Spatial Pattern of Downy Mildew in Hop Yards During Severe and Mild Disease Epidemics

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ABSTRACT

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Hop plants in hills in three or four hop yards were assessed visually during severe and mild epidemics of downy mildew in the Yakima Valley of Washington. Several methods of analysis were used to investigate the spatial pattern of hop plants systemically infected with *Pseudoperonospora humuli* during the two types of epidemics. When disease incidences were low during the mild epidemics and early in the development of the severe epidemics, distributions associated with aggregation of disease provided a good representation of the frequency distribution of systemically infected shoots. A frequency distribution that represented data at high disease incidences was not found. Aggregation of disease within hop hills was indicated by large variance-to-mean ratios and Morisita's index during both types of epidemics. Disease among hills became more aggregated as disease incidence increased as shown by doublet and runs analyses. Evidence of aggregation between nearby hills was shown by the Greig-Smith index and by semi-variograms when incidence of disease was high during the severe epidemics. Spatial association between contiguous hills as well as more widely separated hills was not direction dependent. The statistical methods used were in general agreement and provided useful information about spatial structure at several spatial scales. There was an association between disease occurrence in one year and condition of the same hills in the next year.

Downy mildew of hop (*Humulus lupulus* L.) caused by *Pseudoperonospora humuli* (Miy. et Tak.) Wils. is a serious disease in many areas of the world where hop is grown (22,26). Although downy mildew is more severe in high rainfall areas (25,26), the semiarid environment of the Yakima Valley in Washington still experiences severe epidemics on an average of one in three years (14).

Epidemics of downy mildew in the Yakima Valley are usually of short duration because favorable weather for disease development in May is generally followed by unfavorable hot, dry conditions in June (14,26). The greatest losses in the Yakima Valley occur from crown infections that eventually result in death of crowns during the winter and subsequent stand reductions. Infected crowns range from sound to almost completely destroyed, and disease may progress for several years before death occurs. Losses also result from reduced yields due to infections of main shoots, lateral shoots, flowers, and cones (22).

P. humuli overwinters as mycelium in infected hop crowns (4,25). Mycelium may spread into developing buds and shoots in the spring resulting in systemically infected shoots. These shoots, known as "primary spikes," are typically stunted, chlorotic, and have down-curled brittle leaves. Infected crowns may have zero to many infected shoots. Sporangia are borne on the lower leaf surfaces of the primary basal spikes (29). They are released during rainy weather (10,20) and infect the apical meristem of healthy hop shoots when temperature and moisture conditions are favorable (22). These infected shoots are called "secondary spikes" and possess symptoms similar to primary spikes, except plant tissues below the infected area remain normal in appearance. Many shoots arise from the perennial crown and numerous axillary buds from lateral shoots on the four to eight main shoots selected to grow up two to three twines, 5.5 m high (22). All of these may potentially become infected and produce secondary spikes. Mycelium grows from the secondary spikes towards the crown during the growing season (4,25), and if the crown is reached, the fungus perennates over the winter in the crown.

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Disease assessment in hop yards and a disease-forecasting system are used to manage hop downy mildew in Washington State (13,26). The pattern of downy mildew occurrence in hop yards during mild epidemics was determined to increase sampling efficiency for estimation of downy mildew at low disease incidence (11). Hop is a perennial plant and is grown in hills, areas of soil that are not disturbed by cross-disking. Hills are placed in a square pattern with a distance of approximately 2.13 m between hill centers. This spacing was beneficial in determining the pattern of downy mildew infection by taking into account the actual location of the sampling sites in each test yard. A knowledge of the pattern of downy mildew during severe epidemics and additional knowledge during mild epidemics could improve sampling efficiency and increase knowledge of spread of the disease (19).

A severe downy mildew epidemic occurred in hop yards in the Yakima Valley in 1988 (final disease incidence 28–69%) and a mild epidemic in 1989 (final disease incidence less than 2%). The purposes of this study were to determine and compare the spatial patterns of downy mildew during severe and mild epidemics using several methods of analysis and to determine and quantify any association between primary infection one year and production of primary spikes, death of crowns, or weakness of crowns the next year.

