The Influence of Resistance Gene Frequencies in Rice Plants on Virulence Gene Frequencies in Blast Fungus Population in Japan

Shigehisa KIYOSAWA*, Hidekazu YAMAGUCHI* and Masao YAMADA*

Abstract

Using the data on race frequencies of blast fungus through Japan obtained by Yamada et al., multiple regression analyses were carried out to get an equation for predicting frequencies for virulence genes in each prefecture in Japan. Resistance gene frequencies in each prefecture was calculated from growing areas of each variety in each prefecture in 16 years from 1961 to 1976. After arcsin transformation of frequencies of resistance genes and the corresponding virulence genes, 1) resistance gene frequencies, 2) the first and second component scores in principal component analysis computed from the resistance gene frequencies in the 16 years, and 3) means and slopes of resistance gene frequencies in each five or six year period when the 16 years were divided into three sections were used as three series of independent variables for a regression equation. The choice of a multiple regression equation from many equations obtained was performed to minimize the prediction sum of squares and to gain a better understanding. The virulence gene frequencies (F) in 1976 were expressed by the equation F=0.14-1.89S2+1.44M1+0.53Da-0.62M12+0.63M1Dk+2.42S2Da, where S2 was the slope showing change of resistance gene frequencies from 1966 to 1971, M1 was the mean of resistance gene frequencies from 1972 to 1976, and Da and Dk were dummy variables for Pi-a and Pi-k resistance gene, respectively.

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Key Words: Oryza sativa, Pyricularia oryzae, prediction, gene frequency, virulence.

Introduction

In the various crops, their disease resistance has been broken down by the occurrence and/or increase of new races of the pathogen in various countries in the world. These increases of virulent races have been suggested to be due to the increase of growing areas of varieties with the same resistance genes.

Quantitative studies on the relationship between resistance gene and virulence gene frequencies were begun by theoretical studies rather than by field survey. Mode studied the mutual influence between resistance gene frequency and virulence gene frequency by using differential equations.

Leonard formulated the relationship of infection rate on mixed stand of susceptible
and resistant plants to that on a pure stand of susceptible plants, devised a method to compare the fitness of fungus strains, and on the basis of these results examined theoretically the condition where the equilibrium of frequencies of pathogen genotypes (races) is reached.

Later, Leonard\(^{17}\) indicated more definitely the equilibrium conditions in consideration of field resistance of the host genotypes, aggressiveness of the pathogen genotypes, the effectiveness of resistance gene and the advantage of virulent race on hosts with the corresponding gene for resistance. The Leonard’s results were discussed by Sedcole\(^{26}\) and Leonard and Czochor\(^{18}\).

Kiyosawa and Yabuki\(^{13}\) made a model on the change of frequencies of two virulence genes which correspond to the resistance genes, A and B, depending on frequencies of the resistance genes. They incorporated the difference of multiplication rate (fitness) between genotypes of the pathogen in their model. Using the same model, Kiyosawa et al.\(^{12}\) calculated the optimal proportion of resistant varieties in the mixture to minimize the average relative disease severity, from the standpoint that virulence gene frequencies themselves do not determine directly disease severity. Furthermore, Kiyosawa\(^{7}\) investigated the utility value of virulence analysis which was proposed as a method to know the presence of selection by the frequency of a host gene.

There are very few studies on the relationship between resistance gene frequency and race (pathogen gene) frequency in farmer’s fields. Kiyosawa et al.\(^{10}\) studied the relationship between percent growing area of \(Pi-k\) varieties (varieties with \(Pi-k\)) and the degree of their breakdown by multiple regression analysis in Niigata Prefecture, and obtained various regression equations. In the study, they used breakdown indices [the ratio of percent infected areas of \(Pi-k\) variety growing field to that of \(Pi-k\) variety (variety without \(Pi-k\)) growing field] in 1966 as a dependent variable, and percent growing area of \(Pi-k\) varieties in 1961 to 1965 and the breakdown index of \(Pi-k\) varieties in 1965 as an independent variable. When all these variables were used in a multiple regression analysis, relatively high correlation coefficients adjusted by the degree of freedom were obtained. These ranged from 0.70 to 0.82. Thus, the obtained multiple regression equations may show significant influence of \(Pi-k\) gene frequency in the farmer’s field on the breakdown of \(Pi-k\) varieties. These equations are, however, not easily understood, because percent growing areas which have positive correlations to the breakdown indices often take negative partial regression coefficients. This is due to high correlations of percent growing areas of \(Pi-k\) varieties between years.

