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Quantitative genetic analysis of biotypes of the brown planthopper *Nilaparvata lugens*: heritability of virulence to resistant rice varieties

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Abstract

The virulence of *Nilaparvata lugens* Stål (Homoptera: Delphacidae) to a resistant variety of rice, *Oryza sativa* L., is suggested to be under polygenic control. To determine whether the virulence of *N. lugens* is a quantitative character that has continuous distribution or a threshold character that has a few discontinuous phenotypic forms and is determined by some underlying continuous variable, I examined the frequency distribution of honeydew excretion that has been used as a measure of ability of attacking the rice plants, and the relationship between honeydew excretion and the oviposition rate of *N. lugens* females using a *japonica* rice line Saikai 190 which has a resistance gene *Bph 1*. The frequency distribution in honeydew excretion significantly differed from the normal distribution, but two distributions below and above 10 mg honeydew excretion for two days did not significantly deviate from normality, suggesting a bimodal distribution. There were significant differences in the proportion of females ovipositing and the number of eggs deposited between the female groups that excreted 0–10 mg honeydew and more than 20 mg honeydew. Within these female groups, however, the reproductive performances were not different. Thus, virulence of *N. lugens* can be analyzed as a threshold character that has two distinct phenotypes, virulent and avirulent. I estimated the heritabilities of the virulence of *N. lugens* by parent-offspring regression using the percentage of virulent females in a full-sib family. The estimates of heritability were 0.41 on Saikai 190 and 0.55 on ASD7, carrying the *bph 2* gene. These results show that the *N. lugens* population has substantial genetic variation in virulence. The regression coefficients on female and male parents were similar, suggesting similar genetic contributions by both parents. When the parental families were examined on Saikai 190 and the progeny on ASD7, the regression coefficient approached zero. There may be a low genetic correlation between virulences to the two varieties.

Introduction

The brown planthopper (BPH), *Nilaparvata lugens*, is one of most notorious insect pests of rice in temperate and tropical Asia (Dyck & Thomas, 1979). Varietal resistance of rice to BPH infestation was found, and the genes conferring the resistance were identified (Pathak et al., 1969; Athwal et al., 1971; Lakshminarayana & Khush, 1977). Commercial rice varieties carrying the resistance genes *Bph 1* and *bph 2* have been bred and released since the 1970s, but the resistance was broken down by occurrence of biotypes virulent to the previously resistant varieties (for review see Sogawa, 1982; Gallagher et al., 1994). Three biotypes of BPH, designated biotypes 1, 2 and 3, can be identified by their ability to feed and infest rice varieties with different resistance genes (International Rice Research Institute, 1976). The population or the individual that cannot infest any rice variety with the resistance genes *Bph 1* and *bph 2* is defined as biotype 1, while the population/individual that can attack the varieties with *Bph 1* and *bph 2* are called biotype 2 and biotype 3, respectively. In Southeast Asia, the BPH populations shifted from biotype 1 to biotype 2 in the 1970s (Feuer,

1976; Mochida et al., 1977; Stapley et al., 1979), and at present, they comprise a complex of biotypes 2 and 3 (Medrano & Heinrichs, 1985; Sogawa et al., 1987; Huynh & Nhung, 1988). The BPH populations immigrating into Japan have been changing from biotype 1 to biotype 2 since 1988–1990 (Sogawa, 1992), and this trend has been also found in China and North Vietnam (Yu et al., 1991; Thuat et al., 1992; Zhang et al., 1995).

