by the progress of mortality in the woodlot. Both other types of spread show higher variability, with root graft transmission being slightly less predictable than local overland

TABLE 41. Average Standard Error to Mean Ratios (in %) for Model Output; Total Mortality for Single Parameter Changes of ± 10%

Average of Individual Parameter Changes of	RATIOS FOR-- First 20 years	Second 20 years	All 40 years
+ 10%	22.8 (18.4-27.4)*	18.4 (15.6-22.0)	16.2 (12.4-20.3)
- 10%	29.0 (21.2-40.9)	24.5 (18.0-31.7)	22.5 (17.9-28.4)

* range of values found in runs with single parameter changes

TABLE 42. Average Standard Error to Mean Ratios (in %) for Model Output: 40 Year Results for Seven Output Types at +10% from NS

OUTPUT TYPE	MEAN RATIO FOR ALL +10% CHANGES	RANGE
R.O.G. Mortality	15.6	13.6-23.6
White Oak Mortality	20.6	17.2-23.7
Root Graft Spread	19.4	14.4-24.6
Local Vector Spread	18.2	14.5-24.2
Background Mortality	9.0	6.1-10.6
Number of Pockets	14.5	11.2-20.7
Average Pocket Size	5.2	4.6-6.6

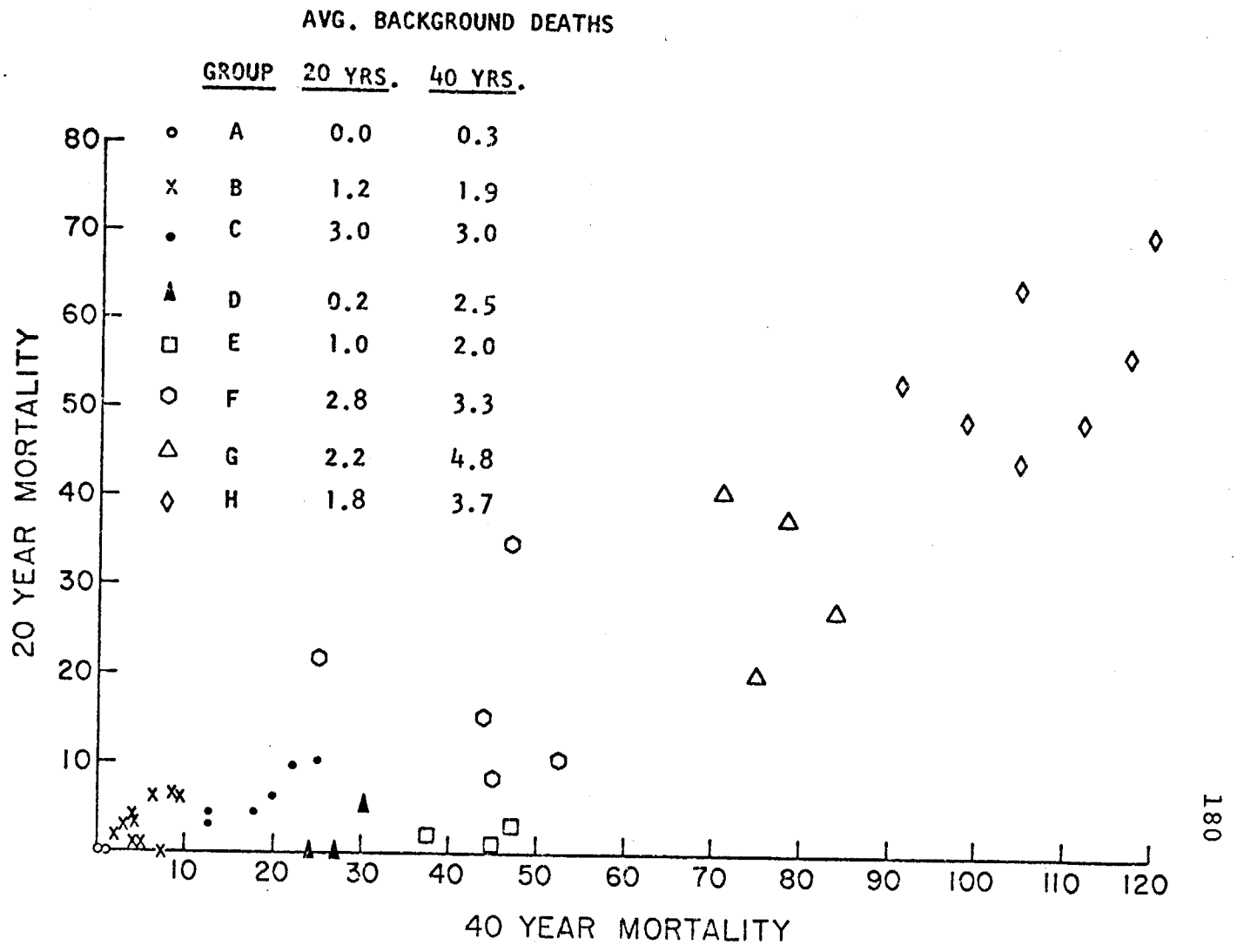
spread. White oak mortality is the most variable of the outputs examined.

In general, less favorable oak wilt spread conditions resulted in larger variances relative to the mean, although the absolute levels of damage were lower. The tremendous variability is a ubiquitous trait of model output and, it is argued, of the oak wilt system. The absence of oak wilt from many woodlots while others are subject to huge damage is a characteristic of the disease.

Now that the overall levels of variation in multiple runs with the same parameters have been presented, the nature of the variation may be considered in more detail. Fig. 6 shows mortality results for 40 runs at nominal conditions and 50% composition, with 20 year mortality plotted against 40 year mortality. The scatter shows that even knowing simulated 20 year mortality, it would be hard to predict what will happen in the next 20 years of the run.

From observation, there appears to be some clumping of results from different runs. The results from the 40 runs were divided into eight classes by inspection. For the lower part of Fig. 6, the average number of actual background spreads in the group was related to the relative position of the group in output space. (Note that the parameters were identical and that the scatter is a function of the stochastic nature of the model.) Thus,

FIG. 6. 20 and 40 Year Mortality, from 40 Runs with Nominal Conditions and 50% Composition

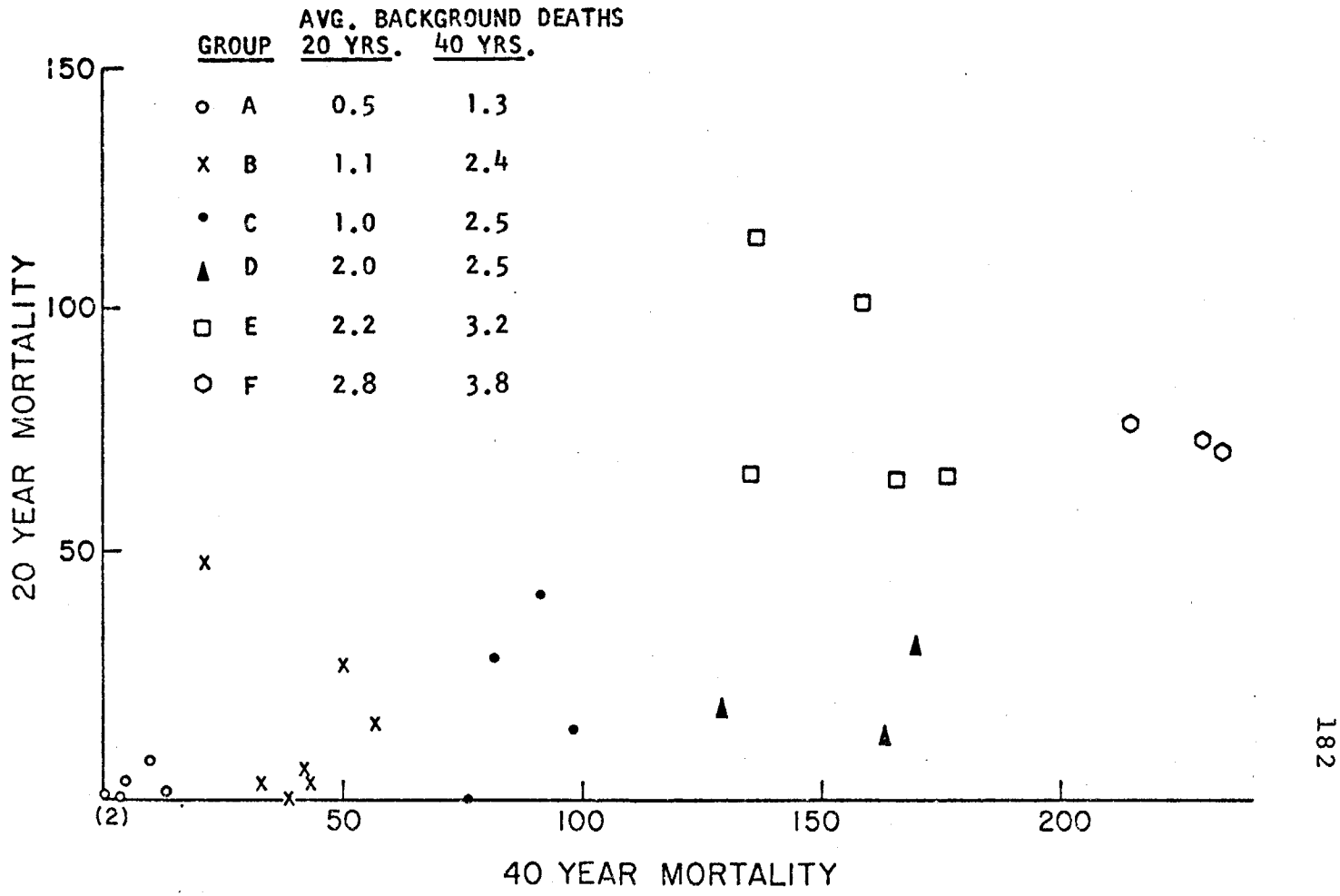


the number and timing (i.e. first or second 20 years) of introductions into the plot was important in determining the amount of damage. This relationship is not expressed in the right-hand side of the plot (groups C,F,G,H), where large differences in mortality (three-fold) result from similar numbers of introductions. This indicates that once the constraint of outside inoculum has been passed, differences in mortality result from other factors. The constraint is not overcome merely by the outside infections (see Group C), but also by subsequent spread of mortality within the plot, which does not always follow introduction.

Similar patterns were found for multiple runs at 65% (see Fig. 7) and 75% red oak group compositions. In each case, groups were fairly arbitrarily distinguished and the relationship of background infection to overall mortality was observed. In these more favorable conditions for root graft transmission, the effects of different levels of high background infection on total mortality (e.g. D vs. E vs. F, Fig. 7) were more evident than in the 50% case. However, individual runs still showed large variability in this relationship.