For eliminating this deficiency, Matsumoto et al.\(^{20}\) used the first component scores of principal component analysis for percent growing areas of resistant varieties during some years in Nagano Prefecture as independent variables, and obtained a multiple regression equation with a high contribution ratio adjusted by the degrees of freedom.

In the present paper, the relationship between percent growing areas of resistant varieties (resistance gene frequencies) and frequencies of virulence gene in forty three prefectures of Japan is examined by a multiple regression analysis.

**The used data**

Frequencies of races of blast fungus in each prefecture in Japan in 1976 were
examined by Yamada et al.\textsuperscript{33)} Blast fungus races were identified by using nine differential varieties established by Yamada et al.\textsuperscript{34)} For these varieties have single genes for blast resistance, frequencies of genotypes or genes for virulence can be determined from the frequencies of races.

Percent growing area of each genotype in each prefecture was examined during 16 years from 1961 to 1976 by "Growing Areas of Each Rice Variety" (Food Agency). Genotypes for blast resistance of these leading varieties were determined on the basis of the lists of Kiyosawa et al.\textsuperscript{8,9)}, and partially of Yamada's unpublished data. The genotypes for blast resistance of varieties in 79.6\%, 86.4\% and 88.2\% of growing areas in prefectures were determined in 1966, 1971 and 1976, respectively, on an average.

Frequencies of virulence genes, $Av-a^+$, $Av-i^+$, $Av-k^+$, $Av-ta^+$ and $Av-ta2^+$, and frequencies of resistance genes, $Pi-a$, $Pi-i$, $Pi-k$, $Pi-ta$ and $Pi-ta^2$, were calculated from the above mentioned data.

Furthermore, average of percent infected areas in prefectures during six years from 1971 to 1976 were calculated from "Sakumotsu Tokei (Crop Statistics)".

**Correlation coefficient between variables**

After an arcsin transformation was made, correlation coefficients between percent growing areas of 16 years were calculated.

Correlation coefficients between resistance gene frequency in each year and virulence gene frequency (Table 1A and 1B) showed that the nearer to 1976 when fungus is isolated, the larger the correlations between $Pi-i$ and $Av-i^+$ frequencies and between $Pi-ta+Pi-ta^2$ and $Av-ta^+$ frequencies. For $Pi-a$ to $Av-a^+$ gene pair, similar correlation coefficients were got during ten years from 1967 to 1976. For $Pi-k$ to $Av-k^+$ gene pair, the highest correlation coefficient was noted in 1974. The difference between the $Pi-a$ and $Av-a^+$ gene pair and other gene pairs seems to concern with relatively small change of resistance gene frequencies during the period from 1957 to 1976 as compared with other gene pairs.

Average percent infected area of six years from 1971 to 1976 did not show regular significant correlation coefficients with percent growing areas of each of four resistant variety groups and four virulence gene frequencies.

**Multiple regression analysis**

*Multiple regression analysis in which resistance gene frequencies themselves are used*

Resistance gene frequencies of sixteen years were used in the analysis with percent infected areas as independent variables.

