A Comparison by Simulation of Disease Dispersal in Pure and Mixed Stands of Susceptible and Resistant Plants

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Disease dispersion from a point source in pure and mixed stands of susceptible and resistant plants was followed by a simulation made by using the equation in respect with disease dispersion, $y = are^{-\delta x}$.

The effect of mixing of resistant plants in susceptible plants to inhibit the disease increase is reduced with the increase of disease dispersal gradient, δ , larger than 0.5. Infection rates calculated by the number of new lesions decrease straightly with the decrease of the proportion of susceptible plants against the logarithm of the proportion. This decrease is inhibited by the increase of disease dispersal gradient, δ . The effect of mixing of resistant plants is decreased with the decrease of spacing density.

The infection rates calculated by the total number of new and old lesions give a dropping curve which is asymptotic to 0.

Introduction

With proposal of uses of host population diversified by various types of resistance as a method stabilizing the pathogen populations, some experimental and theoretical studies were carried out on the effect of mixing of host genotypes to control diseases. However, many studies are required to lay quantitatively hold of changes of the frequency or the amount of virulent races depending upon the frequency of host genotypes for disease resistance.

LEONARD (1969a, b, c) experimentally and theoretically studied the effect of mixing of resistant and susceptible plants and obtained the equation

$$r_m = r_s + \frac{n}{t} \log_e m, \quad (1)$$

where r_m is rate of stem rust increase in a mixture of susceptible and resistant plants, r_s is rate of increase in a plot composed entirely of susceptible plants, m is the proportion of susceptible plants in the host mixture, n is the number of generations of rust increase and t is time in day. This equation is based on

$$\frac{x'}{x_0} = m^n \frac{x}{x_0}, \quad (2)$$

where x_0 is the proportion of host tissue initially infected, x is the proportion of infected host tissue in the fields in which only susceptible plants are grown, and x' is the proportion of infected host tissue in the field in which only half of the plants is susceptible. This equation holds when the distribution of the spores from an inoculum plant is uniform. If the uniformity of spore dispersal is not the case, equation (1) may not be right.

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In general, spore dispersal is expressed by

$$y = a\gamma e^{-\alpha d} = \beta e^{-\delta p}, \quad (d = up, \delta = \alpha u) \quad (3)$$

where y is the number of lesions on a plant, a is the initial number of lesions on an inoculum plant, γ is the increasing rate of lesion number on the inoculum plant, α is dispersal gradient, d is the distance from the inoculum plant, and u and p are distance between plants and plant number from the source plant (KIYOSAWA and SHIYOMI 1972).

Using the equation as fundamental method, KIYOSAWA and SHIYOMI (1972) evaluated the effect of mixing of resistant plants on a line of susceptible plants by a simulation, and showed that equation (2) hold when dispersal gradient, α , is little, namely, when spore scattering is approximately uniform. However, the disease dispersal on a line of susceptible plants is not practical. In this paper, the effect of mixing of resistant plants in a population of susceptible plants on a plane is estimated by the use of computer in the Computing Center for Research in Agriculture, Forestry and Fisheries.

Modeling

If disease is dispersed from a point source (an origin) by equation (3), the number of lesions newly formed after one generation on the plant which is located at the coordinate $I, J(y_{IJ}^{(1)})$ is

$$y_{IJ}^{(1)} = ABa\gamma e^{-\alpha \sqrt{I^2 A^2 + J^2 B^2}}, \quad (4)$$

where A and B are the distances between plants on X and Y axes, respectively. The total number of new lesions on the plants in an unlimited area is

$$y^{(1)} = ABa\gamma \sum_{I=-\infty}^{\infty} \sum_{J=-\infty}^{\infty} e^{-\alpha \sqrt{(IA)^2 + (JB)^2}}. \quad (5)$$

In the computer program,

$$y^{(1)} = 4 \sum_{I=1}^M \sum_{J=1}^N y_{IJ}^{(1)} - 2 \left(\sum_{I=1}^M y_{I1}^{(1)} + \sum_{J=1}^N y_{1J}^{(1)} \right) + y_{11}^{(1)} \quad (6)$$

where M and N are the number of plants on X and Y axes, respectively, and y_{11} is the lesion number on the source plant (original point). Taking distance between plants as a unit distance in the case where seedlings were planted in an equidistance for both between rows and between plants, the equation

$$y_{IJ}^{(1)} = a\gamma e^{-\delta \sqrt{I^2 + J^2}} \quad (7)$$

is available for equation (4).