A similar plot for runs at 25% red oak group composition showed the same general relationship of background spread to mortality. However, there was less apparent

FIG. 7. 20 and 40 Year Mortality, from 30 Runs with Nominal Conditions and 65% Composition



grouping of output results. Apparently, background introduction is still very important, but subsequent mortality is not great enough to separate groups in such discrete fashion.

If the amount of background spread is important in causing discontinuities in output values, what is the effect of varying this constraint? An increase of 10% in BGRND, to 2.53, gave results for 30 runs seen in Fig. 8. There appears to be somewhat less distinct groupings in this case, as the constraint of low rates of introduction is relaxed. An even lower value of BGRND than the NS, one of 2.07, gave quite discontinuous results, as seen in Fig. 9. The most obvious discontinuity is found in the fact that 30% of all runs failed to show any mortality. The levels of background spread actually occurring in each run over 20 and 40 years are related to relative group position on the total mortality plot, with the exception of group D.

The main property of multiple runs with the same parameters is the high amount of variability, particularly for root graft spread and white oak mortality. The amount of background spread and its temporal location in the run is an important determinant of the system response, despite the fact that, of itself, background mortality is a minor component of all oak wilt-caused deaths. The behavior of

FIG. 8. 20 and 40 Year Mortality, from 30 Runs (NS, COMP=50) with BGRND=2.53

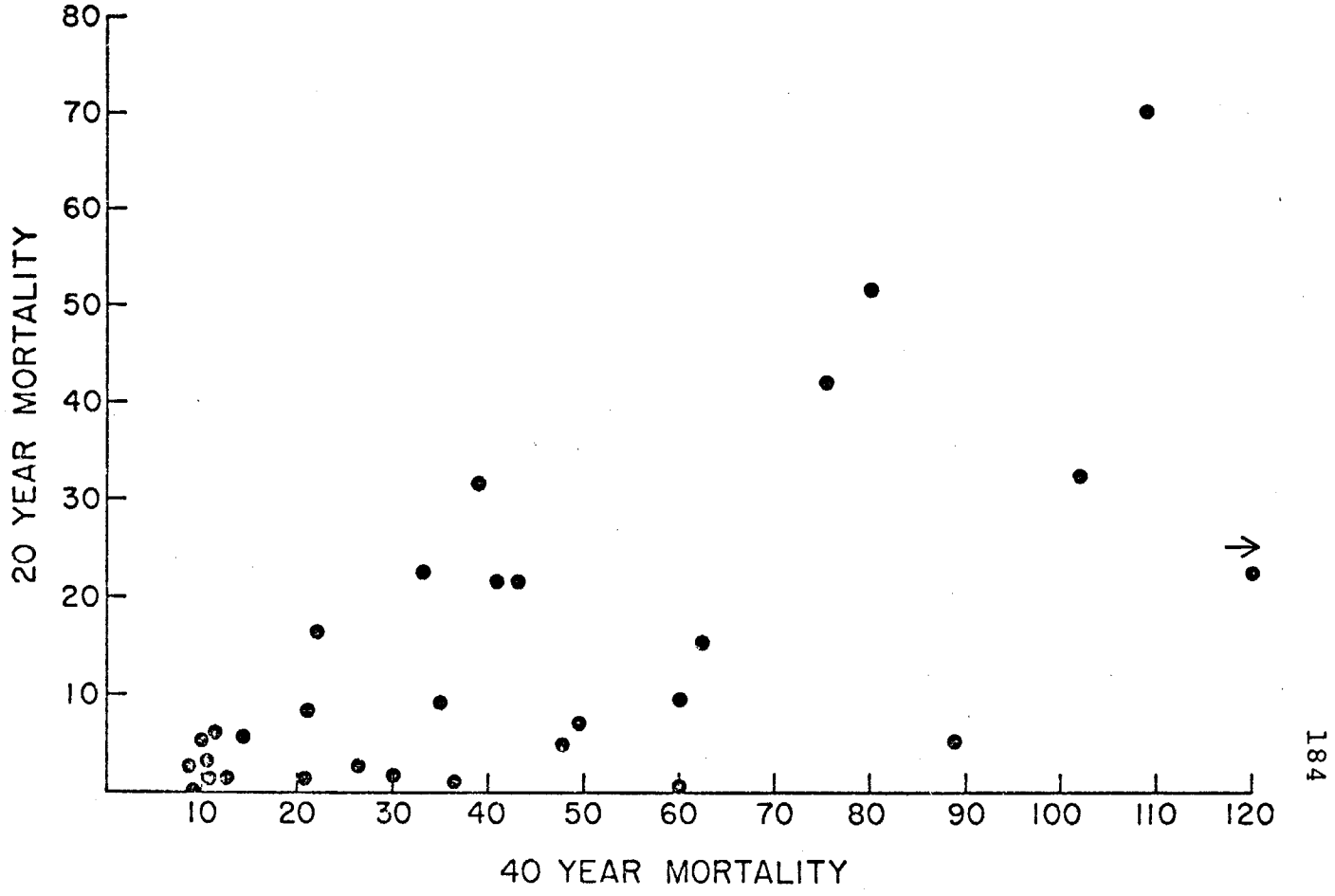
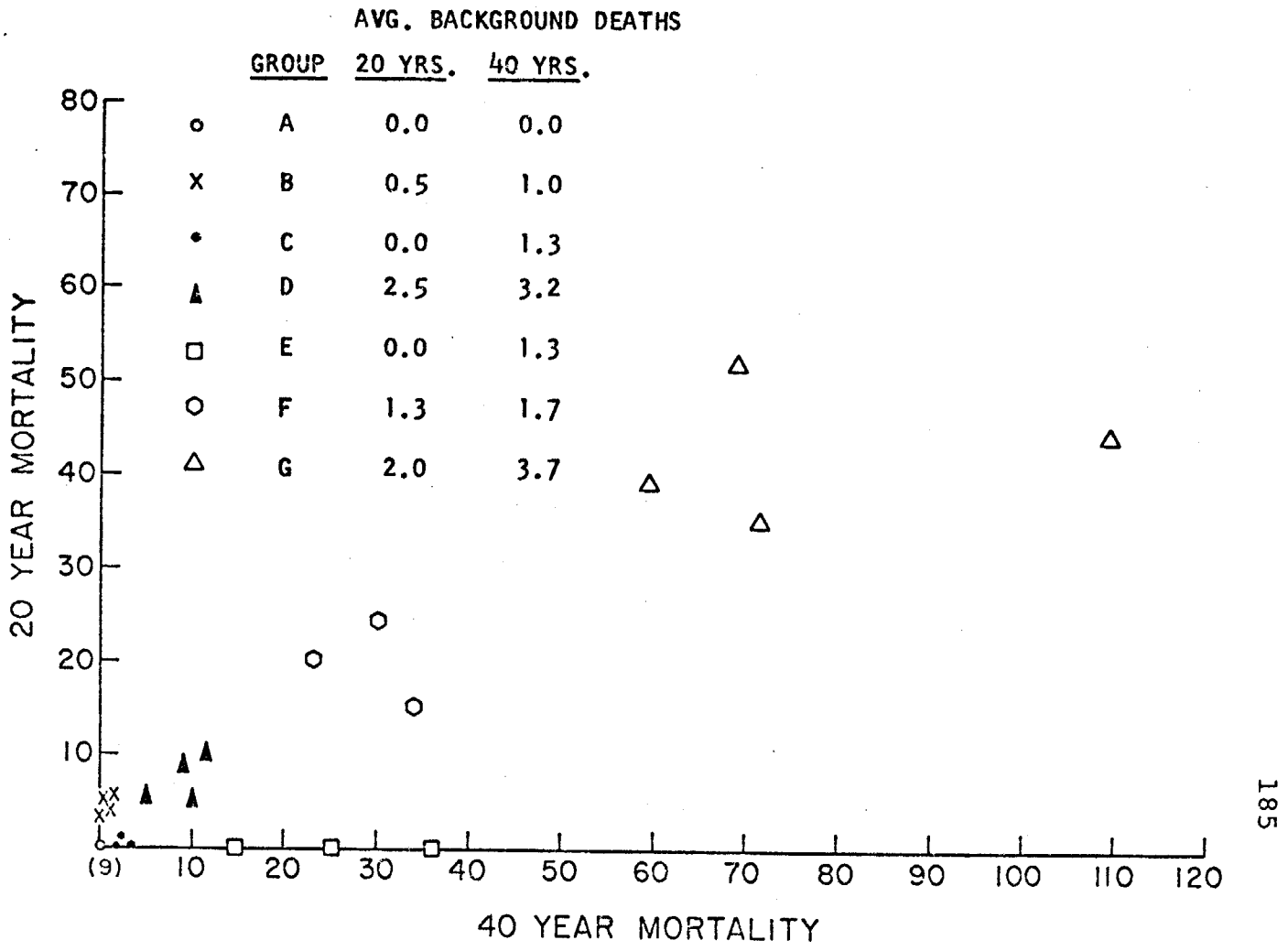


FIG. 9. 20 and 40 Year Mortality, from 30 Runs (NS, COMP=50) with BGRND=2.07



the system appears complex and varies as composition and amount of background spread changes. The next section will explore in a more formal way the causes of this behavior.

D. Sensitivity Analyses for the Effects of Single Parameter Changes on Model Outputs

1. Methods of Sensitivity Analyses

The sensitivity of model outputs to changes in parameter space will be explored with sensitivity analyses. The following output will be examined: total dead trees, dead red oak group trees, dead white oaks, % deaths that are red oak group, root graft mortality, local vector mortality (i.e. from within-plot inoculum), background mortality (vector spread from outside the plot), the % of deaths from root graft spread, the peak year, the mortality in the peak year, the number of pockets, and the mean pocket size.

The sensitivity analyses will consider changes in these output values resulting from given changes in the parameters of the model (Tables 37 and 38), one parameter at a time. The differences resulting from changes of +10% and -10% in each parameter from nominal simulations at 50% composition will be referred to as SA1. (Recall that the NS is over a range of composition values.) A more global sensitivity analysis will consider changes of ±50%

at 50% composition (SA2). Finally, a 25% change in both directions from the values of the NS at 65% red oak group composition will constitute SA3. Output after 40 years will be considered.

These are fairly crude analyses for two reasons. First, the model sensitivity is considered only around nominal conditions at 50% and 65% compositions. Sensitivity might be different in other regions of parameter space, say at 20% composition, or half the background inoculum, or twice the plot size. Secondly, only one factor is varied at a time. Possible interactions of several factors (e.g. density x composition, plot size x background) cannot be considered directly.

A more complete description of model sensitivity to changes in composition is presented, however.

2. Results of Sensitivity Analyses

Table 43 shows the results of SA1. The values in the tables represent differences between simulation results of +10% and -10% for the listed parameter from nominal values, with all other parameters as in the nominal simulation and composition at 50%. Each number is the difference in means of 30 runs each.