When all variables (resistance gene frequencies and percent infected areas) were used as independent variables, relatively high value of contribution ratio adjusted by degrees of freedom ($R^2$) was obtained (Table 2). However, in this case, the deficiency that variables correlating positively with virulence gene frequencies often show negative partial regression coefficients occurs. This leads to the difficulty to understand the obtained regression equations.
To avoid this deficiency, forward, stepwise and backward methods to select effective variable set in regression analysis were used. By these analyses, \( R^2 \) values similar to those shown above were obtained for every resistance-gene and virulence-gene pair.

### Table 1A. Correlation coefficients between resistance gene frequencies in each year, average of percent infected areas from 1971 to 1976 (IA), and virulence gene frequencies in 1976 (VGF)

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</tbody>
</table>

Above diagonal: Relationship between \( Pi-a \) gene frequencies and \( Av-a^+ \) gene frequencies. Below diagonal: Relationship between \( Pi-i \) gene frequencies and \( Av-i^+ \) gene frequencies.

### Table 1B. Correlation coefficients between resistance gene frequencies in each year, average of percent infected areas from 1971 to 1976 (IA), and virulence gene frequencies in 1976 (VGF)

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</table>

Above diagonal: Relationship between \( Pi-k \) gene frequencies and \( Av-k^+ \) gene frequencies. Below diagonal: Relationship between \( Pi-ta+Pi-ta^2 \) gene frequencies and \( Av-ta^+ \) gene frequencies. Correlation coefficients could not be calculated because \( Pi-ta \) and \( Pi-ta^2 \) gene frequencies were zero in all prefectures in 1967.
Table 2. Contribution ratios adjusted by the degrees of freedom obtained in regression
analysis on relationships between percent growing area of resistant varieties
and frequency of virulent races

<table>
<thead>
<tr>
<th>Independent variable set</th>
<th>Resistance-gene and virulence-gene pair</th>
<th>Contribution ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>All variables(^a)</td>
<td>(Pi-a; Av-a^<em>), (Pi-i; Av-i^</em>), (Pi-k; Av-k^*)(^{Pi-ta^+Pi-ta^+Av-ta^+}), (Pi-ta^+Pi-ta^+Av-ta^+)</td>
<td>61.90</td>
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<tr>
<td>Forward(^b)</td>
<td>(70.76(7,16)), (80.44(14)), (77.43(15)), (59.73(8,9,10,14))</td>
<td>60.12(11)</td>
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<tr>
<td>Stepwise</td>
<td>(68.27(16)), (80.44(14)), (77.43(15)), (60.29(11))</td>
<td>60.12(11)</td>
</tr>
<tr>
<td>Backward</td>
<td>(67.19(7,8,9,11,14,15)), (67.19(7,8,9,11,14,15)), (77.43(15)), (59.73(8,11,13))</td>
<td>67.19(7,8,9,11,14,15)</td>
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</tbody>
</table>

\(^a\): All variables including resistance gene frequencies during 1961 to 1976 in arcsin transformation were used as independent variables.

\(^b\): Forward selection method.

\(^c\): For example, 11 and 14 show resistance gene frequencies in arcsin transformation in 1971 and 1974, respectively.

\(^d\): \(Z_{15,1}\) and \(Z_{15,1}\) show first component score obtained from principal component analysis of resistance gene frequency in five years from 1972 to 1976 and from 1967 to 1971, respectively. \(Z_{10,2}\) is the second component score in ten years from 1967 to 1976. \(Z_{16,1}\) is the first component score in 16 years from 1961 to 1976.

\(^e\): Using the data during nine years, because the calculation of principal component analysis is not possible for the variance before 1967 becomes zero.

**Multiple regression analyses in which the first and second component scores in principal component analysis were used**

As mentioned above, relatively high \(R^2\) values were obtained when selected variables were used as independent variables as it was. However, the selected variables were different due to gene pairs. In the \(Pi-a\) and \(Av-a^*\) gene pair, resistance gene frequency in 1971 was chosen. This difference does not necessarily indicate a difference between gene pairs in years when the virulence gene frequency is particularly influenced.