The BPH-resistance in rice is under monogenic control (Athwal et al., 1971; Lakshminarayana & Khush, 1977). The biotypes of some insect pests are also controlled by major genes (Hatchett & Gallun, 1970; Gallun, 1978; Puterka & Peters, 1989). The genetics of the BPH biotypes was investigated by crossing experimentally selected biotypes (Cheng & Chang, 1979; Sogawa, 1980, 1981b; Ito & Kisimoto, 1981; den Hollander & Pathak, 1981). Their results showed that the virulence of F_1 progeny from the crossing of biotype 1 $\times$ biotype 2 and biotype 1 $\times$ biotype 3 was intermediate between the parental phenotypes or similar to the biotype 1 phenotype. The virulence of F_2 and backcross exhibited no distributions predicted by a simple Mendelian model but rather similar to the F_1 phenotypes. From these results we can conclude that the biotype or virulence of BPH is not monogenically inherited but should be under polygenic control (Sogawa, 1980; den Hollander & Pathak, 1981; Roderick, 1994). Sezer & Butlin (1998) carried out a genetic analysis of two BPH populations, one feeding on the cultivated rice *Oryza sativa* and the other feeding on a weed grass *Leersia hexandra*, and suggest that the difference in host adaptation is controlled by a few loci. Thus, to investigate the genetics of virulence in BPH, we should use quantitative genetic methods which have not been conducted so far.

To utilize the resistance in plants for insect pest control, we have to understand the durability of resistance in plants and the potential and genetic variation of pests to overcome the resistance and to predict the evolution of host adaptation in pests (Roderick, 1994). For polygenic characters, the genetic variation of a character can be assessed by determining heritability, and the evolution rate of the character can be predicted from the heritability and the selection pressure on the character (Falconer, 1989). Phenotypes of some polygenic characters vary continuously, and these characters are called quantitative characters (Falconer, 1989). Other characters exhibit discontinuous phenotypes but are not inherited in a simple Mendelian manner (Falconer, 1989; Roff, 1996, 1997). The inheritance of these discontinuous characters is explained by the threshold model (Falconer, 1989). The model states that the phenotypic expression of the character is determined by some underlying variable, called the liability (Falconer, 1989), that is continuously distributed. When the underlying variable is below a particular value, the threshold, the individual has one phenotypic form, whereas when it exceeds the threshold, the individual exhibits the alternate form. Many examples of discontinuous characters that fit the threshold model have been documented (Roff, 1996, 1997). This paper examines whether the virulence of BPH to a resistant rice variety can be measured as a quantitative character or a threshold character, and based on these results, estimates the heritabilities of virulence to rice varieties carrying the *Bph 1* or *bph 2* gene.

Materials and methods

Insects. The BPH used in this study were derived from ca. 300 females which were collected in September 1991, from a *japonica* rice variety Reiho, at paddy fields in Kumamoto. The Reiho has no known BPH-resistance genes, and the BPH populations immigrating into Japan well reproduce on Reiho in the field and the laboratory. The BPH have been reared on the Reiho seedlings in plastic containers (11 cm L $\times$ 12 cm W $\times$ 18 cm H) and maintained continuously for 12 to 18 months (ca. 13 to 20 generations). The rearing of insects and the experiments described below were carried out in the laboratory controlled at 25 °C, L16:D8.

Determination of the character to be measured. The feeding activity and virulence of individual planthoppers can be evaluated by measuring honeydew excretion of females (Sogawa & Pathak, 1970; Paguia et al., 1980), and many studies have used this measure for virulence tests in BPH. I examined whether the honeydew excretion can be used as a quantitative character that quantifies virulence. Since the frequency distributions of most quantitative characters approximate normal curves when their measurements are transformed to an appropriate scale if necessary (Falconer, 1989), I examined the frequency distribution of honeydew excretion of BPH on a resistant variety. If the honeydew excretion exactly quantifies the virulence or host adaptation to a rice variety, it will correlate with the traits that affect the fitness of planthoppers. Hence, I analyzed the relationships of honeydew excretion

to body weight gain and oviposition rate. These experiments were carried out using a *japonica* rice line Saikai 190 having a resistance gene *Bph 1* that was originally derived from an *indica* rice variety Mudgo. I measured the honeydew excretion by the parafilm sachet method (Pathak et al., 1982). Preliminary experiments showed that virgin brachypterous females of BPH excreted much honeydew for two or three days after emergence and thereafter reduced excretion. It is, therefore, convenient to measure the honeydew excretion for two days after emergence.