In the second generation, the number of lesions newly formed on the plant IJ is

$$y_{IJ}^{(2)} = \sum_{K=-\infty}^{\infty} \sum_{L=-\infty}^{\infty} y_{KL}^{(1)} \gamma e^{-\delta \sqrt{(K-I)^2 + (L-J)^2}}. \quad (8)$$

In the computer program,

$$y_{IJ}^{(2)} = \gamma \left[\sum_{K=1}^M \sum_{L=1}^N y_{KL}^{(1)} e^{-\delta \sqrt{(K-I)^2 + (L-J)^2}} + \sum_{K=1}^M \sum_{L=2}^N y_{KL}^{(1)} e^{-\delta \sqrt{(K-I)^2 + (L+J-2)^2}} \right]$$

$$\begin{aligned}
& + \sum_{K=2}^M \sum_{L=1}^N y_{KL}^{(1)} e^{-\delta \sqrt{(K+I-2)^2 + (L-J)^2}} \\
& + \sum_{K=2}^M \sum_{L=2}^N y_{KL}^{(1)} e^{-\delta \sqrt{(K+I-2)^2 + (L+J-2)^2}} \Big]. \quad (9)
\end{aligned}$$

The total number of new lesions ($y^{(2)}$) is

$$y^{(2)} = 4 \sum_{I=1}^M \sum_{J=1}^N y_{IJ}^{(2)} - 2 \left(\sum_{I=1}^M y_{I1}^{(2)} + \sum_{J=1}^N y_{1J}^{(2)} \right) + y_{11}^{(2)}. \quad (10)$$

Thus, in the n -th generation,

$$y_{IJ}^{(n)} = \sum_{K=-\infty}^{\infty} \sum_{L=-\infty}^{\infty} y_{KL}^{(n-1)} \gamma e^{-\delta \sqrt{(K-I)^2 + (L-J)^2}}. \quad (11)$$

$$y^{(n)} = 4 \sum_{I=1}^M \sum_{J=1}^N y_{IJ}^{(n)} - 2 \left(\sum_{I=1}^M y_{I1}^{(n)} + \sum_{J=1}^N y_{1J}^{(n)} \right) + y_{11}^{(n)}. \quad (12)$$

The calculation of lesion number when resistant plants were mixed was conducted by calculating only on susceptible plants in the computer program.

Determination of β

An object of this study is to estimate the influence of dispersal gradient on the effect of mixing of resistant plants in the population of susceptible plants. Therefore, it is required that all other conditions, especially the infection rate, are same between all comparisons in each of pure and mixed stands. To obtain the same infection rate, namely the same increase of lesion numbers after each generation, for various δ 's, the volume (V) of the body of revolution obtained by rotating the curve expressed by equation (3) was obtained from equation (3)

$$V = \int_0^\beta \pi x^2 dy = \frac{2\pi\beta}{\delta^2}, \quad (13)$$

where d in equation (3) was replaced with x .

If the volumes of two bodies of revolution with different values of δ (δ and δ') are equal, that is, infection rates do not vary with different δ 's,

$$2\pi\beta'/\delta'^2 = 2\pi\beta/\delta^2, \quad \therefore \beta' = \delta'^2\beta/\delta^2. \quad (14)$$

Thus, β' for various δ 's was calculated setting $\beta = \gamma a = 1$ at $\delta = 1$. Here, a is the initial number of lesions on the point source and 1 was used for a in all the calculations in the paper.

Computation

At first, some calculations were performed by using the program in which equations (11 and 12) were constructed. However, this program requires too long time to simulate the disease dispersal in a large plot. Therefore, further calculations were carried out by computing the lesion number on 1/8 area of a plot shown Fig.1. Total number of lesions

Fig. 1. The relationship of the area which includes plants directly used for the calculation of the number of lesions and the plot in which plants are placed.