Total mortality is over twice as sensitive to one parameter as any other; that parameter is OVERFN. OVERFN

TABLE 43. Results of ± 10% Sensitivity Analysis at 50% Red Oak Group (R.O.G.) Composition (SA1)*

	PARAMETER VARIED**										
OUTPUT TYPE	OVER-FN	BGRND	RGPARM (1)	DEN	PLOT	COMP (1)	SIZEC (1)	SIZEV (1)	SIZEV (2)	RGPARM (2)	SIZEC (2)
Total Mortality	65.1§§	29.6	29.1	23.2	19.5	18.2	16.4	14.1	12.3	8.1	-0.9
R.O.G. Mortality	61.0	28.4	28.5	22.0	18.2	18.2	15.5	13.2	11.5	7.7	-1.0
White Oak Mortality	4.1	1.1	0.6	1.2	1.2	0.0	0.9	1.0	0.8	0.7	0.2
% R.O.G. Mortality	-3.8	1.0	2.8	-0.2	1.5	2.5	0.2	-0.4	0.5	1.5	-0.4
Root Graft	26.5	11.9	17.0	10.5	5.9	12.1	8.7	7.6	5.8	5.3	-0.3
Local Overland	35.2	14.5	12.1	12.3	10.6	6.6	7.6	6.9	6.5	3.0	-0.9
Background	3.4	3.2	-0.1	0.3	3.0	-0.5	0.1	0.2	0.0	-0.4	-0.3
% Root Graft	-1.6	1.6	14.0	2.0	-3.2	10.8	4.6	4.7	3.6	4.5	-0.2
Peak Year	2.8	3.3	1.0	4.2	-1.4	4.4	1.9	3.5	-5.1	-0.9	-0.2
Peak Number	5.5	2.7	2.3	2.4	2.2	1.0	1.7	0.5	1.2	0.3	-0.2
No. Pockets	26.4	15.0	8.6	8.2	11.5	4.5	4.7	5.2	5.2	3.5	-0.1
Average Pocket Size§	0.5	0.1	0.6	0.4	0.1	0.4	0.2	0.2	0.1	0.1	-0.7

* Values are differences in mean values for the various output resulting from 30 runs each for changes in a single parameter values of ± 10% from NS at 50% composition. Positive numbers indicate the + 10% set of runs had a higher mean value for the particular output. Numbers are in units of number per 10 acres in 40 years.

** See Tables 37 and 38 for definitions.

§ number of dead trees

§§ the three most important parameters for each output type are underlined

represents the average overland infection conditions for the run; it is the number of trees an average wilted tree of the red oak group will infect by vector spread. Each year of the simulation, the current year's equations for both background spread and local overland mortality are calculated using values drawn from a normal distribution with mean OVERFN (see Equations 8 and 10).

The greatest effects of increasing values of OVERFN are in favoring both types of vector spread (see Table 43). Within-plot overland spread is over twice as sensitive to changes in OVERFN as any other parameter. The sensitivity of root graft spread to such changes is considerably less, as is evidenced by the -1.6% for % root graft transmission. However, root graft transmission is still more sensitive to changes in this parameter than any other. This is powerful evidence for the dependency of root graft transmission on vector spread. Increases in OVERFN also increased white oak mortality greatly.

The next most important parameter is BGRND, or the average amount of inoculum coming from outside the plot. Since this parameter appears only once in the entire model (Equation 8), the results may seem surprising. Background spread is very sensitive to BGRND, as might be expected. Small increases in BGRND also increase local overland spread and, to a lesser extent, root graft transmission.

There is one other parameter that had a large impact on total mortality by directly affecting background spread: PLOT, the size of the simulated woodlot (see Table 43). Small changes (± 10%) varied background spread by about three trees per run. This, in turn, caused increases in other types of mortality. As shown in the last section, the timing of increased background spread is also crucial. The great sensitivity of almost all model output to the three parameters affecting background spread (see Equation 8) parallels results showing total mortality to be strongly related to the amounts and timing of actual background spread.

If such large changes can result from small changes in plot size, can the model be considered valid for different sized plots? Recall that BGRND was empirically fitted at PLOT=10. Here, BGRND has been left constant and plot size has been increased and decreased. In effect, the target area has been changed (for an island biogeographic analogy, see Brown and Kodric-Brown 1977). If the amount of mortality due to oak wilt is indeed limited by the amount of outside inoculum reaching the woodlot, then large woodlots should show greater and earlier damage by virtue of a larger number of total introductions, even though introductions/unit area are the same. Smaller woodlots may often show a delay in introductions, which will

constrain total mortality. This result seems highly probable and was predicted by the model, although its sensitivity to PLOT was rather great. One modification of OAKWLT could be an adjustment of outside introduction to be proportional to the border area of the plot, or (4 */$\overline{\text{PLOT}}$) for square woodlots.

The three parameters that influence overland spread have major effects in increasing the number of patches, but, with the exception of OVERFN, the average patch size is less affected.

Density and composition are of intermediate importance to total mortality (Table 43). These are the parameters that will most often be known for actual stands. DEN and COMP have the greatest effects on root graft spread, as can be seen by observing changes in % red oak group, root graft transmission, and % root graft spread. Local vector spread is also increased by feedback from increased root graft mortality. Both COMP and DEN result in later peaks (4.4 and 4.2 years later) when varied $\pm$10%, perhaps indicating that spread after 40 years would be quite significant. In fact, all parameters affecting root graft transmission directly (the other four are the parameters of the root graft transmission equation and parameters specifying red oak group tree sizes, see Tables 37 and 38) show much greater effects of the 10% changes in

the second 20 years.

The second most important parameter for root graft transmission, after OVERFN, is the slope of the root graft transmission equation, RGPARM(1). The predilection of this parameter to influence root graft spread preferentially is seen by values for changes in % red oak group and % root graft spread in SA1, which are changed most by this parameter. Again, this increased root graft spread also effects an increase in local overland spread. The intercept (RGPARM(2)) is somewhat less important than the slope in its effects on model output, but shows similar patterns. This agrees with the importance of tree density (the inverse of average inter-tree distance) which affects transmission probability through the slope.

Output is slightly less sensitive to red oak group tree size. Increases in both mean and variance of tree size (SIZEC(1) and SIZEV(1) respectively) result in major increases in root graft transmission and somewhat lower changes in within-plot vector deaths. The increased mortality due to $\pm$10% changes in the standard deviation of the red oak group size distribution is partly an artifact of the model. Larger standard deviations mean more of the lower tail of the normal distribution is in negative tree sizes. The effect of this is to increase actual average tree size, since new trees are picked to replace

the 'non-trees.' However, changes in standard deviations from 0.0 to 2.0, which should not be affected much by this artifact, also increased mortality significantly. Therefore, it appears that greater heterogeneity in tree sizes does favor more oak wilt mortality by root graft spread, in an analogous manner to heterogeneity in composition (see Nominal Simulation, Table 39). Essentially, a combination of favorable and unfavorable conditions in different parts of the forest leads to more mortality than the average conditions.

White oak parameters did not show consistent correlations with output sensitivities. However, the fairly high value for some changes in output in response to SA1 for SIZEV(2) reinforces the variability of the model. There is no mechanism in the model by which white oak size could exert such a major impact. The 50% analysis will indicate whether the attachment of significance to parameters that affected model output only slightly more than SIZEV(2) was advisable.

The results from the $\pm$50% sensitivity analysis, SA2, are shown in Table 44. It is now possible to dismiss the importance of SIZEV(2), since the trends of output in response to this variable are in the opposite direction of SA1, and are weak indeed.

The three parameters affecting overland spread most directly (OVERFN,BGRND,PLOT) did not result in any mortal-

TABLE 44. Results of ± 50% Sensitivity Analysis at 50% Red Oak Group (R.O.G.) Composition (SA2)*

	PARAMETER VARIED**										
OUTPUT TYPE	OVER-FN	BGRND	RGPARM (1)	DEN§§§	PLOT	COMP (1)	SIZEC (1)	SIZEV (1)	SIZEV (2)	RGPARM (2)	SIZEC (2)
Total Mortality	217.7§	115.4	127.1	---	100.9	148.4	66.4	45.8	-6.5	50.0	-1.4
R.O.G. Mortality	201.2	109.3	121.8	---	94.2	148.0	63.0	43.2	-5.7	48.4	-1.4
White Oak Mortality	16.5	6.0	5.4	---	6.6	0.4	3.3	1.6	-0.7	1.7	0
% R.O.G. Mortality	§§	§§	4.7	---	§§	17.0	1.3	0.4	1.0	3.3	-0.2
Root Graft	75.7	47.7	77.4	---	34.4	89.0	37.2	25.0	-2.5	28.6	-1.2
Local Overland	130.5	54.6	50.0	---	51.8	61.0	29.2	20.9	-4.2	21.6	-0.5
Background	11.5	13.1	-0.2	---	14.7	-0.6	0.0	-0.1	-0.3	-0.1	0.2
% Root Graft	§§	§§	43.0	---	§§	33.3	27.8	17.4	2.0	20.6	1.8
Peak Year	§§	§§	13.1	---	§§	10.0	5.2	10.1	2.4	5.4	0.4
Peak Number	22.9	9.5	12.7	---	8.8	12.6	5.7	4.04	-0.1	3.8	-0.6
No. Pockets	75.9	49.9	30.6	---	55.9	30.7	18.5	13.2	-2.5	13.2	-0.3
Average Pocket Size***	§§	§§	2.0	---	§§	2.2	1.0	0.7	-0.1	0.8	0.1

* Values are differences in mean values for the various output resulting from 30 runs each for changes in a single parameter values of ± 50% from NS at 50% composition. Positive numbers indicate the + 50% set of runs had a higher mean value for the particular output. Numbers are in units of number per 10 acres in 40 years.

** See Tables 37 and 38 for definitions.

*** number of dead trees

§ the three most important parameters for each output type are underlined

§§ could not be calculated because of zero mortality at -50%

§§§ could not be tested at +50% due to computer storage limitation.

ity when varied -50% from NS, and large increases at +50%. The lack of mortality is a necessary consequence of Equation 8. Since NS values of BGRND were specific to PLOT=10, decreasing the latter parameter to 5.0 may not be reasonable. Composition (COMP) was varied from 25% to 75% in SA2 and was the second most important parameter affecting total mortality. It was even more important than OVERFN in affecting root graft spread (see also % root graft, average pocket size).

Other variables showed similar sensitivities in SA2 to those found in SA1, although density could not be tested due to computer storage limitations. Note that a 50% increase in average red oak group size, without a decrease in density, may not be biologically reasonable.