The experiment on weight gain was conducted in September 1992. A newly emerging brachypterous female, up to 24 h old, was anesthetized with carbon dioxide and weighed with a microbalance to the nearest 1 μg. Then the female was confined in a parafilm sachet (2 cm L $\times$ 2 cm W) attached to the leaf sheath of 6- to 7-week-old rice plant in the tillering stage. After two days, the amount of honeydew excreted was estimated by weighing the parafilm sachet before and after blotting honeydew to dry it, and the female was weighed.

The experiment on oviposition rate was conducted in March 1993. After the honeydew excretion of a newly emerging brachypterous female was measured for two days, the female was housed in a test tube (3 cm D $\times$ 20 cm L) with a male for mating. The test tube contained a cut leaf sheath of Saikai 190, and the female, if virulent, could feed and oviposit on it. After two days, the number of eggs deposited was counted. Then the female was enclosed again in a parafilm sachet which was larger (4 cm L $\times$ 2 cm W) to provide an oviposition site. Every two days the female was transferred to a new sachet, and the number of eggs deposited and the honeydew excretion were measured. Since brachypterous females ceased to lay fertilized eggs ca. 10 to 15 days after copulation and mated again, the first copulation contributing 75% of net reproductive rate (Oh, 1979), I carried out the oviposition experiment for ten days including the mating period in a test tube. I conducted the same oviposition experiment using Reiho to compare the reproductive performance of planthoppers between varieties to which the BPH population was well and not well adapted.

Estimation of heritability of virulence. The above-mentioned experiments showed that the virulence of BPH to a resistant variety was a threshold character exhibiting two distinct phenotypes, virulent and avirulent (see Results). The heritability of the threshold character can not be estimated by usual methods for the continuous characters that assume the normal distributions, because the phenotypes of individuals do not have continuous values but are classified into a few, usually two, types. However, we can apply an analogous approach by using a family value, i.e., a proportion of individuals exhibiting one phenotype in a sib, in place of an individual's value (Roff, 1986, 1997).

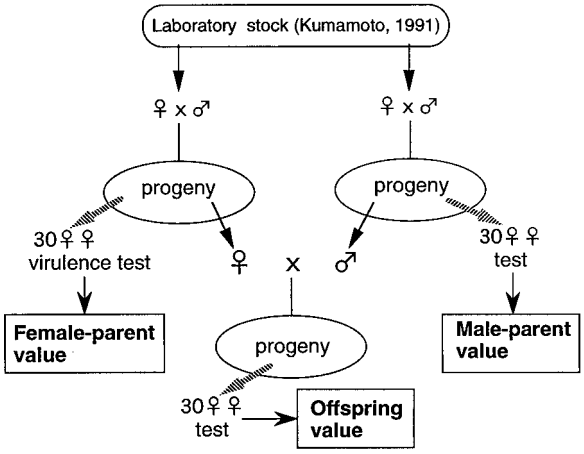

Figure 1. Experimental design for estimation of heritability of virulence in *N. lugens* by using the family values.

The experimental design is shown in Figure 1. A newly emerging female and a male were chosen at random from the laboratory stock, and were housed in a cage containing the Reiho seedlings for mating and oviposition. Newly hatched nymphs were reared on the Reiho seedlings covered with a plastic cylindrical cage (5.5 cm D $\times$ 20 cm H). The progeny from each cross were reared in two cages, each cage starting with ca. 50 nymphs. In F_1 generation, a female and a male from the different families were crossed and their progeny were reared. I produced up to F_4 generation using this procedure.

Since the virulence of BPH is a threshold character, we need only to identify an individual as either virulent or avirulent. When a female consumes sufficient food, its abdomen becomes swollen. Hence, I could easily categorize the virulence of a female by observing the appearance of its abdomen (see Results). I used only females for the virulence test, since the virulence of males cannot be determined by this method. The newly emerging brachypterous females which had slender abdomina were released on test plants of 6- to 7-week-old for two days, and their abdomina were observed. Thirty females in each family were examined