Matsumoto et al.\(^{20}\) obtained a multiple regression equation to predict the virulence gene frequencies in blast fungus populations in Nagano Prefecture, by using the first component scores in the principal component analysis as a variable which represent the percent growing areas of resistant varieties, that is resistance gene frequency, for the past five years. This is the first report that is attempted to predict the change of virulence gene frequencies from the frequencies of the corresponding resistance genes based on field survey.

Frequencies for each of five resistance genes, \(Pi-a\), \(Pi-i\), \(Pi-k\), \(Pa-ta\) and \(Pi-ta^2\), were analyzed by the principal component analysis using the computer programme made by Suzuki\(^{20}\) after arcsin transformation. The variation of resistance gene frequencies can be explained up to 95% and 99% by the first and second scores, respectively, for
Pi-a varieties. Therefore, the two component scores were used in multiple regression analyses as independent variables to explain the prefectural frequencies of virulence genes, Av-a+, Av-i+, Av-k+, Av-ta+ and Av-ta2+.

The first component is related to the mean of resistance gene frequencies and the second one to the slope of the change of resistance gene frequencies during the years.

The first and second component scores were used for regression analysis with average percent infected areas for the six years. Calculations were carried out using the programme made by Hirosaki and Kobayashi\(^4\) to compute a prediction sum of squares (PSS). In this (PSS) method which was proposed by Allen\(^1\), an estimate for \(a\)-th sample (prefecture) is calculated from the next equation.

\[
y_{p,a} = b_{0,a} + b_{1,a}x_{1,a} + \ldots + b_{i,a}x_{i,a} + \ldots + b_{p,a}x_{p,a},
\]

where \(y_{p,a}\) is an estimate of \(a\)-th sample when the number of independent variables is \(p\), \(x_{i,a}\) is an observed value of \(a\)-th sample for \(i\)-th variable, \(b_{0,a}\) is constant, and \(b_{i,a}\) to \(b_{p,a}\) are partial regression coefficients in multiple regression equation computed with all samples but \(a\)-th one. The sum of squares of the difference between this estimate and its observed value (\(y_{a}\)) for all samples,

\[
PSS_p = \sum_{a=1}^{n}(y_{a} - y_{p,a})^2,
\]

are calculated for all combinations of independent variables. From these calculations, a combination of variables which had the smallest PSS was selected as shown in Table 2. When the first and second component scores calculated from resistance gene frequencies for the past 16 years (\(Z_{16,1}\) and \(Z_{16,2}\)) were used together, contribution ratio adjusted by the degrees of freedom (\(R^2\)) were 30.61, 45.05, 62.01 and -1.33 for Pi-a and Av-a+, Pi-i and Av-i+, Pi-k and Av-k+, and Pi-ta and Av-ta+ pairs, respectively. When \(Z_{10,1}\) and \(Z_{16,2}\) were used, \(Z_{16,1}\) is mainly responsible for the change of virulence gene frequencies. The scores obtained from the past ten years and five years showed also similar tendencies. In both cases, Pi-ta\(^a\) and Av-ta2+ pair was also examined with very low contribution ratios adjusted by the degrees of freedom. As a whole, Pi-ta and Av-ta+, and Pi-ta\(^a\) and Av-ta2+ pairs showed very low contribution ratios. This is considered to be dependent on the fact that both genes are on the same locus and all isolates virulent to Pi-ta\(^a\) varieties are virulent to Pi-ta varieties. Kiyosawa\(^6\) explained the fact as Pi-ta has only a part of Pi-ta\(^2\).

Therefore, the sum of frequencies of Pi-ta and Pi-ta\(^a\) was used as an independent variable for frequencies of Av-ta+ and Av-ta2+. The contribution ratios obtained for each virulence gene frequency ranged from 54.43 to 58.23 and 6.34 to 26.12 for frequencies of Av-ta+ and Av-ta2+ respectively, in six analyses (independent variable sets, 1, 4-8 in Table 2) using \(Z_{16,1}\), \(Z_{10,1}\) and \(Z_{5,1}\) with their \(Z_i's\). The low ratios for Av-ta2+ seem to be due to the fact that most of isolates attacking Pi-ta varieties cannot attack Pi-ta\(^a\) varieties, that is, these isolates do not carry Av-ta2+.

