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Comparing Epizootic Systems Using Spectral Analysis and Autoregression: A Case Study on *Tetranychus–Neozygites* Mycosis

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Abstract

Host-pathogen interactions at the population level were studied using the twospotted spider mite, *Tetranychus urticae* Koch, and the pathogenic fungus, *Neozygites floridana* Weiser and Muma, as a model system. The generation time (egg to adult) of the twospotted spider mite is approximately 10 days while the generation time of *N. floridana* (host penetration to release of spores) is approximately 4 days when both are reared at 21°C. Three different systems were studied in relation to epizootic events. In System 1, leaf disks were infested with 250 eggs, 125 immatures, 5 male adults, and 20 female adults. In System 2, 25% of the immatures were added every other day and in System 3, 25% of the immatures were removed every other day. Our objective was to change the rate of increase of the host population. These systems were observed for 200 days. Spectral analyses showed that regulation of the host existed under 3 conditions. First, the pathogen could regulate the host in an epizootic system in which no hosts were removed but in which pathogens were periodically added. Regulation also occurred in systems in which hosts were added or removed, as long as a critical number of pathogen units (mummies) was maintained. Finally, a single introduction of pathogens could regulate the host in systems in which a portion of hosts was periodically removed. Autoregression and cross-correlation analyses of this system indicated that the number of mites that became infected can almost always be predicted by the number of eggs, but it cannot be predicted by the number of immatures. Moreover, densities of infected hosts could be forecast from a range of time lags of 2–16 days except for a time lag of 8 or 10 days.

Introduction

An epizootic system consists of 3 basic components: host, pathogen, and environment (1, 2, 3). Under a given set of environmental conditions, host-pathogen interactions are affected by host density, susceptibility, behavior, interspecific characteristics (4), pathogen density, infectivity, latency, survival, spatial distribution (5) and transmission characteristics (6). Simple models have long been used to study the elementary dynamics of such systems (7, 8, 9). These authors differentiate the host population into 2 distinct subpopulations of susceptible and infected hosts which were treated separately. Similar approaches have also been proposed by Anderson and May (10, 11, 12) who treated the host component as a composite of the 2 subpopulations. These studies have resulted in establishment of the threshold density concept with various epizootiological implications (13, 14) and unification of ecological (predator-prey) and epizootiological (pathogen-host) models (15).

Epizootic systems are often periodic which means that they can be analyzed by identifying the dominant cycles (16). One way to identify those cycles is to use spectral analysis. This analysis has been successfully used in identifying periodicities in human epidemics (17). When the spectral density (variance of evenly spaced data points) is plotted against its frequency, power spectra peak in the dominant cycle(s) of the system. The biological importance of the cycle(s) is identified by correlating it with the biological characteristics of the system of interest.

In this study, we evaluated 2 methods, spectral analysis and autoregression, to analyze epizootic systems using the twospotted spider mite, *Tetranychus urticae* Koch as infected by the pathogenic fungus, *Neozygites floridana* Weiser and Muma, as an experimental model system.

Materials and Methods

Experiments were carried out in the Insect Pathology Laboratory, Department of Entomology, University of Kentucky, Lexington, KY, during January–April 1991. The epizootic model for this study consisted of the twospotted spider mite, *Tetranychus urticae* Koch, reared on bush bean plants (Taylor Strain) and the pathogenic fungus, *Neozygites floridana* Weiser and Muma, maintained in mummified mite cadavers. The original source of *N. flor-*

idana was obtained as a gift from Dr. George G. Kennedy of North Carolina State University, Raleigh, NC. Only adult mite cadavers were used as inoculum in this study. On those days when inoculation was to be performed, a separate population of cadavers was inspected and all cadavers removed. Four hours later, newly-killed cadavers were selected for use as inoculum as described by Brown and Hasibuan (18). Therefore, age of inoculum (newly-formed cadavers) never exceeded 4 hours. Cadavers always sporulated if humidity was at 100% RH.

Experimental units were plastic trays (14 × 17 cm) with wet cotton batting supporting 4 bush bean leaf disks (dia. 3 cm) connected to each other by 1 × 5 cm hardware cloth bridge spans. From an initial pool of 36 units, immatures and adults were placed on them and were randomly assigned to 3 groups of epizootic systems, i.e., System 1 without additions or removals of host mites. In System 1, each leaf disk was infested with 250 eggs, 125 immatures, 5 male adults, and 20 female adults. System 2 was similar except 25% of mite immatures were added every other day whereas in System 3, 25% of mite immatures were removed every other day. Each system was further subdivided into three subsystems or treatments, i.e., control with mites only, treatment A with a single pathogen introduction of 5 mummies, and treatment B with repeated pathogen introductions of 1 mummy every other day. Observations on prevalence of mycosis (number of infected hosts or new mummies) and the surviving mites (number of susceptible hosts) were done every other day. Between any 2 observations, all trays were kept in a growth chamber (Percival, Model I-35 L) set at 21 ± 1°C and 100% RH. These systems were run until all mites died of mycosis or 200 days which ever came first.