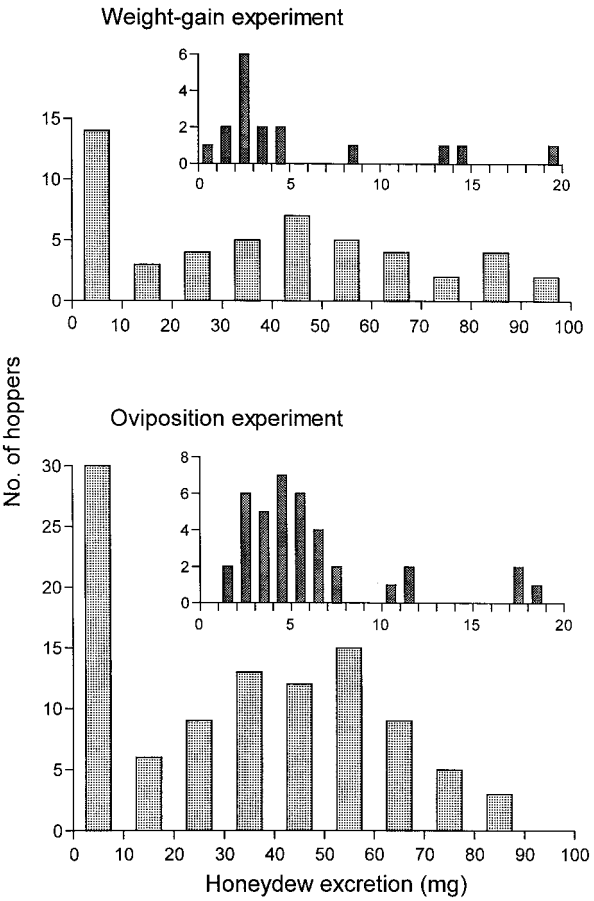

Figure 2. Frequency distributions of honeydew excretion in *N. lugens* females on Saikai 190 for two days in the weight-gain and oviposition experiments. The smaller histograms show the distributions in 0 to 20 mg honeydew excretion in the respective experiments.

for virulence. I used the percentage of virulent females in the full-sib of the female parent as a female-parent value, that of the male parent as a male-parent value, and that of their offspring as an offspring value (Figure 1). The F_1 and F_2 generations were examined on Saikai 190, and the F_3 and F_4 generations were tested on an *indica* rice variety ASD7 carrying the *bph 2* gene.

Results

Character to be measured. The honeydew excretion of BPH on Saikai 190 does not apparently fit a normal distribution but seems to show a bimodal distribution (Figure 2). The tests for normality by the Shapiro-Wilk W-test showed that the distributions of honeydew excretion in all females tested were significantly different from the normal distributions in the two experiments, but those in the females which excreted honeydew of 0–10 mg and more than 10 mg did not significantly deviate from normality except the females which excreted 0–10 mg honeydew in the weight-gain experiment (Table 1). These results suggest that the distribution in honeydew excretion consists of at least two distinct distributions. Two distributions divided at 10 mg honeydew better fit the normal distribution than those divided at 20 mg (Table 1).

Table 1. Shapiro-Wilk W-tests for normality of distributions in honeydew excretion of *N. lugens* females in the weight-gain and oviposition experiments on Saikai 190

Honeydew excretion	N	W	P
Weight-gain experiment			
All	50	0.901	0.0003
≤10 mg	14	0.794	0.0037
>10 mg	36	0.943	0.084
≤20 mg	17	0.725	<0.0001
>20 mg	33	0.924	0.029
Oviposition experiment			
All	102	0.895	<0.0001
≤10 mg	31	0.972	0.639
>10 mg	71	0.970	0.232
≤20 mg	37	0.773	<0.0001
>20 mg	65	0.960	0.084

The weight gain of BPH females on days 1–2 also appears to show a bimodal distribution (Figure 3). It is positively correlated with the honeydew excretion in this period (Figure 4). All the females that excreted 0–10 mg honeydew gained no or slight weight, less than 0.1 mg (Figure 4). The coefficient of determination (r^2) is high 0.730 ($P < 0.0001$) for all females but not high, 0.0017 ($P > 0.8$) for the females with 0–10 mg honeydew, 0.392 ($P < 0.0001$) for >10 mg honeydew, and 0.252 ($P = 0.003$) for >20 mg honeydew.