Some other trends are evident from the first two sensitivity analyses. Absolute mortality in white oaks is highest in conditions favoring red oak group mortality. One of these factors is composition. The amount of white oak mortality in 75% red oak group stands was higher than in 75% white oak stands, although relative white oak mortality was higher in the latter.

Conditions favoring root graft mortality result in a much delayed average peak in mortality relative to less favorable conditions. It may then be more persistent in influences on subsequent mortality.

SA3, a 25% sensitivity analysis at 65% red oak group composition, gave results shown in Table 45. The overall patterns are similar to SA1 and SA2. However, there has been an across the board increase in the sensitivity of total mortality to factors affecting root graft transmission directly (e.g. RGPARM(1),COMP,SIZEC(1)). Output (in particular, spread by vector) was still most sensitive to OVERFN however. As a rule, one could conclude that when conditions are favorable for one type of spread (e.g. root graft favored at 65% composition), small changes in parameters influencing this spread would have profound impacts. However, the system still may be constrained by other factors (e.g. OVERFN).

The outcomes of the three sensitivity analyses are summarized in tabular form in Table 46.

3. Output Sensitivity to Composition

Fig. 10 and 11 examine the importance of changes in composition on oak wilt dynamics in more detail. In stands dominated by white oak, mortality is low and mostly by vector spread (Fig. 10). At compositions of 50% red oak group and up, there is a marked increase in total mortality, mostly resulting from increased root graft transmission. The relative increase in sensitivity of model output to changes in composition in SA2 and SA3 is

TABLE 45. Results of ± 25% Sensitivity Analysis at 65% Red Oak Group (R.O.G.) Composition (SA3)*

	PARAMETER VARIED**										
OUTPUT TYPE	OVER-FN	BGRND	RGPARM (1)	DEN	PLOT	COMP (1)	SIZEC (1)	SIZEV (1)	SIZEV (2)	RGPARM (2)	SIZEC (2)
Total Mortality	254.4***	170.9	182.1	103.1	148.8	147.6	120.4	60.1	-24.5	55.2	-19.8
R.O.G. Mortality	246.1	166.1	177.2	101.2	144.0	147.2	117.5	58.6	-23.6	54.4	-19.6
White Oak Mortality	9.4	4.8	4.8	2.8	4.8	0.5	2.9	1.5	- 1.1	0.8	- 0.2
% R.O.G. Mortality	3.7	4.7	-0.2	1.4	1.7	4.7	0.3	-0.1	3.0	0.7	- 0.5
Root Graft	117.2	86.3	117.1	59.5	69.8	87.9	74.0	39.6	-11.9	38.4	-13.3
Local Vector	120.5	77.8	65.6	44.0	70.8	59.9	46.7	21.4	-12.2	16.9	- 7.3
Background	6.9	6.8	- 0.7	0.5	8.2	- 0.3	- 0.2	-0.8	- 0.4	-0.1	0.7
% Root Graft	11.8	8.2	34.6	12.8	5.0	16.3	11.4	10.0	- 0.3	9.2	- 2.0
Peak Number	22.8	14.9	17.1	9.9	12.8	14.0	10.7	5.6	- 1.3	5.4	- 0.9
No. of Pockets	70.2	54.7	27.3	21.5	57.8	25.9	23.2	6.7	- 8.9	7.3	- 3.0

* Values are differences in mean values for the various output resulting from 30 runs each for changes in a single parameter values of ± 25% from NS at 65% composition. Positive numbers indicate the + 65% set of runs had a higher mean value for the particular output. Numbers are in units of numbers per 10 acre in 40 years.

** See Tables 37 and 38 for definitions

*** the three most important parameters for each output type are underlined

TABLE 46. Summary of Results of the Three Sensitivity Analyses

PARAMETER	MEANING	SENSITIVITY RANKING* SA1	SA2	SA3	BIGGEST IMPACT ON WHICH OUTPUT?
OVERFN	Average vector spread per oak wilt-killed tree	1	1	1	All types, but especially local vector spread, background spread, white oak mortality, number of pockets
BGRND	Average vector spread from outside the plot	2	4	3	Background spread, local overland spread
COMP(1)	% R.O.G. composition	6	2	5	Root graft spread, delay of peak year
DEN	Total oak density	4	**	7	Root graft spread, delay of peak year
PLOT	Plot size	5	5	4	Background spread, number of pockets
RGPARM(1)	Slope of root graft transmission equation	3	3	2	Root graft spread, average pocket size, delay of peak year
RGPARM(2)	Intercept of this equation	10	7	9	Root graft spread
SIZEC(1)	Mean R.O.G. tree size	7	6	6	Root graft spread
SIZEV(1)	Variance associated with SIZEC(1)	8	8	8	Root graft spread
SIZEC(2)	Mean White Oak tree size	11	9	10	Not consistent
SIZEV(2)	Variance associated with SIZEC(2)	9	10	11	Not consistent

* One is highest ranking. Rankings denote relative sensitivity of total mortality to parameter changes.

** Was not tested due to storage limitation

FIG. 10. Effect of Changing Composition on Simulated 40 Year Mortality
(20 runs for each point)

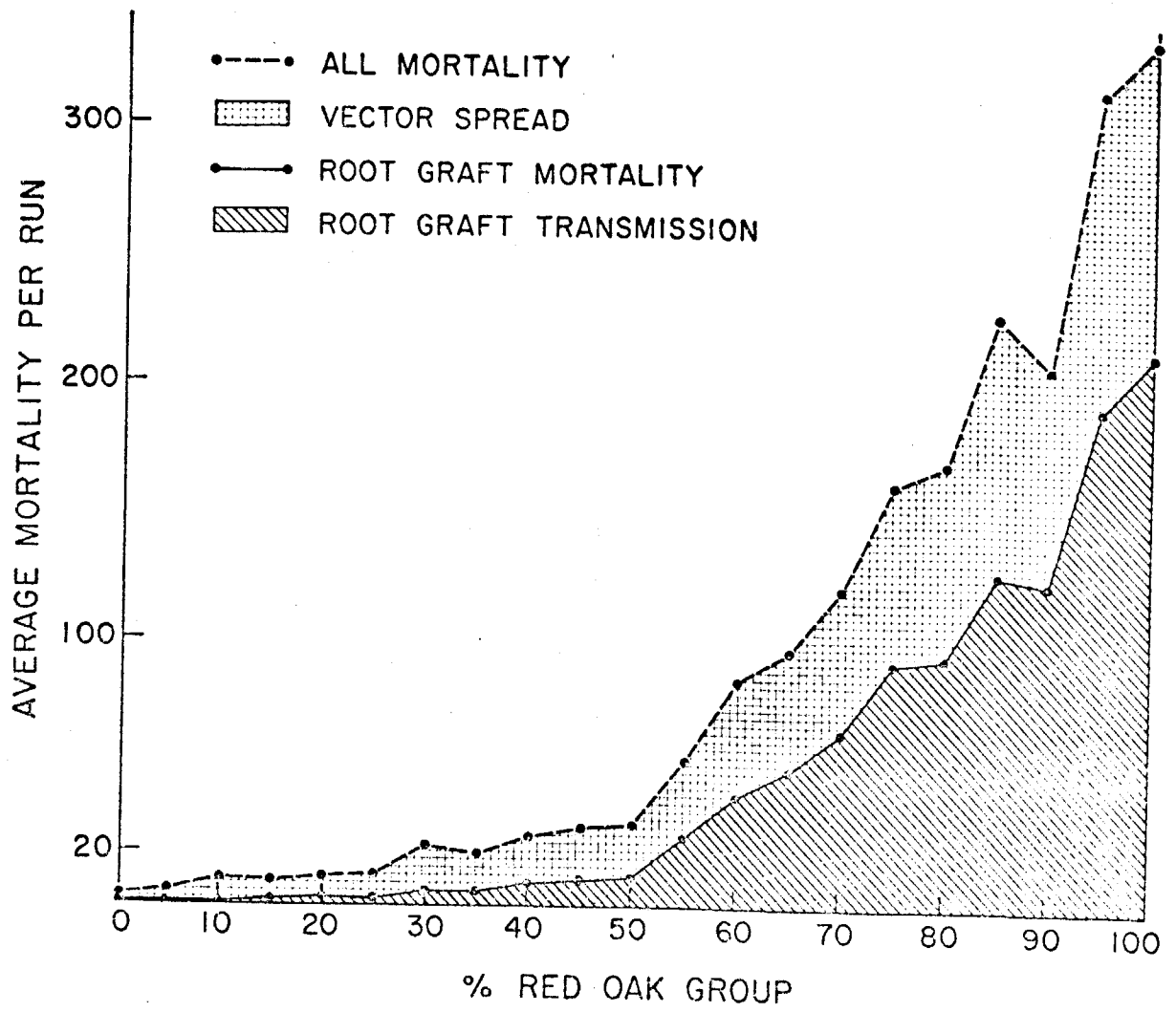
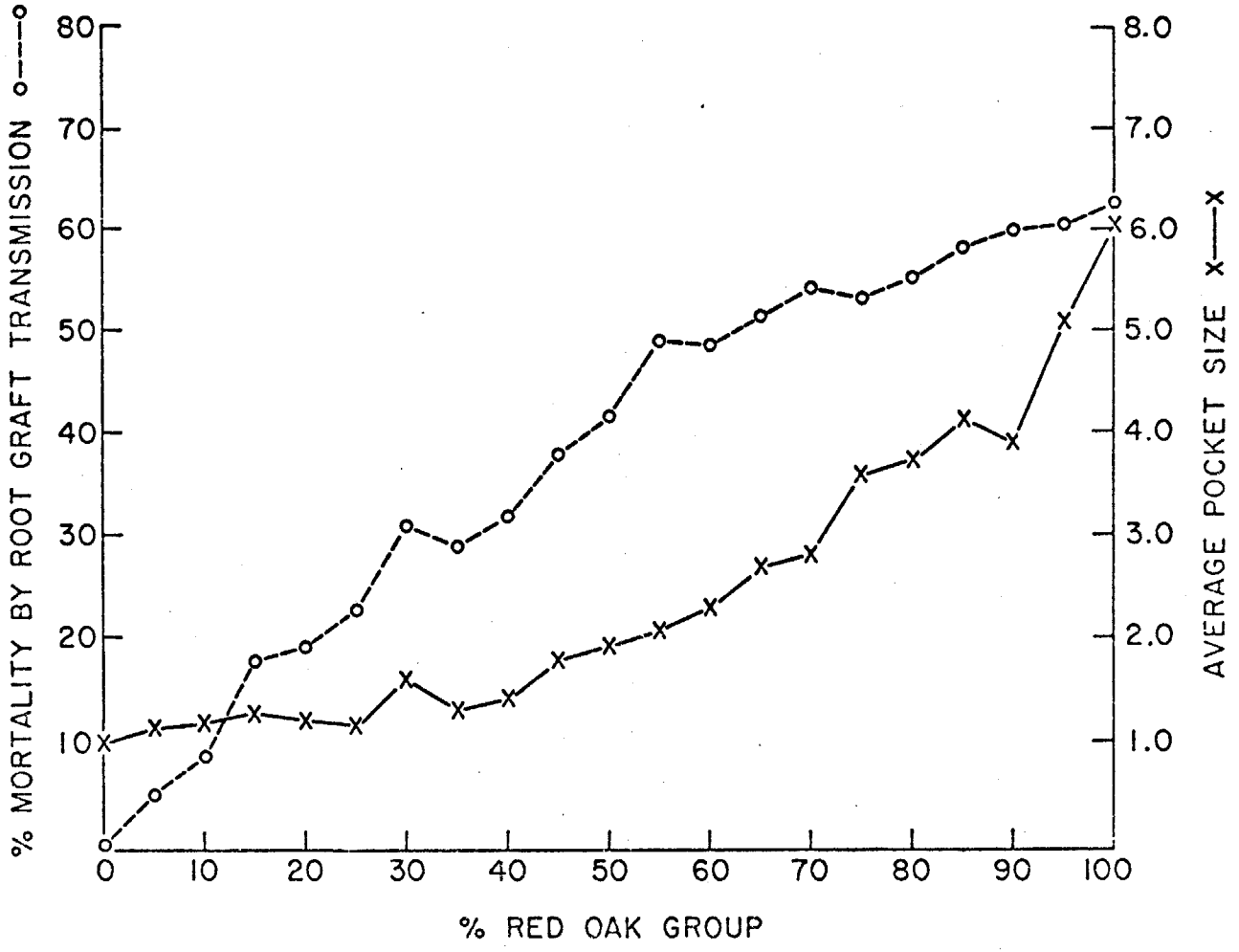