MATERIALS AND METHODS

Four hop yards (varying from 5–10 ha in area) of the cluster cultivar L-1, which is very susceptible to downy mildew, were chosen for study. Two yards were near the town of Sunnyside (Sunnyside I,II), one near Mabton, and the fourth near Wapato. All hills in a rectangular section (2.3–1.9 ha in size) of each yard were assessed visually in 1988 and, with the exception of Sunnyside I, assessed again in 1989 for the number of primary and secondary spikes per hill. Yard Sunnyside I was removed from production after the 1988 harvest because of severe disease damage. Dates of assessment are shown in Table 1.

The study section of each yard was selected by choosing a hill randomly to begin observations that would include the desired

length and width. In 1988, dimensions were 60 × 88 hills (126 × 185 m) at Sunnyside I (total of 5,280 hills assessed), 60 × 80 hills at both Sunnyside II and Wapato, and 48 × 90 hills at Mabton. In all directional analyses, zero degrees was defined to be parallel to the long axis of the sampled area. Disease incidence was calculated as the percentage of total hills of living plants that had spikes. Some hills in each yard did not have live crowns so the number of spikes was missing from these hills.

The same plots were used in 1989 as in 1988 at Mabton, Sunnyside II, and Wapato. Hills with dead or weak (five or less shoots per hill and all shoots severely stunted) hop plants in 1989 that had living plants in 1988 were recorded. Chi-square contingency table analysis (7) and analysis of residuals (1) for each location examined the relationship between hills with or without primary spikes in 1988 and the disease status of the same hills in 1989.

Distribution fitting, variance-to-mean ratios, Morisita's index (18), doublet and runs analyses (5,7,8), Greig-Smith analysis (9), and semi-variograms (16) were used to examine the spatial distribution of downy mildew within hills, between nearby hills, and for hills more separated in space. Chi-square goodness-of-fit distribution fitting, variance-to-mean ratio, and Morisita's index allowed examination of variability of number of spikes within the hill. Runs and doublet analyses were used to search for small-sized clumps of diseased hills. Greig-Smith analysis was used to search for larger clumps in the number of spikes for contiguous hills.

A FORTRAN program developed by Gates and Ethridge (6) was used to calculate the chi-square goodness-of-fit statistic of the data to the Logarithmic with Zeros, Negative Binomial, Neyman Type A, Poisson, Poisson-Binomial, Poisson with Zeros, and Thomas Double Poisson distributions (15). The program pooled adjacent categories that had small expected frequencies until the cumulative frequency exceeded one.

Two indices of dispersion were used to indicate departures from randomness in spatial patterns. Morisita's index of dispersion is expected to be less than one for a uniform spatial pattern, equal to one for a random spatial pattern, and greater than one for a spatially aggregated population (23). The ratio generally increases as aggregation becomes more intense. The ratio of the variance to the mean was also computed and compared to one in the same manner as Morisita's index (28).

A method suggested by Greig-Smith was used to indicate the number of hills in disease clumps (18,20,24). The number of spikes in each hill was counted. Adjacent pairs of hills were then combined to give oblong two-hill blocks that were twice as big and half as numerous as the original number of hills. Adjacent two-hill blocks were combined to give square four-hill blocks, and so on, up to blocks of 128 hills. The total variation about the mean for single hill blocks was then apportioned as in a hierarchical analysis of variance, and the components of variation were plotted against the number of hills in successively doubled block sizes. A peak in variation indicates a clump of disease of size equal to the number of hills in the corresponding block. The peak will be maintained for larger blocks provided the clumps are not regularly spaced. An approximate F test (27) was used to indicate existence of clumps by identifying the clump size associated with the Greig-Smith value, which first exceeded a 0.01 critical F value. Peak values that were greater than the F value and were both preceded by smaller variation and followed by smaller or nearly constant variation values were interpreted as indicating a clump.