Contribution ratios obtained above are not necessarily adequately high. Previously, Matsumoto et al.\(^20\) suggested that percent growing area of resistant varieties before more than five years influenced the virulence gene frequencies. Taking this suggestion into consideration, ten years before 1976 were divided into two sections and the effect
of the former five years on the virulence gene frequencies was examined. The results are shown in Table 2 in terms of contribution ratio adjusted by the degree of freedom \((R^*\text{2})\). The scores calculated from resistance gene frequencies during the former five years \((Z_{5,1}^*\text{,}1\text{ and }Z_{5,2}^*\text{,}2)\) did not give a large influence on virulence gene frequencies, as, for example, shown by low \(R^*\text{2's}\) for \(Z_{5,1}^*\text{,}1\) and small differences between \(Z_{5,1}^*\text{,}1\) and \(Z_{5,1}^*\text{,}1+Z_{5,2}^*\text{,}1\) (Table 2).

**Multiple regression analyses when the change of resistance gene frequencies for 16 years were divided into some components**

It is not easy to know intuitively the tendency of the change of resistance gene frequencies from the component scores of principal component analysis. In the next analysis, virulence gene frequencies in 1976 were analyzed by dividing the change of resistance gene frequencies during 16 years before collection of blast fungus to six components, means \((M)\) and slopes \((S)\) of resistance gene frequencies during 1961 to 1966, 1967 to 1971 and 1972 to 1976 \((M_3, S_3, M_2, S_2, M_1\text{ and }S_1, \text{ respectively})\).

As ALLPSS in which PSS's are computed from all possible combinations among all variables cannot be calculated with the Hirosaki-Kobayashi's programme \(^4\), because the number of variables in the present case exceeds the limitation in the programme, variables were selected by STEPPSS. The STEPPSS is carried out as follows. 1) Some variables are selected as an initial set and PSS is calculated from them. 2) One of remaining independent variables is added and PSS is calculated. This calculation is carried out for all of remaining variables and if the lowest PSS is smaller than PSS of the previous set, a variable showing the lowest PSS is added to the previous set of variables. 3) In the similar way to the second step one variable is removed to get a PSS less than PSS of previous set. 4) The process of addition and removal of a variable is alternatively repeated to obtain the minimum PSS. 5) When a less PSS is not found, the final set of variables is the objective one.

Sets of variables thus obtained were partially different due to initial set of variables. Among them the set of variables with the lowest PSS was chosen: \(S_5, M_1, D_3, D_4, D_6, M_7, M_3D_4, M_3D_5, S_2D_4, S_4D_7\text{ and }M_3^2D_4\), where \(D_4, D_7\text{ and }D_8\) are dummy variables given to \(Pi-a, Pi-i\) and \(Pi-k\), respectively. A slight difference of PSS is not considered to be significant biologically or statistically. Therefore, a variable showing a negligible increase of PSS by removing it from variables in the set of the lowest PSS was removed. This process was repeated and five variables were removed in the order shown in Fig. 1. There is no large change till removing \(S_2D_4\). Accordingly, variables in left-side of \(D_1\) were removed from the set of variables.
variables selected based on PSS, and the following regression equation was obtained.

\[
F = 0.14 - 1.89S_2 + 1.44M_1 + 0.53Da - 0.62M_1^2 + 0.63M_1D_k + 2.42S_2D_a. \quad (R^2 = 90.79)
\]