The number of lesions on individual plants was counted only on plants marked by ● and the total number of lesions in the plot in each generation was calculated in whole area including ○-marked plants.

newly formed in each generation in the plot is

$$y^{(n)} = 8 \sum_{I=1}^M \sum_{J=1}^N y_{IJ}^{(n)} - 4 \left(\sum_{J=1}^N y_{IJ}^{(n)} + \sum_{I=1}^M y_{II}^{(n)} \right) + y_{11}^{(n)}. \quad (15)$$

Even using this equation, more than ten hours are necessary for the author's program to calculate the disease dispersal in the plot containing 200×200 plants in the computer (HITAC 8000) until the third generation when δ is less than 0.2. Therefore, disease dispersal was computed in the plot containing 97×97 plants. An inoculum plant was located in the center of this plot. The program was executed by giving 1 to a , 1.0, 0.7, 0.5, 0.2, 0.1 or 0.05 to δ in equation (3), 49 to M and N in equation (11) and 3 to generation number.

Disease dispersal in a mixed stand of resistant and susceptible plants was obtained by calculating lesion numbers only on odd-numbered plants in odd-numbered rows and even-numbered plants in even-numbered rows.

The results of calculations are shown in Table 1. In Table 2, the ratios of lesions of the mixed stand and the pure stand in each generation are shown, with similar values obtained in simulations of disease dispersal on a line which is surrounded by resistant plants with the program produced in the previous paper (KIYOSAWA and SHIYOMI 1972).

Table 1. The increase of the numbers of new lesions in pure stand of susceptible plants and in mixed stand of them with resistant plants ($M=N=49$, $a=1$)

δ	Pure stand			Mixed stand (1 : 1)		
	Generation			Generation		
	I	II	III	I	II	III
1.0	6.507	42.342	275.521	3.452	11.917	41.136
0.7	6.361	40.458	257.347	3.250	10.564	34.336
0.5	6.311	39.831	251.405	3.182	10.122	32.203
0.2	6.282	39.392	246.000	3.143	9.861	30.815
0.1	6.090	35.099	193.379	3.045	8.779	24.192
0.05	4.717	18.883	71.762	2.358	4.722	8.974

Table 2. The ratio of the number of new lesions in mixed stand of susceptible and resistant plants in 1 : 1 ratio to one in pure stand of susceptible plants (Mixed/Pure)

δ	Plane model			Line model		
	Generation			Generation		
	I	II	III	I	II	III
1.0	0.530	0.281	0.149	0.607	0.368	0.223
0.7	0.511	0.261	0.133	0.557	0.310	0.172
0.5	0.504	0.254	0.128	0.530	0.281	0.149
0.2	0.500	0.250	0.125	0.505	0.255	0.129
0.1	0.500	0.250	0.125	0.502	0.252	0.126
0.05	0.500	0.250	0.125	0.502	0.252	0.126
Uniform	0.500	0.250	0.125	0.500	0.250	0.125

As indicated in the previous paper (KIYOSAWA and SHIYOMI 1972), the ratio of new lesions in the mixed stand of resistant and susceptible plants and the pure stand of susceptible plants reveals the effect of mixing of resistant plants in susceptible plants on disease increase. The effect of mixing of resistant plants is dependent upon the dispersal gradient, δ , of spores. As shown in Table 2, the ratio in each generation increases as the value of δ increases. This indicates that an increase of dispersal gradient brings a decrease of the effect of mixing of resistant plants. The largest effect of mixing of resistant plants occurs when spores disperse uniformly on all plants, as shown in the last row in Table 2.

In the plane model (PM) used in this paper, the ratio of mixed and pure stands on new-lesion numbers shows a slight increase at $\delta=0.5$. On the other hand, the ratio increases to a relatively large extent at $\delta=0.5$ in the line model (LM) of disease dispersal used in the previous paper (KIYOSAWA and SHIYOMI 1972). This indicates that the inhibition by a large dispersal gradient of the effect of mixing of resistant plants is little as compared with one expected by the line model. It is considered that, when δ is less than 0.5, the relative amounts of lesions in two fields, pure and mixed, are given by

$$\frac{y'}{y_0} = m^n \frac{y}{y_0}, \quad (12)$$

where y and y' are the new-lesion numbers of pure and mixed stands, respectively, after n generations, y_0 is the new-lesion number at the initial stage of infection and m is the proportion of susceptible plants in the mixed stands. Therefore, equation (1) can be given for the relation between infection rates (r_m and r_s) on the mixed and pure stands when disease severity on a plant is not heavy.