Oviposition rate on days 3–12 among the females classified by the honeydew excretion on days 1–2 showed significant differences in the proportion of females ovipositing and the egg production between the females that excreted 0–10 mg and more than 20 mg honeydew (Table 2). Seventy-three percent of the females that had excreted 0–10 mg honeydew did not oviposit, and most of them died before day 4. On the

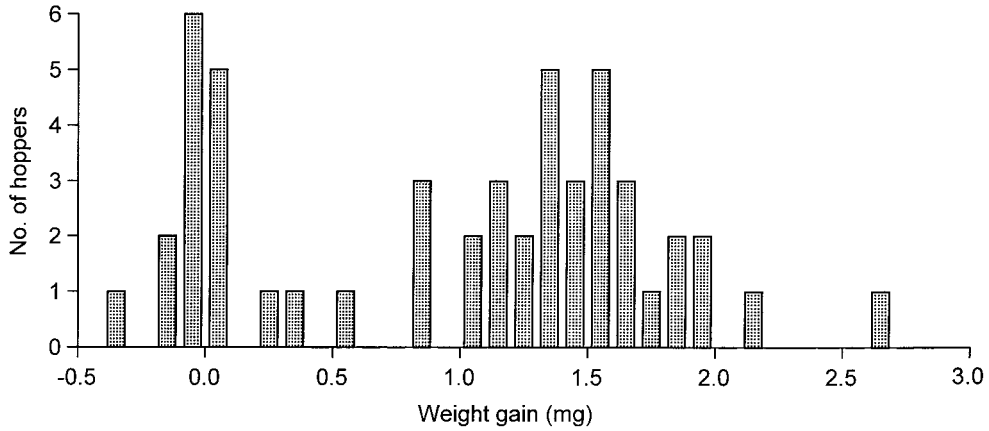

Figure 3. Frequency distribution of the weight gain in *N. lugens* females on Saikai 190 for two days.

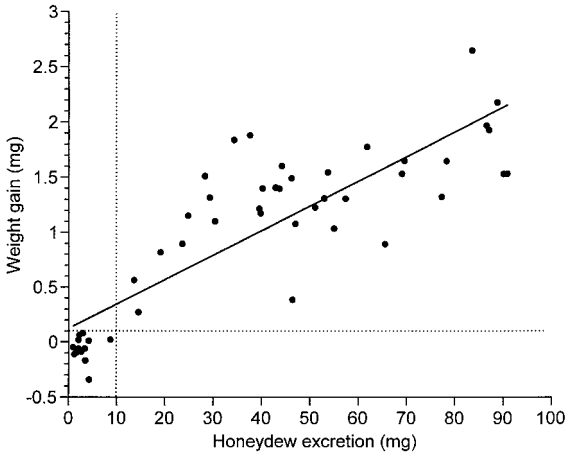

Figure 4. Relationship between the honeydew excretion and the weight gain in *N. lugens* females on Saikai 190 for two days. The solid line indicates the regression line of the weight gain to the honeydew excretion for all data.

other hand, 95% of the females that had excreted more than 20 mg honeydew oviposited, and 82% continued to reproduce throughout the experimental period. In the females excreting more than 20 mg honeydew, there are no significant differences in the proportion of females ovipositing and the egg production among the female groups with different honeydew excretion. In the females excreting 0–10 mg honeydew, the egg production is not significantly correlated with the honeydew excretion ($r^2 = 0.0028$, $P > 0.7$, $N = 30$). The proportion of females ovipositing and the egg production in the females that excreted 10–20 mg honeydew were intermediate between those in the females that excreted 0–10 mg honeydew and more than 20 mg honeydew. On Reiho, which carries no resistance gene, all the females ($N = 17$) excreted more than 30 mg honeydew on days 1–2 and oviposited. Their mean egg production for ten days, 323 ± 22 s.e., is not significantly different from that of the females which excreted more than 20 mg honeydew on Saikai 190, 298 ± 14 ($P > 0.4$, ANOVA).