Giving 1 and 0 to dummy variables, \(D_a\) and \(D_k\), respectively, a regression equation to predict \(Av-a^+\) gene frequency is obtained. In the same way, regression equations to predict \(Av-i^+\), \(Av-k^+\) and \(Av-ta^+\) gene frequencies are obtained by giving 0 and 0; 0 and 1; 0 and 0 to the dummy variables, respectively. Hence,

\[
F_a = 0.67 + 0.53S_2 + 1.44M_1 - 0.62M_1^2, \quad (R^2 = 31.1)
\]

\[
F_i = 0.14 - 1.89S_2 + 1.44M_1 - 0.62M_1^2, \quad (R^2 = 41.0)
\]

\[
F_k = 0.14 - 1.89S_2 + 2.07M_1 - 0.62M_1^2, \quad (R^2 = 63.0)
\]

and \(F_{ta+ta^2} = 0.14 - 1.89S_2 + 1.44M_1 - 0.62M_1^2. \quad (R^2 = 46.0)\)

To compare these resistance-gene to virulence-gene relationships, upper equations were transferred to

\[
F_a = 1.51 + 0.53S_2 - 0.62(M_1 - 1.16)^2,
\]

\[
F_i = 0.98 - 1.89S_2 - 0.62(M_1 - 1.16)^2,
\]

\[
F_k = 1.87 - 1.89S_2 - 0.62(M_1 - 1.67)^2,
\]

and \(F_{ta+ta^2} = 0.98 - 1.89S_2 - 0.62(M_1 - 1.16)^2\).

These equations indicate the followings if ignoring the \(S_2\) member, as shown in Fig. 2. The \(Av-i^+\) and \(Av-ta^+\) gene frequencies have curves similar to each other on \(Pi-i\) and \(Pi-ta+Pi-ta^2\) gene frequencies. The \(Av-a^+\) gene frequencies show a similar curve to \(Av-i^+\) and \(Av-ta^+\) curves at a higher level than the latter two. The curve of \(Av-k^+\) on \(Pi-k\) shows a sharper slope than other three. This is based on the difference of axes between these quadratic curves as shown in the above equations.

The relationship between \(Av-a^+\) and \(Av-i^+\) curves is similar to that between curves of \(Av-a^+\) on \(Pi-a\) and \(Av-i^+\) on \(Pi-i\) in Nagano Prefecture. The difference between two curves was explained by longer past record of use of \(Pi-a\) gene and higher field resistance of the \(Pi-i\) variety\(^{20}\). Similar reasons may be applied also to resistance gene and virulence gene relationship of both gene pairs through Japan, as many of \(Pi-i\) varieties, especially Todoroki-wase\(^3\) and a major one of \(Pi-ta^2\) varieties, Reiho\(^28\), have high field resistance. A sharper slope observed for \(Av-k^+\) gene frequencies on \(Pi-k\) gene frequencies may be explained by high field susceptibility of \(Pi-k\) varieties grown in the field.
The above comparison was performed by ignoring the $S_2$ term. Practically, the $S_2$ term has a large effect on virulence gene frequencies as shown in Fig. 1. This indicates that resistance gene frequencies in six to ten years before the examination of virulence gene frequencies play a role in virulence gene frequencies. The difference found between gene pairs suggests that the degree of influence differs between gene pairs.

**Discussion**

There are many observations in which the influence of growing area of resistant varieties on the race frequencies are qualitatively shown, but little quantitative studies are reported, especially on causal relationship of frequency of virulent race and resistant variety. To predict the longevity of disease resistance, it is essential to know causal relationship between percent growing areas of resistant varieties and frequencies of virulent races.

Matsumoto *et al.* analyzed the relationship between percent growing area of resistant rice varieties with $Pi$-a or $Pi$-i resistance gene for blast disease and frequencies of virulent races in terms of gene frequencies in Nagano Prefecture. In this analysis, they used resistance gene frequencies for five years before the year when the fungus is isolated. To avoid that some of variables correlating positively with dependent variable have negative partial regression coefficient, the first component scores obtained from the principal component analysis were employed as independent variables, with a high contribution ratio adjusted by the degrees of freedom.