The data series of susceptible hosts were documented as the number of eggs, immatures, adult males, and adult females. The series of susceptibles and infecteds (cadavers) were smoothed using a five point polynomial method (19). Spectral analyses and autoregression were then conducted on these smoothed data.

1. Spectral Analysis

In this study, the SAS SPECTRA procedure (20) was used to generate spectral density plots in each epizootic system from the time series of susceptibles and infecteds. Some predictions about the epizootic systems were then inferred.

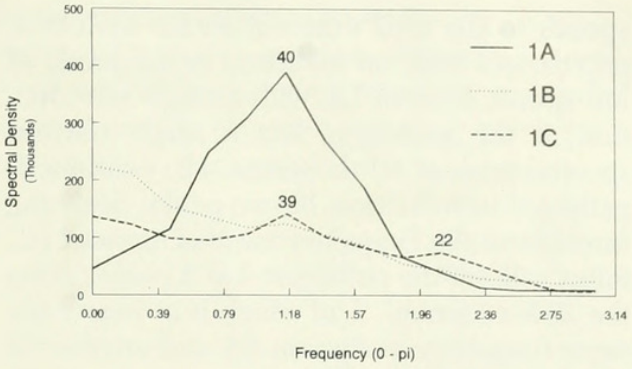

FIG. 1. Spectral density plots for total mites in System 1. System 1A (solid line) has a single obvious peak while system 1B (dotted line) has no peak. System 1C (dashed line) has two peaks that satisfy the criterion that the peak must be at least 10% higher than the preceeding and succeeding spectral densities. The numbers on the graph correspond to the entries for total mites, System 1 A, B, and C in Table 1.

2. Autoregression and Cross-Correlation Analysis

This analysis sought to correlate a data point with its future value with the expectation that the value at some future time (Δt) could be predicted by the original data point. To do this, pairs of regressor-predicted variables to be analyzed were $S(t)$ versus $S(t + \Delta t)$, $S(t)$ versus $I(t + \Delta t)$, $I(t)$ versus $S(t + \Delta t)$, and $I(t)$ versus $I(t + \Delta t)$, where S is the number of susceptibles and I is the number of infecteds. The values of Δt used were 2, 4, 6, 8, 10, 12, 14, or 16 days. The upper limit of 16 days lag was chosen in this study because the total developmental period of the mites is about 16 days (21).

RESULTS AND DISCUSSION

The criterion for identifying peaks in spectral density (which imply periodic fluctuations in these population systems) was that an increase of at least 10% in the spectral density was followed by a decrease by at least 10%. An example of the spectral density curves obtained from the spectral analyses is shown in Figure 1. This example, chosen because it displays the array of observed responses and demonstrates the use of this criterion, corre-

sponds to the total mite curves for System 1, populations with no additions or removals of immatures. System 1A, with a single introduction of the pathogen, has a single obvious spectral peak at 40 d. System 1B, continuous pathogen introduction, has no peaks; declining throughout the frequency interval. System 1C, mites without the pathogen, has 2 peaks (using the 10% criterion), 1 of which is at nearly the same frequency as System 1A and another of which is at 22 days.

The periodicities of all 9 systems are shown in Table 1. Over half (52%) of the variables investigated had one periodicity while 29% had none and the remaining 19% had more than one. Populations in System 2 (host additions) had more periodicities than those in the other systems. System 1 had the largest overall mean (32.8 ± 2.1 d), System 3 had the smallest (26.2 ± 3.1 d) and System 2 was intermediate with a mean (± S.E.) periodicity of 30.3 ± 2.9 d. None of these differences were significant.

The mean periodicity for each population is also shown in Table 1. These means were compared using all possible pairings in a two-tailed *t*-test ($P = 0.05$). In Systems 1 and 3, the mite population with no pathogen had significantly faster periods than the system with a single pathogen introduction. In contrast, these 2 population means were almost identical in System 2. Examining these means in order of increasing population growth rate (3—removal of immatures, 1—no manipulation of immatures, 2—adding immatures), reveals a clear trend of increasing periodicity with increasing growth rate on the populations with no pathogen. However, populations with the pathogen did not show an obvious trend.

This trend is somewhat clearer if one examines the individual variables in order of increasing growth rate. This trend is observable for immatures (System C), total mites (Systems A and C), proportion of eggs (System C).

Susilo had predicted that the infected hosts would show periodicities of 15–16 d with longer periods possible. This prediction was based, in part, on the spider mite's longevity which, under the conditions used here, is 15–17 d (21). The results in Table 1 confirm this in Systems 1 and 2 but not in 3 and, even then, only in the cases where the pathogen was repeatedly introduced. No regular peri-

TABLE 1. Periodicities (days) of the infecteds and classes of susceptibles in three epizootic systems tested. System 1 = no addition or removal of hosts; System 2 = addition of 25% of hosts every other day; System 3 = removal of 25% hosts every other day; C = no pathogens introduced; A = a single introduction of pathogens (five mite mummies initially); B = repeated introductions of pathogens (five mummies initially and one mummy every other day); p = proportion of corresponding host class to total susceptibles; x = no data; — = no periodicities; single values indicate single cycles; double or triple values indicate multiple periodicities.