Spatial autocorrelation was examined with semi-variograms, which display the degree of correlation between disease counts from spatially contiguous hills at successively larger distances. The distance over which the semi-variance continues to increase indicates the range of influence of diseased hills on nearby hills. A spherical model was fit to the observed semi-variogram using a method suggested by Journel and Huijbregts (16). The fit was achieved by considering the following features of the experimental semi-variogram. The tangent to the curve at the origin was approximated by the average linear behavior of the first three

TABLE 1. Summary of disease incidence and indices for incidence of primary and secondary infections of *Pseudoperonospora humuli* in hop plants in 1988 and 1989

Location and date[a]	Disease incidence[b] (%)	Distribution (P value)[c]				Morisita's index[d]	Greig-Smith peaks[e]
		Poisson with zeroes	Negative binomial	Neyman type A	Logarithmic with zeroes		
Wapato							
4/29/88 P	1.7	0.07	···	0.11	···	52.4	···
4/29/88 P+S	1.7	0.13	···	0.18	···	52.7	···
5/13/88 P+S	18.9	···	···	···	···	19.7	2V, 4H, 16V, 64V, 128+H
5/31/88 P+S	21.1	···	···	···	···	16.2	2V, 4H, 16V, 64V, 128+H
6/15/88 P+S	26.0	···	···	···	···	6.4	2V, 4H, 64V, 128+H
6/28/88 P+S	28.2	···	···	···	···	3.8	2V, 4H, 64V, 128+H
5/12/89 P+S	1.7	···	0.40	···	0.39	105.9	···
Sunnyside II							
4/28/88 P	2.7	···	0.20	···	0.25	47.8	2H, 64V, 128H
4/28/88 P+S	2.7	···	0.52	···	0.49	49.4	2H, 128H
5/12/88 P+S	25.2	···	···	···	···	13.3	4V, 8H, 16V, 32H, 64V, 128+H
5/26/88 P+S	30.6	···	···	···	···	10.7	4V, 8H, 16V, 32H, 64V, 128+H
5/11/89 P+S	0.6	···	0.71	···	0.71	391.1	2V, 8V, 16H, 64H, 128V
Mabton							
5/6/88 P	15.6	···	···	···	···	8.3	2H, 64H, 128V
5/6/88 P+S	25.4	···	···	···	···	6.8	2H, 8V, 64H, 128+V
5/23/88 P+S	61.3	···	···	···	···	4.1	2V, 4H, 64H, 128+V
5/16/89 P+S	1.8	···	0.48	···	0.47	62.5	4H, 8V, 16H
Sunnyside I							
5/3/88 P	1.3	···	0.36	···	0.40	73.2	64H, 128V
5/3/88 P+S	1.4	···	0.18	···	0.34	100.5	4B, 64H, 128V
5/17/88 P+S	68.9	···	···	···	···	3.1	2V, 4H, 16V, 128+B

[a] P = data for primary spikes only; P+S = data for both primary and secondary spikes.
[b] (Number of hills with spikes/total hills with living plants) × 100.
[c] Probability value for chi-square goodness of fit test. ··· = significant lack of fit ($P < 0.05$).
[d] All values greater than 1.0 ($P < 0.01$).
[e] Direction and block size of significant peaks. H = horizontal orientation (0 degrees); V = vertical orientation (90 degrees); B = both 0 and 90 degrees; ··· = no indication of clumps of diseased plants for block sizes examined; 128+ = index greater than critical F value and still increasing at largest block size examined.

experimental points. The horizontal part of the curve, the sill, is at a value around which the semi-variogram becomes stable, taken as the variance of the data. The distance at which the tangent intersects the sill was taken as two-thirds of the value of the range of influence.

Doublet analysis (5,8) compares the observed number of pairs of adjacent diseased plants to the number expected if disease were randomly distributed in the yard. If the observed number is greater than the expected number, contagion is suspected. Doublet analysis was used to examine spread in the direction of each axis of the rectangular hops yard and in two diagonal directions.