All results shown above suggest that the virulence of BPH females to the resistant variety can be assessed by the honeydew excretion with a critical value of 10 to 20 mg. Below and above the critical value, however, the honeydew excretion is not a reliable measure that exactly represents the virulence. Hence, the virulence of BPH to the resistant varieties should not be analyzed as a continuous character but as a discontinuous character with two phenotypes, i.e., virulent and avirulent. It is reasonable to define avirulence as a honeydew excretion less than 10 mg or a weight gain less than 0.1 mg for two days. There are intermediately virulent females that excreted ca. 10 to 20 mg honeydew. I conveniently include them to virulent females.

Heritability of virulence. To examine whether the virulence of BPH can be determined by the appearance of their abdomina, I measured the honeydew excretion of females on Saikai 190 for two days and observed their abdomina (Figure 5). Females that had excreted less than 10 mg honeydew became thinner. By contrast, females that had excreted 40 mg or more honeydew had swollen abdomina. A few females excreted intermediate amount of honeydew (15–25 mg) and had indeed intermediately-swollen abdomina. These results are consistent with the results of the weight gain experiment (Figure 4). I defined the females which had more or less swollen abdomina as virulent.

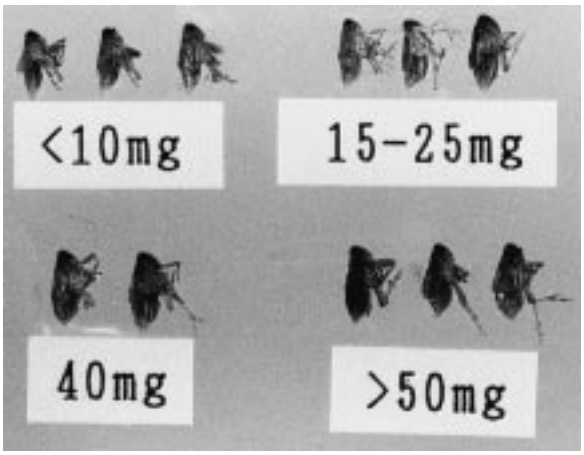

Figure 5. Nilaparvata lugens females that have different appearance of abdomina corresponding to the amounts of honeydew excretion which are shown by the figures below the specimens.

Table 2. Oviposition on days 3–12 in relation to honeydew excretion on days 1–2 in *N. lugens* on Saikai 190

Honeydew excretion (mg)	N	% females ovipositing[a]	No. of eggs deposited (mean ± s.e.)[b]
0–10	30	26.7 a	15 ± 7 a
10–20	6	66.7 ab	90 ± 39 ab
20–30	8	87.5 b	222 ± 36 b
30–40	8	87.5 b	273 ± 59 b
40–50	7	100.0 b	322 ± 42 b
50–60	6	100.0 b	331 ± 31 b
60<	9	100.0 b	346 ± 31 b

[a]Values followed by the same letter are not significantly different ($P > 0.05$, pair-wise comparisons by Fisher's exact probability test with adjusted significance level by Bonferroni method). Groups having the same percentage are treated as one group.
[b]Values followed by the same letter are not significantly different ($P > 0.05$, Tukey–Kramer method).

I calculated the parent-offspring regression using the percentage of virulent females in a family that was transformed into a probit value for normalization (Table 3). From the regression coefficients of the offspring values on the midparent values, the heritabilities of virulence were estimated to be 0.41 on Saikai 190 and 0.55 on ASD7. The regression coefficients on the female and male parent values were similar on both varieties, suggesting similar genetic contributions by the female and male parents. If both parents contribute equally, the estimate of heritability is expected to be twice the regression coefficient on single parents (Falconer, 1989). The regression coefficients on the single parents were about half of those on the midparents (Table 3). Thus, the heritability estimates from

Table 3. Coefficients in parent-offspring regression for virulence of *N. lugens* to resistant rice varieties

Varieties[a]	Slope	s.e.	*r*
Saikai190→Saikai190 ($N = 50$)			
midparent	0.41	0.17	0.33*
female parent	0.21	0.21	0.24
male parent	0.18	0.12	0.21
ASD7→Saikai190 ($N = 65$)			
midparent	−0.097	0.12	−0.10
female parent	−0.099	0.098	−0.13
male parent	−0.021	0.084	−0.031
ASD7→ASD7 ($N = 60$)			
midparent	0.55	0.18	0.38**
female parent	0.27	0.12	0.28*
male parent	0.24	0.13	0.23

[a]Varieties on which offspring and parents were tested are shown on left- and right-sides of an arrow, respectively.
* $P < 0.05$, ** $P < 0.01$.