FIG. 11. Effect of Changing Composition on Simulated % Root Graft Transmission and Average Pocket Size (20 runs for each point)



easily shown by the increased slope to the right of 50% in Fig. 10. Also, the necessity of fitting the NS to a full range of composition values (see Section VIII A) is emphasized, since the damage at 50% composition in no way represents the average conditions over a range of compositions found at Sinnissippi.

Table 11 shows the effect of composition of the two other types of output: average pocket size and % mortality from root graft transmission. In these cases, there is no breakpoint in output with changing composition. Both types increase regularly with increasing amounts of red oaks. Fig. 11 allows a tentative answer to why pocket size is smaller in the East. Typically, density of oaks is somewhat lower, and this corresponds to a lower composition at constant density (left half of Fig. 10). With root grafting parameters from the Midwest, the average pocket size is less than 2.0 (typical outside of the Midwest, Jones and Bretz 1958, True and Gillespie 1961, Rexrode and Frame 1974, see Table 18), for densities up to 75 red oak group trees per acre. Due to greater species richness, many forests in the East may have these densities (e.g. Keever 1973, Skeen 1973). Thus, the simulation results show that small pocket sizes may be merely the result of decreased red oak density and not necessarily due to lower rates of root grafting.

The very regular changes in the last two figures might seem anomolous considering the extreme run to run variation in model output (see Section VIII C). This indicates that the high variability is not merely a result of an inadequate 'sample,' i.e. an insufficient number of runs. The number of runs is sufficient to produce these smooth trends. Rather, the construction of the system imparts high variation to the output. In the next section, I will make general arguments as to why this is so.

IX. DISCUSSION

A. Characterizing a Disease System

The emphasis in this paper has been on analyzing the characteristics of a disease caused disturbance, oak wilt. Although the emphasis has been on the specific attributes of oak wilt spread, a general description of this phenomenon should point to pertinent questions that might address other disturbances.

What are the boundary conditions for persistance of the oak wilt system? One set of such conditions permits persistence of the disease in the woodlot or area. The sensitivity analysis pointed out that factors affecting overland spread, OVERFN and BGRND, are most important in specifying system response (with respect to changes from NS). In fact, a graphical argument showed that much of the system response could be predicted from the number of introductions into a woodlot. The -50% sensitivity analysis treatment for three parameters (OVERFN, BGRND, and PLOT) resulted in no mortality. Thus, one set of boundary conditions, given other values as in the nominal simulation, was OVERFN at about 0.289 vector infections per previous oak wilt death, BGRND at about 2.07 (or 0.026 introductions per acre per year), and PLOT of five acres.

Other factors may be limiting given specific levels of these factors affecting overland spread. Also, on a regional level, BGRND (the rate of inoculum coming from outside the woodlot) might be considered a variable that can be affected by the amount of oak wilt in the area, instead of varying randomly. Likewise, low levels of oak wilt should affect OVERFN (average vector spread efficacy) beyond simple weather fluctuations. For example, population fluxes of vectors could result from changes in the availability of oak wilt inoculum or of oaks themselves.

At this point, there is little solid information on the boundary conditions influencing long-term oak persistence. Classical host-parasite models that show density dependence often show global stability, especially when time lags are built in. There is every reason to believe that the oak wilt system is density dependent (especially in the red oak group, Fig. 10) on both a local and regional scale. Time lags would be expected to arise due to the spatial partitioning of the system. This partitioning is two-fold: fragmentation of living oaks into woodlots and the patchy nature of disease damage within woodlots. The later peak for root graft transmission, despite its high frequency, implies another time lag. Thus, general considerations imply that the boundary conditions for oak wilt persistence on a local level (as explored by

sensitivity analyses) are different from regional levels, and that the system is probably globally stable in the sense that neither oaks nor oak wilt will go extinct.

This almost trivial conclusion does not mean that the system is necessarily locally stable, having equilibrial conditions from which little numerical fluctuation in oaks and oak wilt occurs. Again, we have not specified community response (i.e. ingrowth into pockets), although this will be discussed in the next section (IX B). The division of output into moderately discrete classes hinted that there might be preferred levels of damage, but these will vary with particular conditions (Figs. 6-9). In particular, discontinuity of mortality levels appears to be more pronounced when damage is low (Fig. 9). Some stability is implied by the constant values of vector mortality over time found in Sinnissippi (Table 7). Nevertheless, the absence of oscillation subsumes tremendous variability in smaller blocks.

Figs. 6-9 give an indication of the somewhat discontinuous nature of the system response, which is supported by anecdotal evidence. In addition, the variation in output from multiple runs with the same starting conditions is notable (Tables 41 and 42); again this parallels anecdotal evidence. Why should this be so?

Consider an oak woodlot. If neighboring woodlots

contain oak wilt inoculum, trees in this woodlot may become infected. However, it was shown that vector spread probably occurs mostly over short distances, and thus the rate of introduction is low. There was no way of directly partitioning the contribution of other areas from local inoculum production, but the model simulations for 10 acres showed the best fit to Sinnissippi data when the rate of introduction was low, on the order of six introductions/10 acres/20 years. It was also argued that this rate will probably decrease in larger plots, as the number of introductions becomes more and more a function of border length of the woodlots and not area.

Once introduction is effected, three courses are possible: 1) there can be no additional spread; 2) there can be local inoculum production; 3) there can be root graft transmission. All three are common results in model simulations. When there is local inoculum production, more new pockets will be started, and the probability of additional inoculum production within the woodlot is increased. In addition, there will be oak wilt in more than one area of the woodlot, and the possibilities for root graft spread are increased. With more root graft transmission, there is more possibility of additional inoculum production. Thus, there is positive feedback between both components of within-plot spread. On a geographical level,

greater within-plot spread will increase overall background inoculum levels.

Since the background spread will usually be of the lowest frequency, due to the local nature of vector transmission, the result is high variability in total mortality between different woodlots, facilitating separation into typical classes of damage. Both these phenomena and the nature of feedbacks were evident in the sensitivity analyses (Tables 43-45).

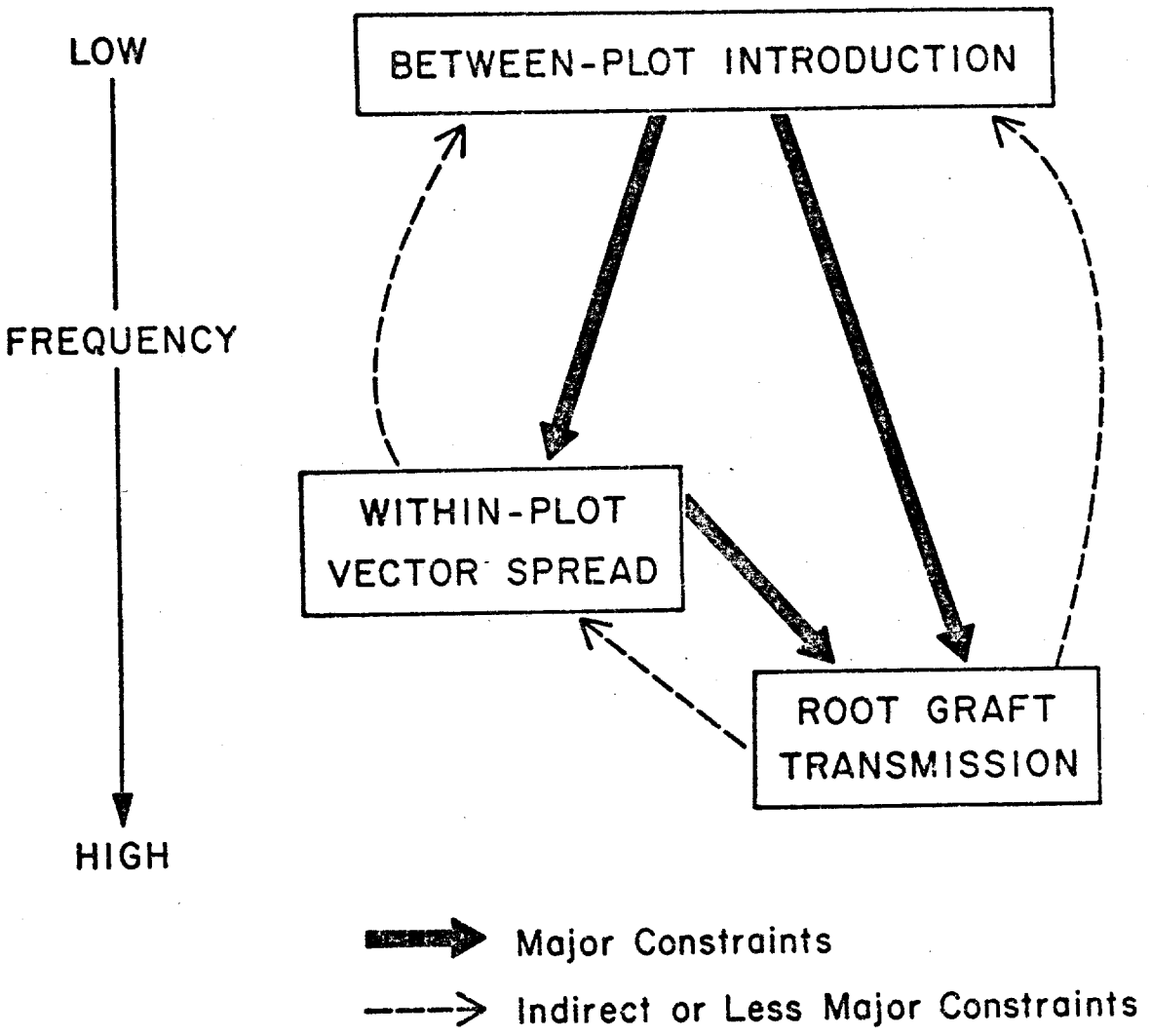
I visualize a conceptual model of the oak wilt system as being defined by constraints in frequency. Since disease can only spread in a stand after introduction, low frequency introduction of disease into woodlots constrains its further development. The relaxation of constraint requires more introductions (associated with a higher BGRND), as well as subsequent within-plot spread. Fig. 8 showed that when the average rate of introduction is increased, the system becomes less variable and more continuous. After enough introductions, the higher frequency components of local vector spread and root graft transmission take over. Eventually, however, the system must be forced by additional introductions if oak wilt is to persist. It should be emphasized that the discussion is in terms of frequency of certain types of spread, which is not related to the real time of actual tree to tree

spread.