A runs analysis (7) was also used to examine aggregation of diseased hills compared to the mixing of diseased and healthy hills expected for randomly distributed occurrence of disease. A runs analysis considers larger sequences of diseased hills in contrast to doublet analysis, which focuses on occurrence of adjacent diseased hills. A run is defined as a succession of one or more healthy or diseased plants that are followed and preceded by a plant of the other disease status or no plant at all. There would be few runs if there were aggregation of diseased or healthy plants and a large number of runs for a random mixing of diseased and healthy plants. Under the null hypothesis of randomness, the expected value and standard deviation of the total number of runs in the i^{th} row, denoted by $E(U_i)$ and $S(U_i)$, respectively, may be obtained from equations presented by Madden et al (17).

The ordinary runs analysis (7) was modified to allow for multiple rows of observations and for missing values. A row was defined

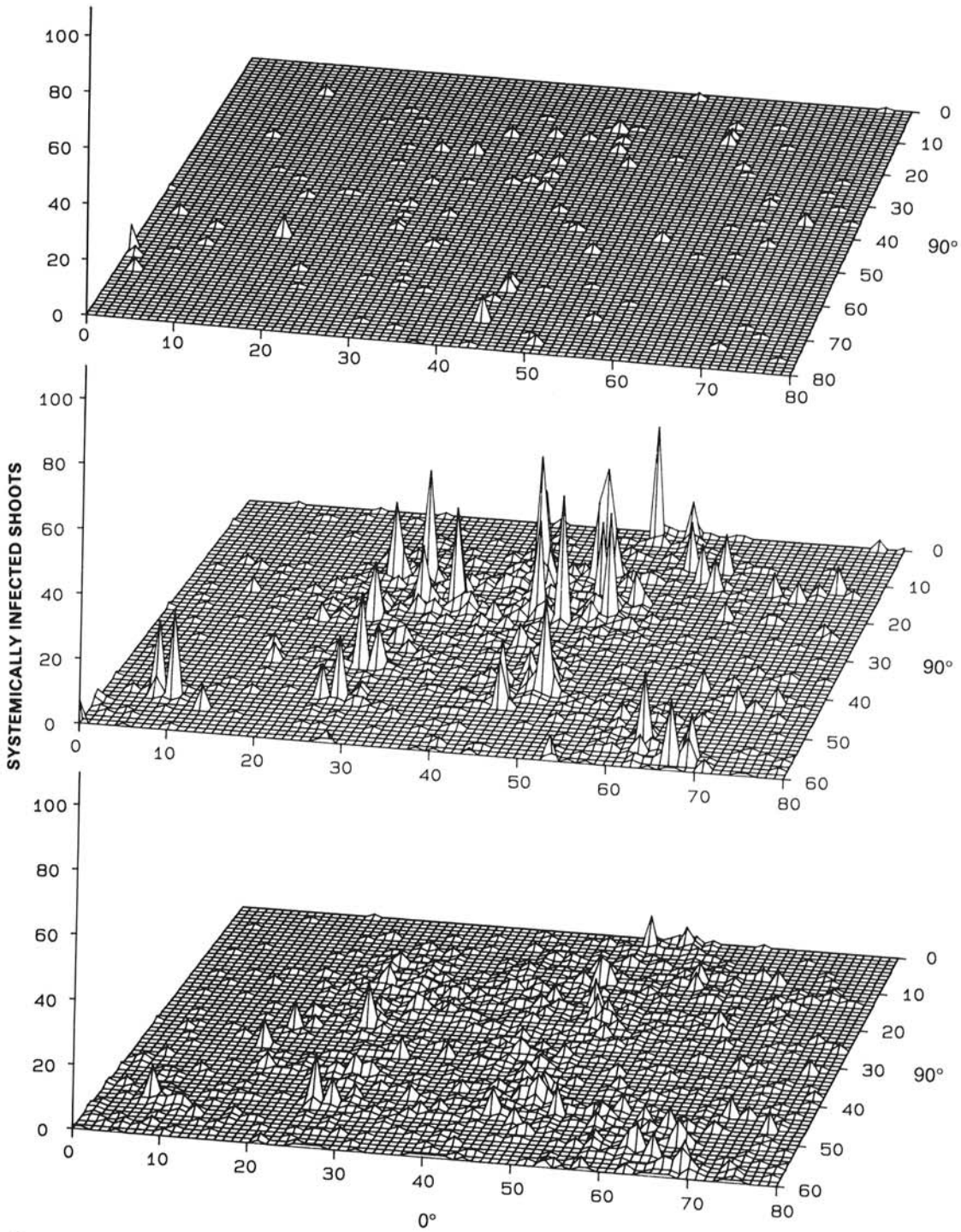

Fig. 1. Pattern of hop plants systemically infected with *Pseudoperonospora humuli* at Wapato on 29 April (top), 13 May (middle), and 28 June 1988.

as a series of adjacent hills in the specified direction terminated either by the boundary of the study section in the yard or by a missing hill. A test statistic with an asymptotic normal distribution may be defined as:

$$Z(U) = [\Sigma U_i + 0.5 - \Sigma E(U_i)] / [(r\,\Sigma[N_i - 1]\,S^2[U_i]) / \Sigma(N_i - 1)]^{1/2}$$

in which N_i is the total number of hills in the i^{th} row and r is the number of rows in the sampled area as defined above. The value of $Z(U)$ will be a large negative number if there is clustering; so the test for nonrandomness is left-tailed. The runs test was performed in the same directions as the doublet analyses.

RESULTS

Weather favored development of a severe and a mild epidemic in 1988 and 1989, respectively (14). Incidences of hills of hop plants with spikes for the various sampling dates in 1988 and 1989 are shown in Table 1. The pattern of infected hop plants at Wapato during three dates is illustrated (Fig. 1).