Spacing density and effect of mixing of resistant plants

As shown above, the distribution of spores (y) released from a point source is given by the equation

$$y = \beta e^{-\alpha d} = \beta e^{-\delta p}.$$

This distribution gradient may not be largely influenced by the spacing density of plants, although infection rate (multiplication rate) may be greatly affected. In the previous section, it is clarified that the effect of mixing of resistant plants is dependent upon δ of the equation. As $\delta = \alpha u$ and α is constant for each pathogen, the effect of mixing of resistant plants depends upon u , distance between plants. This indicates that the effect of mixing is largely dependent upon the spacing density of plants. That is, the effect of the mixing to inhibit the disease progress is slightly decreased by low spacing density of the host. The decreasing effect appears at a high value of αu (δ) than 0.5. Being lower than this, equation (12) and so equation (1) may be available under the conditions where disease severity is not so heavy, that is, $y = y_0 e^{rt}$ or $dy/dt = ry$ is acceptable.

Flattening of dispersal distribution curve

CAMMACK (1958) reported that the slope of dispersal distribution curve of lesions from

Fig. 2. Comparison between distributions of newly formed lesions calculated with plane model (PM) and line model (LM).

the outside sources. The distribution curves of lesions calculated with the plane dispersal model in this paper are shown in Fig. 2 in semi-log scale with that of the line model. This indicates that the flattening of distribution curve occurs earlier in the plane distribution model than in the line model. Of course, curves or curved surfaces drawn with the plane dispersal model are nearer to the natural conditions. In consideration of contamination from outside sources and the decrease of reproduction rates on heavily diseased plants as reasons for flattening of disease dispersal curve or curved surface, the flattening may occur under the field conditions faster than expected by the simulation with the plane distribution model.

Fig. 3. The relationship of infection rates in terms of lesions newly formed to the logarithm of the proportion of susceptible plants.

○: $\delta=0.2$

●: $\delta=1.0$

the point source flattens near the point source after some vegetative cycles. He attributed the reason to the contamination of spores from outside sources and increasing infection cycles. Kiyosawa and Shiyomi (1972) indicated by the simulation with the line dispersal model that the flattening occurs after a few cycles of vegetative reproduction (generation) without the contamination from

Infection rate and disease dispersal gradient

LEONARD (1969a) showed that the relationship between infection rate based on newly formed lesions and the logarithm of the proportion of susceptible plants in mixed stand is expressed by a straight line as shown in equation (1). Fig. 3 is given for this relationship at $\delta=1.0$ and 0.2 . At $\delta=0.2$, namely near uniform distribution, the relationship is expressed by a straight line, but at $\delta=1.0$ it is curved.

The influence of distribution gradient on the effect of mixing of resistant

Table 3. The ratio of mixed/pure* at the high δ , 1.0, to that in uniform distribution ($M=N=99$, $a=1$)

Proportion of susceptible plants		Generation		
		I	II	III
1/2	High δ	0.530	0.281	0.149
	Uniform	0.500	0.250	0.125
	Ratio**	1.05	1.13	1.19
1/4	High δ	0.306	0.0938	0.0287
	Uniform	0.250	0.0625	0.0156
	Ratio	1.23	1.50	1.84
1/9	High δ	0.196	0.0386	0.00758
	Uniform	0.111	0.0124	0.00137
	Ratio	1.77	3.13	5.53
1/16	High δ	0.196	0.0386	0.00758
	Uniform	0.0625	0.0039	0.000244
	Ratio	2.68	7.18	19.25
1/25	High δ	0.158	0.0251	0.00397
	Uniform	0.0400	0.00160	0.000064
	Ratio	3.96	15.68	62.08
1/36	High δ	0.155	0.0241	0.00375
	Uniform	0.0278	0.000772	0.0000214
	Ratio	5.59	31.27	175.14

*: See Table 2.

**: High δ /Uniform.

Fig. 4. The relationship of infection rates in terms of total lesions to the logarithm of the proportion of susceptible plants

○ : $\delta=0.2$ ● : $\delta=1.0$

new lesions. At higher multiplication rates, the ratio may approach to the ratios in Table 2. The increase of the ratios below $\delta=0.2$ might be dependent upon the fact that a relatively large proportion of spores produced in the plot set up in the computation flies away to the outside of the plot because of the small dispersal gradients.