Simon (1973) noted that the reaction of a system to low frequency phenomena will be so slow that they are treated by the system as constants that function as constraints for higher frequency processes. In the oak wilt system, typical levels of background infection may be seen as constraining system behavior. What controls the integrative behavior of the system is the combination of constraints on all levels (Pattee 1973). Once the background spread is high enough, factors affecting other types of spread become more important in influencing system behavior (see SA 2, Table 46). The raising of background levels may be seen as a change in the pattern of constraint.

Figure 12 shows diagrammatically the position of the three types of spread in frequency space, and the relative strength of the constraints. Specific conditions will alter actual values and thus change the integrative system response (Pattee 1973). For example, the constraint of local vector spread on root graft spread is presumably related to the necessity of bypassing local concentrations of non-susceptible species. This is considerably less in forests dominated by the red oak group (compare Tables 43 and 45). In Fig. 12, an increase in the density of the red oak group would be represented by a downward displacement of the root graft spread compartment.

FIG. 12. Conceptual Model of Oak Wilt Dynamics in Frequency Space (see text)*



*-Position of types of spread in frequency space will vary with conditions. For example, in dense red oak group stands, the frequency of root graft transmission may be higher, thus relieving the constraint from between-plot introductions, but making that from within-plot vector spread more important. As an epidemic proceeds, the less major and indirect interactions may change the positions of the different spreads in frequency space.

The description of the oak wilt process in terms of a series of constraints is fundamentally simpler than the OAKWLT simulation model and is certainly more coherent and concise than a posteri field-derived explanations of disease patterns. Most of the descriptions of oak wilt that even attempt explanation are anecdotal. Such accounts suffer from trying to make generalizations from too few observations of the typically rich and complex behavior.

That the behavior of the system is complex is confirmed by the model results that show wide variability (Figs. 6-9) even under the patently unrealistic assumptions of constant parameter values over time. There are indications that oak wilt mortality patterns tend to concentrate in particular areas of output space, i.e. the behavior is discontinuous. In the parlance of catastrophe theory, oak wilt mortality patterns exhibit a cusp phenomenon (Zahler and Sussman 1977). Whether a cusp is necessarily crossed in a given woodlot is largely stochastic, but distributions of typical levels of damage can be predicted statistically, based on characteristics embodied in model parameters. Note that only mortality is being explicitly considered here. Characteristics of oak regrowth may change the exact patterns of the fungus-tree system. Since regrowth occurs at a longer time scale than local patterns of oak wilt damage, it is expected that the

complex behavior of the system will be preserved by the interaction of differently paced processes. This occurs in the spruce budworm system analyzed by Ludwig et al. (1978).

How can the simple model presented in Fig. 12 be expected to generate this complexity? Allen and Starr (1979) argue that richness of behavior can be generated by implicit hierarchical structure, with emerging cycling times of the various compartments being of paramount importance. Complex systems are likely to be hierarchical because the generation of stable intermediate forms facilitates evolution (Simon 1962). The results of such system organization are 'complex systems (that) are not accumulations of large number(s) of specific behaviors, but ... are instead (slow behaving) very general patterns of constraint imposed on faster behaving, more specific constraints' (Allen and Starr 1979).

I have argued that infection from outside the woodlot is an important constraint. Allen and Starr suggest that two parts in a system that have only somewhat different cycling times will generate complex and difficult to predict behavior. For oak wilt spread, the rich system behavior is generated by the two mutually reinforcing types of spread: local vector spread and root graft transmission. When these two occur at different but similar

frequencies, the behavior of the system is most complex. It is expected that when one occurs at a much higher frequency, the other becomes more of a constraint, and the diversity of system behavior is necessarily reduced (as observed at the scale of model output). The model results so far are not sufficient to confirm this.

Despite the complexities, the OAKWLT model gives reasonable system response, because oak wilt dynamics are 'nearly decomposable' (Simon 1962), meaning interactions between components are weaker than within components. This simplification allowed OAKWLT to be formed from description of the component spread processes.

Is it reasonable to expect other diseases to exhibit discontinuous and complex behavior? One way for this to happen is to have a suitable separation in frequency space of the various components of spread. If separation is too short, there will be a lack of sufficient constraint by the low frequency component. This would happen if different types of spread were at the same frequency.

One feature of diseases that would result in discontinuous and unpredictable behavior would be the existence of two types of spread occurring at different frequencies. Another possibility is the fragmentation of the host, if dispersal of the disease is poor. Chestnut blight formed continuous damage (no cusps here) because of easy disper-

sal of the fungus, a more or less continuous forest area, and the re-sprouting behavior of chestnut. Dutch elm disease damage is somewhat discontinuous (Richardson and Cares 1976), but probably not so much as oak wilt, despite two mechanisms of spread, because its insect vectors are more efficient than those of oak wilt.

B. Oak Wilt and the Dynamics of Midwestern Oak Forests

1. The Nature of Oaks and S. Wisconsin Oak Forests

Curtis (1959) and his associates have provided a thorough description of forests in southern Wisconsin, many of which are typical of other areas in the Midwest. Curtis and McIntosh (1951) arranged the upland forests of this region along a unidimensional axis, or continuum, which placed stands between hypothetical end-points of 100% sugar maple and 100% bur oak (see Appendix 1 for species list). Peet and Loucks (1977) took environmental measurements in upland stands and showed that the continuum is actually a diagonal through a two dimensional environmental hyperspace having as its axes moisture-nutrients and dynamics gradients.

Forests at the mesic, late-successional sugar maple end of the continuum contain red oak as the only important oak. Curtis (1959) and others considered red oak stands

to result from large scale fire, or perhaps from former northern hardwood associations. Red oak does not persist in these stands, being replaced by elm, basswood, and sugar maple. Mesic forests have probably existed continuously since before settlement in areas that were protected from fire (Harper 1963, Curtis 1959).

More xeric upland forests in southern Wisconsin are dominated by oaks. Curtis (1959) divided these into dry forests (dominated by black oak, white oak, bur oak, black cherry, red oak, and Hill's oak) and dry-mesic forests (dominated by red oak, white oak, basswood, and sugar maple). It is in these forests that oak wilt has played a major role.

Surveyor's data indicate that 83% of southern Wisconsin was maintained as prairie or oak savanna by fires before settlement by white man (Curtis 1959, Barnes 1974). Tree vegetation in savannas was dominated by scattered fire-resistant bur oaks and low-growing grubs of black and white oak. With the cessation of fire, there was a huge increase in stem density by sprouting from grubs, particularly from black oaks , and seeding, that changed the open savanna to a dense, brushy woodland (see descriptions in Curtis 1959). Xeric forests have increased 40% since presettlement times and most are presently in blocks of 40 acres or less. Cottam (1949) studied what

is now classified as dry-mesic forest and showed that about 95% of all oaks were of grub origin.

Therefore, most of the drier oak forests are less than 140 years old, and their future is open to question. Intensive land use in this time has had major effects on these forests (Curtis 1956). Farming has fragmented woodlots (Curtis 1956), and heavy seeded species such as sugar maple cannot reach many isolated woodlots. Island biogeographic theory (MacArthur and Wilson 1967) predicts that species richness should equilibrate at lower values in individual woodlots than if forests were continuous. Small woodlot size also creates more edge and thus more pioneer-like conditions (Auclair and Cottam 1971). Intense grazing and cutting has affected many characteristics of the woodlots. Grazing has hurt tree reproduction of many species, although oaks are generally considered unpalatable (Carvell and Tryon 1961, Arend and Scholz 1969). Many oak forests in parts of the Midwest and East date from cutting and are primarily coppice stands (Arend and Scholz 1969, Curtis 1956). Among the best sprouting species are black oak, white oak, and red oak (Ebinger 1973, Johnson 1975, Rogers 1959). Finally, diseases such as oak wilt and dutch elm disease have been very important in removing oaks and elms respectively.

Of all the species in oak forests in southern Wiscon-

sin, bur oak and Hill's oak are the most intolerant and rarely will reproduce under any canopy. They are likely to be found on steep slopes and on sandy soils where light is able to penetrate to the ground. Hill's oak hybridizes with black oak, which is variously described as very intolerant or moderately tolerant (Curtis 1959, Peet and Loucks 1977). Curtis (1959) placed black oak on a subterminal part of the continuum, but later workers (Goff and Cottam 1967, Goff and Zedler 1968, Peet and Loucks 1977) moved it to the xeric terminus. The last named authors cited several examples of black oak and/or bur oak stands that seemed to be stable.

The slower growing white oak is a common associate of black oak in dry and dry-mesic forests. Most investigators rate white oak as the more tolerant of the two. Oak reproduction by seed for both species is highly variable (Gysel and Arend 1953, Curtis 1959, Carvell and Tryon 1961, Olson and Boyce 1971, Skeen 1974) and, even when abundant reproduction exists, it may be shorter in height than many other plants (Arend and Scholz 1969) Seed reproduction is lowest under mature, closed canopies in non-severe sites, indicating that both species are fairly intolerant (Carvell and Tryon 1961). In addition, both species germinate best on litter of shallow depth (Minckler and Jensen 1959), although there is disagreement as to whether deep litter

layers decrease germination strongly. Arend and Scholz (1969) recommended various types of cutting to improve the chances of reproduction by seed, sprout, and seedling sprout. Carvell and Tryon (1961) also recommended cutting for enhanced seed reproduction and found the best oak germination in disturbed areas (fire, logging, grazing) and on dry exposures on midslopes.