There was evidence of aggregation of spikes within hop hills. Neyman Type A, Logarithmic with Zeros, and Negative Binomial were the best fitting distributions in 1989 and for most initial sampling dates in 1988. For later dates in 1988, none of the distributions considered provided an adequate fit to the number of spikes (Table 1). For both 1988 and 1989 the variance-to-mean ratio and Morisita's index were significantly ($P < 0.01$) greater than one at all locations and on all dates (2,23). There was also evidence of aggregation of spikes among nearby hills. With few exceptions, clumps were identified by Greig-Smith approximate F tests for all locations in 1989 and for all locations on all dates in 1988 (Table 1). Figure 2 illustrates a Greig-Smith plot for Wapato data. Semi-variograms indicated little spatial autocorrelation for early dates in 1988 and for 1989. Data from the later dates in 1988 showed some autocorrelation in all yards. The semi-variograms for Wapato showed autocorrelation between number of spikes in hills in all four directions examined with a range of influence of approximately eight hills. Semi-variograms for the last two dates for Sunnyside II showed some autocorrelation in the 0 degree direction with a range of influence of about four hills. Mabton and Sunnyside I showed spatial association in two and four directions, respectively, with ranges of influence between four and eight hills. Figure 3 illustrates the fit of a semi-variogram model to Wapato data and estimation of the range of influence.

Aggregation of hills considering only presence and absence of disease was indicated by both doublet and runs analyses. For the first sampling date in 1988 in all locations these methods showed aggregation in fewer directions than the analyses of data from subsequent samples. Significant z-tests were computed for all directions for the later dates at all locations in 1988. In 1989, significant z-tests were obtained by the doublet analysis in all yards in all directions except one for Wapato and one for Mabton. The doublet and runs analyses showed no evidence of an increase in disease spread in the direction of the prevailing wind in either year (Table 2). The modified runs test showed fewer aggregations than the doublet analysis. Significant z-tests in doublet but not runs analysis occurred on the first sampling date for Sunnyside II, Mabton, and Sunnyside I in 1988 and for at least one direction in each yard in 1989 (Table 2).