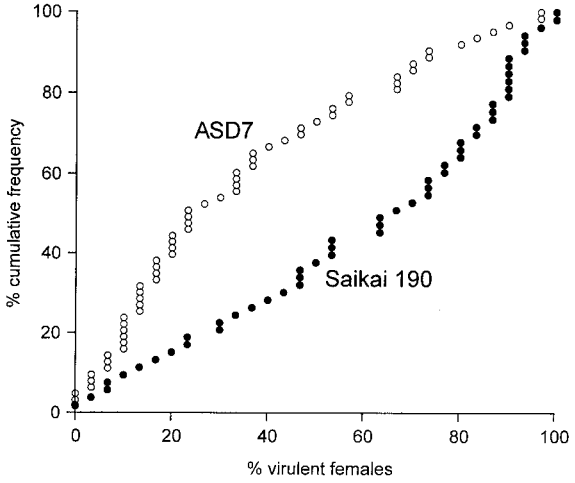

Figure 6. Cumulative frequencies of the percentage of virulent *N. lugens* females in a family tested on Saikai 190 and ASD7.

the regression on single parents and on midparents are consistent with each other. When the parental families were tested on Saikai 190 and the progeny on ASD7, the regression coefficients approached zero (Table 3). These results suggest that there is a low genetic correlation between virulences to the two varieties which have different resistance genes.

Figure 6 shows the cumulative frequencies of the percentage of virulent females in a family in the offspring generation tested on the two varieties. If the virulence is inherited in a simple Mendelian manner,

i.e., a single locus-two allele system, then the percentages of virulent females are expected to distribute around 0% (0:1), 25% (1:3), 50% (1:1), 75% (3:1), or 100% (1:0). On the other hand, in a threshold character under polygenic control, the groups of individuals, e.g., families or populations, can have any values in percentage of individuals exhibiting one phenotype, though the individuals can have only a few possible forms or values (Falconer, 1989). The percentages of virulent females have rather continuous values (Figure 6), suggesting the polygenic inheritance of virulence. Furthermore, I crossed females and males whose sib all had either a virulent (V) or an avirulent (A) phenotype on Saikai 190, and tested the virulence of their progeny. The ratios of virulent: avirulent females in the progeny were 23:17 in V $\times$ V, 28:14 in V $\times$ A, and 16:24 in A $\times$ A. If virulence is controlled by alleles of a single locus, all the progeny from one or both crosses of V $\times$ V and A $\times$ A will be of a single phenotype. Thus, the virulence can not be explained by a simple Mendelian model.

Discussion

Threshold character. Several studies analyzed the genetics of BPH biotypes by crossing experimentally selected biotypes (Cheng & Chang, 1979; Sogawa, 1980, 1981b; Ito & Kisimoto, 1981; den Hollander & Pathak, 1981), but none of their results fit a simple Mendelian model. Thus, some researchers suggest that the virulence of BPH is under polygenic control (Sogawa, 1980; den Hollander & Pathak, 1981; Roderick, 1994). The results of this study are consistent with polygenic inheritance of this character.