In the present study, however, high $R^2$ values were not always obtained when the first and second component scores representing resistance gene frequencies during 16 years before 1976 in data through Japan were used as independent variables to explain the virulence gene frequencies in 1976. Therefore, the method in which resistance gene frequencies during the 16 years were divided into three sections and their means and slopes were used as independent variables seems to be useful, because these independent variables are easier to understand than component scores of them.

The difference between resistance-gene and virulence-gene pairs was analyzed using dummy variables given to each gene. Thus, curves characteristic for each gene pair were obtained, with relatively high contribution ratios adjusted by the degrees of freedom.

The genic difference of the curves of virulence gene frequencies on resistance gene frequencies was explained by the difference of resistance gene frequencies, that is growing areas of the resistant varieties including gene in question before examined years and the difference of field resistance in major varieties with resistance gene in question, in the present and previous studies. However, this is a circumstantial evidence, and many experiments are required to conclude it. Many investigations in this kind will be required to establish a method to predict virulence gene frequency affected by resistance gene frequency.

The mean of percent infected areas during 1971 to 1976 was used as an independent variable, as the incidence and severity were considered to influence the increase of virulence gene frequencies or diversity of gene constitution in fungus population.
Multiple regression analysis led almost always to removal of this factor from multiple regression equations, indicating that the disease severity did not play an important role in virulence gene frequencies. This does not always mean that disease severity does not at all play a role in virulence gene frequencies. Collection density of fungus isolates in the used data is too small to know the influence of minor factors influencing virulence gene frequencies including disease severity, wind dispersal from neighboring prefecture and so on. A systematic study in a larger scale is expected to know the influence of various factors on virulence gene frequencies more delicately.

Literature Cited


和文摘要
日本におけるイネ病原性遺伝子の病原性遺伝子頻度
に及ぼす種植体中の抗性遺伝子頻度の影響

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日本におけるイネ病原性遺伝子頻度に関する山田らの調査成績を用いて、それぞれの県の病原性遺伝子頻度を予測するための式をうるために重回帰分析を行った。それぞれの県における抵抗性遺伝子頻度は、1961年から1976年までの16年間のそれぞれの品種の作付面積、「米穀の品種別作付状況」（食糧庁）によるから計算した。抵抗性遺伝子（Pi-a, Pi-i, Pi-k, Pi-ta, Pi-ta'）の頻度と、それに対応する病原性遺伝子（Ar-a*, Ar-i*, Ar-k*, Ar-ta*, Ar-ta2*）の頻度を arcsin 変換した後、1976年の病原性遺伝子頻度を従属変数とし、1）それぞれの抵抗性遺伝子頻度と、2）16年間の抵抗性遺伝子頻度から主成分分析法によってえた第1主成分と第2主成分、および 3）16年を 5, 5, 6 年の 3 つに分けたときのそれぞれの期間中の抵抗性遺伝子頻度の平均値とそれらの変化の勾配を独立変数として用いた 3 つの重回帰分析を行った。いずれも予測値の差を最少にするような変数を選んで 3 つの方法を比較し、最後の方法、すなわち 16 年間を 3 つに分けそれぞれの期間の抵抗性遺伝子頻度の平均値（Mi, Mi, Mi）と変化の勾配（Si, Si, Si）を独立変数とした場合に、自由度で調整した寄与率が高く、また理解し易いという点でも利用価値が高いため重回帰式がえられた。1976年の病原性遺伝子頻度（F）は F = 0.14 – 1.89S1 + 1.44M1 + 0.53D1 + 0.62M12 + 0.63M1Dk + 2.42S2Da で表わされた。ここで、S1 は1967年から1971年までの 5 年間の抵抗性遺伝子頻度の変化の勾配、M1 は1972年から1976年までの抵抗性遺伝子頻度の平均値、D1, D2 はそれぞれ Pi-a 遺伝子、Pi-k 遺伝子に与えられた dummy 変数である。