Chi-square contingency table analyses indicated an association between hills with primary spikes in 1988 and disease status in

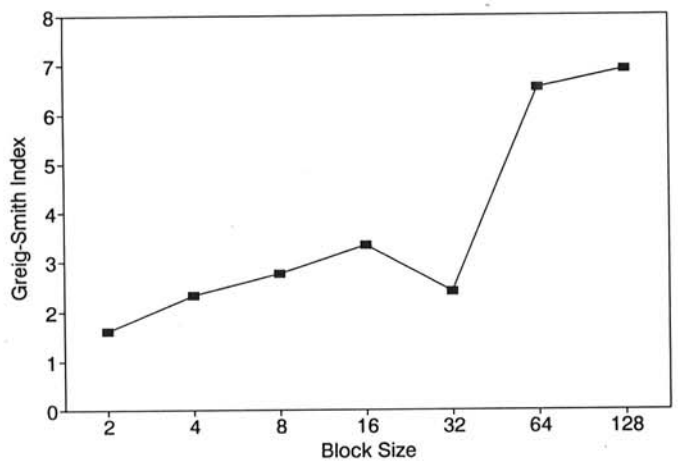

Fig. 2. Greig-Smith index as a function of the number of hills in vertically oriented (90 degrees) blocks for Wapato on 13 May 1988.

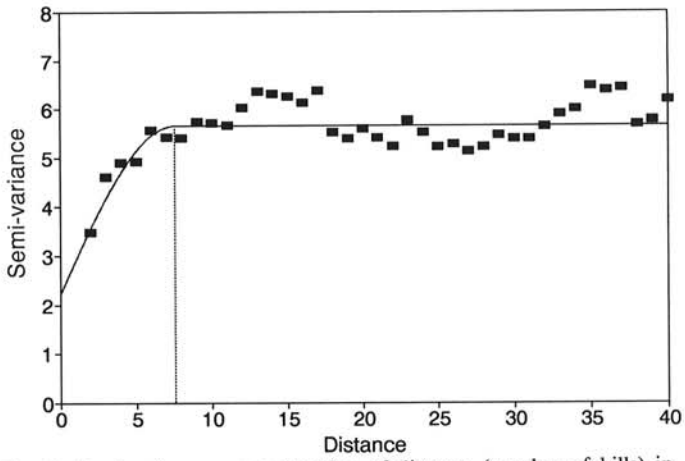

Fig. 3. Semi-variogram as a function of distance (number of hills) in the vertical (90 degrees) direction for Wapato on 13 May 1988. Solid line obtained by fitting the spherical model $F(h) = C[1.5(h/a) - 0.5(h^3/a^3)] + C_O$, in which C_O is the Y intercept, $C + C_O$ is the sill value, and h is the number of hills. Dotted line indicates the estimated range of influence (a) of a diseased hill on nearby hills.

TABLE 2. Doublet and runs analyses of hop plants with primary and secondary infection of *Pseudoperonospora humuli* in hop yards for one mild (1989) and one severe epidemic year (1988)

Location and date[a]	Angle[b] 0	45	90	135
Wapato				
4/29/88 P	−−	−−	−−	−−
4/29/88 P+S	−−	−−	−−	−−
5/13/88 P+S	**	**	**	**
5/31/88 P+S	**	**	**	**
6/15/88 P+S	**	**	**	**
6/28/88 P+S	**	**	**	**
5/12/89 P+S	*−	−−	*−	*−
Sunnyside II				
4/28/88 P	*−	*−	−−	−−
4/28/88 P+S	*−	*−	−−	−−
5/12/88 P+S	**	**	**	**
5/26/88 P+S	**	**	**	**
5/11/89 P+S	**	**	**	*−
Mabton				
5/6/88 P	**	−−	**	−−
5/6/88 P+S	**	*−	**	**
5/23/88 P+S	**	**	**	**
5/16/89 P+S	**	*−	*−	−−
Sunnyside I				
5/3/88 P	**	−−	−−	*−
5/3/88 P+S	**	−−	*−	*−
5/17/88 P+S	**	**	**	**

[a] P = data for primary spikes only; P+S = data for both primary and secondary spikes.