Many studies have used the honeydew excretion as a measure of virulence and host adaptation of BPH individuals to the resistance of rice plants. Indeed, the honeydew excretion is substantially different between virulent and avirulent BPH females, and therefore, it can be used for discriminating between virulent and avirulent females. This study shows that the critical value for determining the virulence of BPH females is around 10 to 20 mg honeydew excretion for two days after emergence. Within the groups of virulent and avirulent females, however, the honeydew excretion, at least by measuring in a short term as long as two days, is not a reliable measure that exactly quantifies virulence or host adaptation. This may be due to daily fluctuations in feeding activity of BPH, variation in assimilation rate of food intake among BPH individuals, and/or variation of volume or nutrient contents in phloem sap in rice plants. Thus, we should not estimate the heritability of virulence by using the honeydew excretion as a quantitative character. We should also not use weight gain, oviposition rate, and longevity as quantitative characters because their distributions deviated from normality and the avirulent females had similar values for these characters. At present, therefore, it is a realistic method to analyze the virulence of BPH as a threshold character. When the physiological and biochemical mechanisms of BPH that overcome the resistance of rice are found, we may identify the underlying quantitative variable that determines the virulence of BPH and may be able to analyze the genetics of virulence by using it.

Heritability of virulence. Quantitative genetic analysis of BPH biotypes has not been conducted so far, though polygenic inheritance of the biotypes was suggested as early as 1980 (Sogawa, 1980; den Hollander & Pathak, 1981). This study analyzed the virulence of BPH to resistant rice varieties as a threshold character, and estimated that the heritabilities of virulence were 0.41 on Saikai 190 carrying the *Bph 1* gene, and 0.55 on ASD7 carrying the *bph 2* gene. Roderick (1994) calculated the realized heritabilities of virulence in BPH using the survival rate of nymphs in the selection experiments presented by den Hollander & Pathak (1981), Claridge & den Hollander (1982), and Pathak & Heinrichs (1982). The estimates of heritability were 0.279 to Mudgo (*Bph 1*) and 0.320 to ASD7, and do not deviate considerably from the estimates of this study. These heritability estimates show that the BPH population has substantial genetic variation in virulence. The results are consistent with the fact that the damage by BPH was observed on IR26, which was the first commercial rice variety carrying the *Bph 1* gene, in the Philippines in 1975, two years after its release (Feuer, 1976). Virulent biotypes also evolved in many crop pests (Diehl & Bush, 1984), which provides evidence that genetic variation in virulence or host adaptation is common in nature. Indeed, herbivore populations have considerable genetic variability in relative performance on various host plants (for review see Via, 1990).

The method of estimating heritability used in this study utilizes the family values instead of the individual's values; hence, the estimates are underestimates and have large standard errors (Sokal & Rohlf, 1995). This method, however, has the advantage that the genetic contributions of the female and male parent can

be evaluated separately. Similar contributions of the female and male parent were shown, suggesting no maternal effect. This study measured only the virulence of females. We still need to establish a method to assess the virulence of males.

Low genetic correlation is suggested between virulences to the two varieties which have different resistance genes, *Bph 1* and *bph 2*. Changes of biotypes on the two varieties, i.e., from biotype 1 to biotype 2 and 3, may occur independently of each other. This tentative conclusion is consistent with the results of selection experiments which have provided no evidence that selection on the BPH population by a *Bph 1*-carrying variety had a positive or a negative effect on virulence to a *bph 2*-carrying variety, and vice versa (Sogawa, 1981a; Ito & Kisimoto, 1981; den Hollander & Pathak, 1981; Claridge & den Hollander, 1982; Pathak & Heinrichs, 1982).

Nine or ten genes conferring resistance to BPH have been found in rice plants (Nemoto et al., 1989; Ishii et al., 1994). We should investigate the genetic variability and the heritability of virulence in BPH populations to resistance genes other than *Bph 1* and *bph 2*. Genetic correlations between virulence to the different resistance genes and between virulence and other life history traits of BPH are also important to be established. If negative genetic correlations exist between virulence to two or more varieties or between virulence to a particular variety and the fitness of BPH on this variety, rotation cropping or patchwork cultivation of the two or more rice varieties will hinder or delay the development of virulent biotypes of BPH.

Using the estimated heritabilities, we can predict the rate of biotype change based on a quantitative genetic model. I have obtained a prediction that the proportion of virulent individuals in a BPH population will exceed 50% in several generations on a single resistant variety (Tanaka, unpubl.). Predictions of biotype changes under various cultivation of rice are of great significance for establishing the BPH management strategy using resistant rice varieties.

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