The infection rates on host mixtures in various proportions of susceptible plants are drawn on the logarithm of the total number of new and old lesions in Fig. 4. In this case

Table 4. The ratio of the total number of new and old lesions in mixed stand of susceptible and resistant plants in 1:1 ratio to one in pure stand of susceptible plants in the plane model

δ	Generation		
	I	II	III
1.0	0.593	0.328	0.177
0.7	0.577	0.310	0.161
0.5	0.572	0.303	0.156
0.2	0.569	0.300	0.153
0.1	0.571	0.304	0.157
0.05	0.587	0.328	0.177

varieties to reduce the infection rate increases with the increase of proportion of resistant plants and advance of generation (Table 3).

The change of the number of lesions newly formed was considered above. The total number of new and old lesions is examined here. The ratio of total number of new and old lesions in the mixed stand to that in the pure stand is shown in Table 4. The ratio is generally higher than that of new lesions and indicates the smallest value near 0.2 of δ . The general increase of total number of lesions as compared with that of new lesions is due to a relatively large number of old lesions in comparison with

the relationships are expressed by curves, and all infection rates are larger than 0. In other words, the effect of mixing of resistant plants to reduce infection rate is slightly reduced by the decrement of the proportion of susceptible plants at the high δ , and the total number of lesions does not decrease, although it is possible that the number of lesions newly formed decreases relatively with the lapse of time under unfavorable conditions or by mixing of resistant plants in a high rate.

Future problems

This study on the simulation of disease dispersal from a point source was begun to obtain much more information of disease dispersion, for example dispersion of disease from many sources, disease dispersion on a field in which resistant and susceptible plants are mixed at random in various ratios, disease dispersion in a larger area, disease dispersion in fields where various varieties were monocultured, etc. However, the time required for calculation is too long to solve these problems. In this study, I was obliged to reduce the size of experiment to 1/32 (the size for setting lesion numbers to 1/8 as shown in Fig.1, and the number of plants in a row to 1/2, thus the total number of plants to 1/4) of prearrangement at the beginning. Particularly, that the size for setting lesion numbers was limited to a half of a quadrant largely decreased the practical utility of this simulation model. This limitation of the size to a half quadrant does not permit the random distribution of lesions in the area, because the distribution obtained by this model should be symmetrical with respect to four lines, $X=1$, $Y=1$, $Y=X$ and $Y=-X$ in Fig.1. Other methods for solution of many parts of the problems considered at the beginning of this study must be devised.

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罹病性個体と抵抗性個体の単独栽培と混合栽培における
病気の伝播のシミュレーションによる比較

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点伝染源からの病原菌胞子の飛散およびそれによる病斑の形成数 y は

$$y = a r e^{-\alpha d} = \beta e^{-\delta p} \quad (d = up, \delta = \alpha u)$$

により表わされる。ここで a は伝染源植物上の病斑数, r は伝染源植物上での病斑数の増加率, d は伝染源からの距離, α は伝播係数, p は伝染源からの植物数, u は植物間距離である。新病斑の形成が毎代この式に従って行なわれるものとして, コンピューターで縦横等間隔に罹病性品種と抵抗性品種を植えた圃場の中央に伝染源をおいた場合の病斑の増え方を調べた。

δ が小さいときは胞子が均一に飛ぶと考えた場合の抵抗性品種の混合による伝染速度を減ずる効果が表われるが, δ が大きい場合にはこの効果は小さくなる。新病斑のみにより計算された伝染速度 r は δ が小さいとき罹病性個体の率の対数の減少に対して直線的に減少するが, δ が大きくなると罹病性個体率の減少によりその効果が落ちる程度が大となり, 直線の下降速度は減ずる (Fig. 3)。すなわち抵抗性品種混合による病害の防除効果は δ が大きい程小さくなる。 $\delta = \alpha u$ のため u (植物間距離) が大きい程抵抗性品種混合による伝染抑制効果は小さくなる。

古い病斑まで含めた全病斑数により伝染速度を計算すると抵抗性品種混合状態での伝染速度は罹病性品種の混合率の対数の減少に対して下降して 0 に漸近する曲線を描きこの下降曲線の低下度は δ が高い程減少する。