[b] Zero degrees is defined to be parallel to the long axis of the sampled area. ** Significant aggregation according to approximate z-test for both doublet and runs analyses ($P < 0.05$). *− Significant aggregation according to approximate z-test for doublet but not for runs analysis ($P < 0.05$). −− No significant aggregation by either doublet or runs analysis.

1989 ($P < 0.05$) for all three yards considered. Among the 1988 diseased hills there were significantly ($P < 0.05$) more weak hills at Sunnyside II and Wapato in 1989 than expected. More missing hills were observed than expected in 1989 at Mabton. The incidence of hills with primary spikes in 1989 was significantly greater than expected at Wapato (Table 3).

DISCUSSION

Individuals of a population are rarely spread uniformly throughout a given space. Spatial variation is a property of natural populations and is often dynamic, changing as individuals in the population increase or decrease in number or migrate (3,21). Likewise, the pattern of downy mildew changed as the incidence of disease increased from low to relatively high levels during the progress of the epidemic in 1988 (Fig. 1).

During the first sampling dates in 1988 when disease incidences were low, distributions associated with aggregation of disease provided a good representation of the frequency distribution. None of the distributions examined represented the data when disease incidences were higher for the later dates in 1988. The Negative Binomial distribution provided a good representation of the data for all locations in 1989 and in another study in 1985 when the incidence of disease was low (11).

Aggregation of disease was evident within the hill in both 1988 and 1989 as shown by variance-to-mean ratios and Morisita's index having values greater than one. Morisita's index for primary and secondary spike data combined decreased after the first sampling date as disease incidence increased during the severe epidemics at all locations in 1988 (Table 1).

The Greig-Smith analysis gave evidence of clumps of spikes among nearby hills for all locations and dates except the initial date at Wapato in 1988 and Wapato in 1989. The size of the clumps varied from two hills to 128 hills, the largest block size examined. Semi-variograms for 1988 indicated more spatial association among hills with spikes for later dates when disease incidence was higher. Semi-variograms for later dates at Wapato in 1988 indicated more autocorrelation than for other data. For Wapato a range of influence of approximately eight hills was estimated, which supports the size of clumps identified by Greig-Smith analysis (e.g., 64 hills = 8 × 8 hill clumps). This agreement is not surprising because semi-variance computed for a series of increasing lag distances is related to the nested variance computed from clumps of increasing number of hills.

In 1988, aggregation as shown by doublet and runs analyses was more consistent after the first sampling date when disease incidences had increased. The doublet analysis showed more aggregation than the runs analysis at the low incidences of disease in 1988 and 1989 because aggregation of disease was mostly of adjacent hills and not of larger sequences of diseased hills.

No difference in aggregation due to prevailing winds was observed from doublets or runs analyses in 1988 and 1989. This agrees with the 1985 study, which pointed out that only sporangia liberated during wet periods survived to produce zoospores and that wind direction at ground level in the Yakima Valley is quite variable during rainfall. Therefore, an aggregation in the direction of the prevailing winds would not be expected (11).

An aggregated rather than random or regular pattern was expected for hop downy mildew because new infections are more likely to occur near an inoculum source (22). Dispersal of primary inoculum of *P. humuli* in hop yards in England and Germany was found to be short-ranged, and secondary infections began on leaves and shoots neighboring primary spikes. This is because primary spikes are close to the ground and often sheltered within a canopy of healthy basal shoots. When secondary spikes occur higher on the bine, dispersal of inoculum is farther (22). Observations and data analyses in this study support the above findings that primary inoculum is usually dispersed over a short distance early in an epidemic and during mild epidemics.