Black oak also hybridizes with red oak. Curtis gave the latter species a climax adaptation number of 5.5, which Peet and Loucks (1977) decomposed to a low dynamics value (a pioneer) and a fairly low moisture-nutrients value (mesic). The shade tolerance of red oak has variously been considered as fairly high (Keever 1973, Curtis 1959) and fairly low (Carvell and Tryon 1961, Cove 1971, Phares 1971).

Two gap phase species, black cherry and shagbark hickory, play interesting roles in oak forest dynamics. The former species has received some study by Auclair and Cottam (1971, 1973, Auclair 1975). Its widespread dispersal, seed dormancy (perhaps similar to pin cherry in New England, see Marks 1974), and flexible seed-bed requirements allow it to colonize many isolated woodlots. A seven to eight year cycle of dieback and sprouting of suppressed individuals allows cherry to take advantage of gaps with younger, less shade-conditioned stems (Auclair 1975).

Cherry is most common in Wisconsin in small size classes in dry-mesic forests (Curtis 1959, Auclair and Cottam 1971), while in Pennsylvania it may be a canopy dominant on mesic sites (Hough 1960). Auclair (1975) showed that cherry vigor and height growth were associated with gaps of dead trees. High mortality of cherry, sprouting, and low vigor occurred under full canopies. This type of behavior allows cherry to persist in shady conditions, leading Auclair and Cottam (1971) to consider cherry as shade tolerant under the specific conditions of the forest, although physiologically it is fairly intolerant (Hough 1960). Peet and Loucks (1977) drew similar conclusions from size class data.

Various other species are able to replace oaks and cherry under closed canopy conditions, especially in the more mesic stands. However, the most tolerant trees, sugar maple and basswood, are poor dispersers and may not be present in areas in which they might persist (Auclair and Cottam 1971, Ward 1956).

Shrubs are an important component of dry-mesic forests (Curtis 1959, Goff and Zedler 1968, Auclair and Cottam 1971). Auclair (1975) showed that they were found in similar gaps to cherry, and Auclair and Cottam (1971) presented data showing a large increase in shrub importance in disturbed forests (cutting and grazing). Shrub cover

may decrease light on the forest floor to such an extent as to preclude successful tree reproduction (Auclair and Cottam 1971). Clearcutting and grazing (Arend and Scholz 1969, Auclair and Cottam 1971) may favor oak reproduction over shrub growth.

2. Species Replacement

What are possible species replacement patterns in southern Wisconsin oak forests, given the species autecologies and the forest history? Curtis (1959) hypothesized a species replacement pattern from bur to black to white oak. Auclair and Cottam (1971) disagreed, finding no directional trends in species replacement. They argued that all of the oaks were essentially pioneers.

Later species replacement is also complicated. McIntosh (1957), using association measures, outlined two proposed species replacement sequences starting at white oak and proceeding to a sugar maple-dominated climax. However, in many southern Wisconsin forests, there is little evidence for succession to sugar maple (Ward 1956, Curtis 1959). Various disturbances may allow re-establishment of oaks, cherry, and shagbark hickory, even in stands mesic enough to otherwise allow invasion by sugar maple. In addition, the heavy seeded sugar maple and basswood may not be good dispersers in the fragmented woodlot system

(Auclair and Cottam 1971).

Peet and Loucks (1977) studied undisturbed forests in southern Wisconsin and summarized the interaction of moisture and species replacement in an elegant diagram, recopied as Fig. 13. Basically, more xeric stands have shortened successional sequences and may show persistence of bur and black oak. They hypothesized that dry-mesic stands will tend toward a steady state of cherry, hickory, and white oak, which will be maintained by gap phase replacement. There is evidence that white oak may be a climax species in areas of the East (Keever 1973, Oosting 1942), with periodic canopy thinning resulting in even-aged recruitment of white oaks. Indeed, white oak is more tolerant when young (Core 1971). Forests in the East are somewhat older and perhaps Wisconsin forests will tend the same way. Species replacement in mesic forests is thought to result in replacement of oaks by more tolerant species. The treatment of Peet and Loucks considers the fairly hypothetical situation of no disturbance and does not discuss the effects of shrub reactions to gaps.

3. Species Replacement with Disturbances

The effects of cutting, grazing, and disease on the species replacement patterns outlined above are largely unknown and must be hypothesized from the autecological

FIG. 13. Model Showing Location of Southern Wisconsin Forest Stands on Dynamics and Moisture-Nutrients Gradients. From Peet and Loucks (1977, Fig. 3)

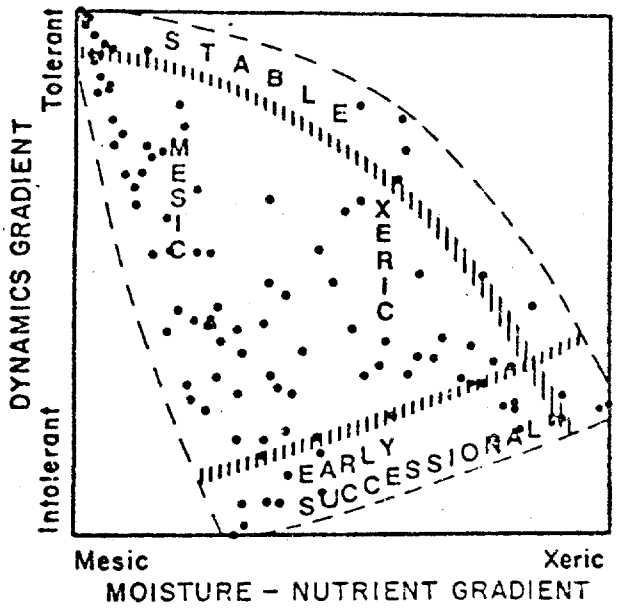


FIG. 3. A gradient analytic representation of southern Wisconsin forests. Each dot represents the location of one of four size strata from each of the thirty sampled stands. The abscissa is a moisture-nutrients gradient derived from soil sand content. The fertile, mesic stands are on the left and the sterile, xeric stands on the right. The ordinate represents the dynamics gradient which is closely related to stand successional status. The dashed line suggests the normal limits to stand composition. Stands located near the upper dashed line are approaching stable composition.

information already outlined.

Cutting will favor advance reproduction in many cases, which on dry-mesic sites may be cherry and hickory, and on mesic sites sugar maple and basswood. However, clear-cutting is used to favor oaks, because of their prodigious stump sprouting (Arend and Scholz 1969). Partial cuttings may also favor oaks in some cases, with the larger cuts presumably doing so to a greater extent.

Grazing may also favor oaks, especially if overgrazing creates a sod (Cawley 1960). Shrubs also increase in disturbed areas (Auclair and Cottam 1971), and many of the thorny forms are certainly unpalatable. Auclair and Cottam (1971) cite grazing and drought as factors favoring cherry invasion. Very heavy grazing may eliminate all tree reproduction (Curtis 1956).

Oak wilt persists in the vascular systems of killed trees for several years, precluding sprouting from wilt-killed oaks. There is also little soil disturbance resulting from oak wilt mortality. The extent of growth by white oaks left in an oak wilt gap is unknown, but presumably such gaps would favor the species. Auclair (1975) has demonstrated the ability of the cyclically sprouting cherry to fill small gaps. Although he did not substantiate the cause of gaps, he presumed that they were oak wilt openings. Shrubs also were favored by such gaps

(Auclair 1975).

Thus, if cherry, hickory, shrubs, and to some extent white oak are present in oak wilt gaps, it is unlikely that black or red oaks could persist as seedlings. The abundances of cherry and shrubs were shown to be lower under two sets of conditions: very dry sites and high overstory basal area (Auclair and Cottam 1971, 1973, Auclair 1975). In these cases, oak wilt openings that are large enough may favor black oak. Grazing and cutting along with oak wilt may also give black oak or red oak a chance to reproduce by seed, especially if there is some soil disturbance.

One point to keep in mind is that oaks can be considered as fire adapted species and are not necessarily adapted well to other disturbances (Auclair and Cottam 1971). Peet and Loucks (1977) point out that the dry-mesic forests in Wisconsin have not been around long, and there are no species adapted for growth in steady state xeric or dry-mesic forests in which fire has been excluded.

In the presettlement Midwest, these species mainly existed as grubs and rarely grew to adult size. Thus, the lack of resistance of adult trees to oak wilt may be a result of selection rarely acting on adult trees. This adaptive explanation might be used to explain the partial resistance of bur oak, which more often grew to tree size in presettlement savannas. However, it fails to provide any

explanation for the differences in resistance between white oaks and the red oak group, especially given their joint historical importance in mature forests of the East.

It may also be true that oak wilt has not had a long history as a pathogen, although information previous to the 1940's would be sketchy even if it had. The efficient local spread of the fungus through root grafts is ideally suited to the dense closed stands that were the result of fire cessation. The extent of root grafting in the grub savannas is largely unknown, as is the result of fires on survival of the fungus. Thus, it is impossible to determine the previous importance of oak wilt in oak savannas and forests before settlement.

4. Specific Predictions for Southern Wisconsin Upland Forests

The levels of oak wilt mortality in forests with a given composition, density, and size for the red oak group can be estimated and interpolated from the model results used in the sensitivity analysis, as well as many other simulations that were not reported here. I wish to apply such an analysis to specific southern Wisconsin forests.

The data used by Peet and Loucks (1977) are utilized. They picked 30 stands from Univ. of Wisconsin Plant Eco-

logy Laboratory studies, three from each tenth of the continuum. In these stands, the densities of all species, by size class, were tallied. The densities of oaks were transformed to the appropriate composition at nominal density values, and allowance was made for the average size of red oak group trees, which were all treated as one species. Only stems greater than or equal to 3.5 inches dbh were used to generate mortality. This provided a better fit to normal distributions of tree sizes used by the model, as well as avoiding problems due to a lack of data on small tree sizes. From this, it was possible to make approximate predictions of 40 year mortality from oak wilt using simulation results.

The problem of regeneration in oak wilt gaps was handled by assuming that the understory component, as quantified by Peet and Loucks (1977) would grow into any gaps caused by oak wilt in the succeeding 40 years.

Table 47 provides the detail for the predictions of both oak wilt mortality and subsequent compositional change after 40 years in the individual stands used by Peet and Loucks. This table is largely self explanatory. Recall the variability in output from the stochastic model in realizing that the predicted mortalities listed are only average values.