	System 1			System 2			System 3		
Host classes	C	A	B	C	A	B	C	A	B
Infecteds	x	—	28; 17	x	—	71; 16	x	—	—
Suscept.									
Eggs	38; 22	40	42	32; 17	—	23	21	40	17
Immat.	37; 24	39	34	56; 17	—	21	21	23	—
Males	—	40	—	50; 17	54; 19	21	34	—	63
Femal.	24	—	—	—	53; 25; 16	54; 17	—	76; 18	20
Total	39; 22	40	—	46	53	23	25	24	21
pEggs	24	38	48	27; 17	17	17	21; 17	18	17
pImm.	24	37	60	55; 17	53; 18	18	—	51; 18	20; 17
pMales	23	—	23	48; 20; 18	20	—	22	—	17
pFem.	25	—	—	—	16	—	—	16	18
Mean ± S.E.	27.46 ± 2.06	39.0 ± 0.51	36.0 ± 5.67	31.21 ± 4.30	31.27 ± 5.31	28.10 ± 5.93	23.00 ± 2.04	31.56 ± 6.82	23.33 ± 4.99
Sign. Diff.	c	a	ab	bd	bcd	bde	f	bcde	ef

TABLE 2. Significance test for time lags for predicting susceptibles or infecteds.

Variables		Lags (days)							
Predicted	Predictor	2	4	6	8	10	12	14	16
$s(t + \Delta t)_{Eggs}$	$S(t)_{Eggs}$	**	**	—	—	—	*	**	*
$I(t + \Delta t)$	$S(t)_{Eggs}$	—	**	**	**	**	**	*	*
$S(t + \Delta t)_{Eggs}$	$I(t)$	—	—	—	—	—	—	*	*
$S(t + \Delta t)_{Imm}$	$S(t)_{Imm}$	**	**	**	—	—	*	**	**
$I(t + \Delta t)$	$S(t)_{Imm}$	—	—	—	—	—	—	—	—
$S(t + \Delta t)_{Imm}$	$I(t)$	—	—	—	—	—	—	—	—
$I(t + \Delta t)$	$I(t)$	**	**	*	—	—	*	**	**

— Non-significant.
* Significant at $P < 0.05$.
** Significant at $P < 0.01$.

odicity was observed in the populations subjected to a single introduction of the pathogen. The pathogen did persist in these populations, sometimes at high levels of incidence, there just wasn't a regular periodic fluctuation. Brown (in press) has presented evidence that the interaction between the host and pathogen is chaotic and aperiodic.

Brown (22) and Hasibuan (23) have both suggested that mite populations on the leaf disks used here tend to overdamp. This is why periodicities would tend to increase with increasing growth rates when growth rates are manipulated by increasing the population by a predefined *percentage* of those already present. However, when the pathogen is present, it serves to reduce the mite growth rate thereby lessening the overdamping effect. Consequently, in these populations, the pathogen would tend to cause the periodicities to decrease as more immatures are added. It is this apparent conflict between the mite's intrinsic dynamics and those of the mite-pathogen system that cause some of the periodicities to decrease in System 2.

For the autoregression analysis, numbers of mite eggs, immatures, and infected hosts in System 2B were used to represent a continuum of susceptible and infected host classes tested because the prediction about host-pathogen regulation in that system was the resistant to perterbation. Results of autoregression and cross-correlation analyses on the above three host-pathogen classes revealed that the number of infected hosts can almost always be predicted by the number of eggs (Table 2). Results indicate that the number of eggs can be predicted by the number of infected hosts using the time lags of 14 or 16 days. However, the number of infecteds cannot be used to predict the number of immatures, and vice versa. Moreover, a future value of infecteds can almost always be predicted by a previous value of infecteds, except when time lags of 8 or 10 days were used. The same exception was true for self-predicting the number of immatures. Self-prediction of the number of eggs was possible using time lags of 2, 4, 12, 14, and 16 days.

CONCLUSION

Spectral density analysis on 3 epizootic systems of *Tetranychus–Neozygites* mycosis showed that pathogen cycles were maintained in systems where pathogens were repeatedly introduced. Host-pathogen regulation may occur in epizootic systems when a portion of that host was routinely added or removed or in systems with host additions or removals as long as the pathogen base-level was maintained.

Autoregression analysis on susceptibles (mite eggs or immatures) and infecteds (mite mummies) demonstrated that the number of infecteds can almost always be predicted by the number of eggs. Future values of infecteds, eggs, or immatures can be predicted by their corresponding previous values for a range of time lags of no more than the developmental time of the host (16 days), except for the time lags of 8 or 10 days.

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