Hills with primary spikes were scattered throughout all the yards during the first sampling date in this study (Fig. 1) and in the 1985 study (11). This widely scattered pattern of diseased hills may have originated from the initial planting containing infected root stocks. The infected planting material probably would have either been in a clustered or random pattern. Over time, infections occurred, and hills with downy mildew infection would have been clustered. Some of the crowns of the newly infected plants would have become infected after mycelium of the fungus grew down the infected shoot into the crown (4); a low proportion of the infected crowns would produce primary spikes the following year. For example, in this study a hill with primary spikes in 1988 did not necessarily produce primary spikes in 1989; Skotland also observed that only a small number of infected crowns produced primary spikes year after year (25).

TABLE 3. Disease evaluation and contingency table analysis during 1989 for hop hills with primary spikes of downy mildew in 1988

Location	Condition of hill in 1988		Condition of hill in 1989				
			Primary	Missing	Weak[a]	Other	Total
Wapata	Primary infection	Obs[b]:	5	1	9	101	116
		Exp:	1.6287	0.8790	1.7321	111.7602	
		Res:	2.6417*	0.1291	5.5223*	−1.0178	
	No primary infection	Obs:	58	33	58	4,222	4,371
		Exp:	61.3713	33.1210	65.2679	4,211.2398	
		Res:	−0.4303	−0.0210	−0.8996	0.1658	
		Total	63	34	67	4,323	4,487
Mabton	Primary infection	Obs:	8	68	39	551	666
		Exp:	10.8793	47.1435	30.9034	577.0739	
		Res:	−0.8729	3.0376*	1.4565	−1.0854	
	No primary infection	Obs:	61	231	157	3,109	3,558
		Exp:	58.1207	251.8565	165.0966	3,082.9261	
		Res:	0.3777	−1.3142	−0.6301	0.4696	
		Total:	69	299	196	3,660	4,224
Sunnyside II	Primary infection	Obs:	1	2	12	142	157
		Exp:	0.4740	6.9071	3.7921	145.8268	
		Res:	0.7640	−1.8671*	4.2149*	−0.3169	
	No primary infection	Obs:	13	202	100	4,165	4,480
		Exp:	13.5260	197.0929	108.2079	4,161.1732	
		Res:	−0.1430	0.3495	−0.7890	0.0593	
		Total:	14	204	112	4,307	4,637

[a]A weak hill was defined as having five or fewer shoots and all shoots severely stunted.
[b]Obs = observed; Exp = expected; Res = residual (Obs-Exp)/Exp. *Significant Z (residual) or chi-square (overall chi-square) value, $P < 0.05$. Overall chi-square at Wapata, 39.55*; at Mabton, 15.78*; and at Sunnyside II, 22.70*.

The above sequence of events would continue each year when infections occurred. Some hills with infected crowns would eventually die during the winter and then be replaced with healthy plants by the grower. Patterns of hills with primary spikes having a lack of spatial autocorrelation such as has been observed in this study may then result. The time involved in the development of the patterns of hills with primary spikes in this study was 6 yr for Sunnyside II, 9 yr for Sunnyside I, and 28 yr for Mabton. Observations over the years have demonstrated that infected root stocks are an important source of original inoculum for new hop yards (D. A. Johnson, *unpublished*).

Downy mildew can rapidly increase in hop yards (20), as was observed in this study. Weak plants were associated with primary infection the previous year. Most likely crowns of these plants had been infected for several seasons and after a few years would eventually die. The incidence of primary spikes increased in yards where the severity of the epidemic was high the preceding season in another study (12). The population of hills and not individual hills was examined in the earlier study. In this study when individual hills were examined, primary spikes were associated with primary infection the previous year in one of three yards (Table 3).

The statistical methods used were in general agreement about spatial structure at several spatial scales. Morisita's index and the variance-to-mean ratio consistently identified aggregation of disease incidence within hills even though probability distributions associated with clumped data sometimes did not fit the data well. Doublet and runs analyses were usually consistent in identifying aggregation of disease occurrence among contiguous hills. The Greig-Smith method indicated many clumps of varying size whereas semi-variograms showed more autocorrelation among disease incidence clumps for the later dates in 1988. The increased aggregation of disease occurrence over time in 1988 was also identified by the runs and doublet analyses.

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