The information is summarized by each tenth of the

TABLE 47. Effects of Predicted Oak Wilt Mortality after 40 Years on Southern Wisconsin Oak Forests

STAND*	STARTING RED OAK GROUP ** no./acre	% of oaks	PREDICTED MORTALITY per acre	REMAINING RED OAK GROUP no./acre	% of oaks	REPRO-DUCTION***	PROBABLE EFFECT OF OAK WILT--40-YEAR RESULTS
11	288	84	? > 100	? < 180	? < 80	Hills and white oak	Large drop in Hills oak despite damage. Shift toward white oak
12	6	4	0	6	4	Elm, box elder	Little change
13	2	5	0	2	5	Bur oak, cherry, red oak	Little change
21	80	100	7	73	100	Black oak	Black oak reproduction should replace
22	10	28	0	10	28	Black oak, hickory	Little change
23	104	50	35	69	40	Bur oak, black oak, red oak, cherry	Some shift to bur oak and cherry, but persistence of R.O.G.
31	62	50	16	46	43	White oak, black oak	Shift toward white oak
32	26	27	3	23	25	Cherry, black oak	Little change
33	80	41	8	72	38	White oak	Shift toward white oak
41	28	23	3	25	21	White oak	Little change
42	44	48	4	40	45	Cherry, white oak, hickory	Some gap phase replacement by cherry and others
43	26	22	3	23	20	White oak, hickory	Little change

TABLE 47. (continued--page 2)

STAND*	STARTING RED OAK GROUP ** no./acre	% of oaks	PREDICTED MORTALITY per acre	REMAINING RED OAK GROUP no./acre	% of oaks	REPRO-DUCTION***	PROBABLE EFFECT OF OAK WILT--40-YEAR RESULTS
51	108	93	70	38	83	Cherry	Mass replacement by cherry
52	52	60	18	34	50	White oak, hickory, elm, others	major shift to non-R.O.C. species
53	76	52	35	41	37	Cherry, white oak	Mass replacement by cherry and white oak
61	92	66	30	62	56	Basswood, yellow-bud hickory	Accelerated replacement by mesic species
62	74	76	25	49	51	Red maple, white ash	Accelerated replacement by red maple
63	6	19	0	6	19	Yellowbud hickory, slippery elm	No effect on species replacement
71	12	50	1	11	48	Very little	?
72	20	77	3	17	74	Red oak, yellow-bud hickory	Possible red oak replacement
73	58	94	13	45	92	Yellowbud hickory	Accelerated replacement
81	4	9	0	4	9	Sugar maple	No effect on species replacement
82	52	100	19	33	100	Sugar maple	Accelerated replacement by sugar maple
83	2	10	9	2	10	Little	No effect on species replacement

TABLE 47. (continued--page 3)

STAND*	STARTING RED OAK GROUP** no./acre	% of oaks	PREDICTED MORTALITY per acre	REMAINING RED OAK GROUP no./acre	% of oaks	REPRO-DUCTION***	PROBABLE EFFECT ON OAK WILT--40-YEAR RESULTS
91	4	100	0	4	100	Sugar maple	No effect on species replacement
92	0	0	0	0	0	Sugar maple, hackberry	No effect on species replacement
93	6	100	1	5	100	Sugar maple, yellowbud hickory	No effect on species replacement
101	0	0	0	0	0	Sugar maple yellowbud hickory	No effect on species replacement
102	2	50	0	2	50	Sugar maple	No effect on species replacement
103	4	100	0	4	100	Sugar maple	No effect on species replacement

* Tens place in stand number indicates tenth of continuum on which stands are placed.
** Date from Peet and Loucks (1977).
*** Trees less than or equal to 3.5 inches dbh. Listed in order of density. For generic names, see Appendix 1.

continuum in Table 48. The 4/10 of the continuum on the xeric end are predicted to show highly variable damage. However, if understory trees fill oak wilt gaps, damage should not often result in major species shifts away from oaks. In many cases, however, increased abundance of the white oak group is predicted.

The fifth segment, and some other stands in neighboring segments, show large amounts of oak wilt damage on red and black oaks. Replacement by gap phase species such as cherry and shagbark hickory is postulated. The next three most mesic segments (6-8) show low to moderate oak wilt damage which, in most cases, merely serves to accelerate species replacement by sugar maple and other tolerant species. In one case, red maple replacement is postulated. The virtual absence of oaks from the most mesic end of the continuum leads to the obvious conclusion that species replacement should proceed without any effect of oak wilt.

Several caveats to this analysis bear mentioning. First, the effect of shrubs is ignored; this may be important in some of the xeric and dry-mesic stands. Secondly, ingrowth was assumed to be proportional to the number of stems less than 3.5 inches, and not to their relative height (see Arend and Scholz 1969). Thirdly, the effects of other disturbances are ignored. Also,

TABLE 48. Summary of Predicted Oak Wilt Mortality and Prediction of Species Replacement for Southern Wisconsin Forests

1/10th of CONTINUUM	RANGE OF DAMAGE	PREDICTED EFFECTS OF OAK WILT ON SPECIES REPLACEMENT
1 (dry)	None-Extreme	None or shift toward White Oak
2	None-Large	None or replacement by cherry with persistence of black oak
3	Moderate	Shift toward white oak
4	Low	Little effect
5	Extreme	Cherry, hickory replacement
6	None-Large	Accelerated species replacement
7	Low-Moderate	Various
8	None-Moderate	Little effect or accelerated species replacement
9	None	Little effect
10(mesic)	None	Little effect

many of the values for oak wilt damage are rough approximations from comparable simulations, not the results of explicit model runs. Finally, regional oak wilt dynamics will likely vary conditions of the model, e.g. the level of background spread.

One could presumably take the results after 40 years and plug in the predicted oak wilt mortality values again. This was not done for several reasons. First, there would then be no information on what the ingrowth would likely be. Secondly, long term system stability should also be a function of germination probabilities, which will depend in complex ways on the extent of disturbance. Thirdly, there is no knowledge of the effects of oak wilt mortality and species replacement on the effective seed rain in oaks. Finally the epidemiological conditions implied by the nominal simulation will almost certainly change in 40 years.

5. Conclusions for Oak Forests

It was previously argued that it is reasonable to expect that the oak forest-oak wilt system is globally stable, i.e. there will not be large scale extinctions of red oak group species or of the fungus. The extent of several disturbances will favor oak persistance in the face of competitors. Also, the slow spread of Ceratocystis allows a fairly low level of other disturbances to provide

enough available opportunities for oaks. The patchy conformation of both the disease and woodlots in the Midwest should also be important. As oak densities decrease, inoculum levels should drop also. Local concentrations of red oak group trees may thus escape damage due to the poor overland dispersal of the fungus.

Since oaks should persist, and since the oak wilt fungus can survive for some time in root systems, it is also unlikely that the fungus will go extinct over large areas. An additional factor favoring the disease organism is the role of white oaks in harboring inoculum for further spread. Since white oaks are less often killed, they provide a dampening effect on the density dependent system.

Still, I anticipate a decline in the importance of the red oak group. These species are adapted to fire and do not have appropriate germination requirements for undisturbed forests. In addition, oak wilt will be important in many situations.

The implications of oak wilt for the local persistence of oak, as predicted by this analysis, depend on the specific woods. In more mesic situations, oak wilt will likely accelerate succession. In xeric situations, it should cause a shift in composition to members of the white oak group, but black oaks are likely to remain important. In dry-mesic areas, the effect of oak wilt mortality and

the gap phase behavior of cherry and shagbark hickory will probably cause large compositional shifts. I agree with the predictions of Peet and Loucks (1977) that posit an equilibrium of cherry, hickory, and white oak in dry-mesic forests, an equilibrium maintained by gap phase replacement. It also appears as if the second possibility outlined by Auclair and Cottam (1971) is the most likely: equilibrium at lower amounts of oak in southern Wisconsin forests.

The above analysis assumes that openings caused by oak wilt favor the advanced reproduction. If they change the microenvironment greatly, or if there is little advanced reproduction present (see Auclair and Cottam 1971), then trees of the red oak group may be able to take advantage of gaps. There is presently a dearth of information on the importance of different gap sizes for oak reproduction, one that parallels the continuing lack of investigation on spatial dynamics of species replacement.

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APPENDIX 1. Tree Species Mentioned in Text*

Generic Name	Common Names Used
Acer Negundo	Box elder
A. rubrum	Red maple
A. saccharum	Sugar maple
Carya cordiformis	Yellow-bud hickory, bitternut hickory
C. ovata	Shagbark hickory, hickory
Castanea dentata	American chestnut
Celtis occidentalis	Hackberry
Erythrobalanus	Red Oak Group
Fraxinus americana	White ash
Leucobalanus	White Oak Group
Prunus serotina	Black cherry
*Quercus alba***	White oak
*Q. ellipsoidalis***	Hill's oak, northern pin oak
*Q. macrocarpa****	Bur oak
*Q. rubra***	Red oak
*Q. velutina***	Black oak
Ulmus americana	American elm, elm
U. rubra	Slippery elm
Tilia americana	Basswood

*- Nomenclature after Fernald (1950).

**- Erythrobalanus

***- Leucobalanus

APPENDIX 2. Distance of Vector Spread

For data taken at Sinnissippi Forest from 1956 to 1971, the distance from each red oak group tree killed by vector spread to the nearest red oak group tree dying of oak wilt the previous year was approximated from maps. This is the shortest distance over which vector transmission could have occurred, assuming that all vector spreads take one year between successive deaths. Curl (1955) found that the latter was true for over 80% of the cases he studied.

In this appendix, it will be shown that these nearest neighbor distances found are shorter than one would expect under a random null hypothesis. In particular, the nearest neighbor distances resulting from random association of newly-wilting trees with respect to previously wilting trees will be compared to the actual distances.

The analogy used will be with the quarter method; a plotless sampling method commonly used in quantitative phytosociology (Curtis 1959, Mueller-Dombois and Ellenberg 1974). Consider the population of previously wilting trees as objects of measurement (entities) in the quarter method. Further consider the newly wilting trees as points placed to measure distances to the entities. If the 'point' is placed randomly with respect

to the entities (i.e. if the newly wilting tree is located randomly with respect to previously wilting trees), the nearest neighbor distance should equal $0.5/\sqrt{\text{Mean Area}}$, where the mean area is 1/density of the entities. This relationship was first empirically derived by Cottam and Curtis (1956) and proven by Morisita (1954). The nearest distances between points and entities correspond to the first distances in the quarter method.

If the points are closer than expected to the entities, then the average distance will be less than $0.5/\sqrt{\text{Mean Area}}$. This assumes that the entities (i.e. the previously wilting trees) are randomly distributed. Since many of these trees were probably infected by root graft transmission, they are almost certainly highly aggregated. This will increase the average closest distance to the point (i.e. newly wilting tree) and make the test more difficult (i.e. more conservative). A significant difference will be more highly significant than the level of significance would indicate.

The actual distances (mean 540 feet) were significantly less ($p < .05$, Chi square test) than expected (mean 584 feet) despite the assumption that previously wilting trees were randomly distributed. There is thus a very strong indication from statistical evidence that vector spread occurs most often over short distances.