THE INHERITANCE OF TUNGRO RESISTANCE IN RICE SELECTION CR94-13 AND ALLELIC RELATIONSHIPS OF GENES FOR TUNGRO RESISTANCE IN CR94-13 AND IR833-6-2-1 LINES

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The thesis attached hereto, entitled "THE INHERITANCE OF TUNGRO RESISTANCE IN RICE SELECTION CR94-13 AND ALLELIC RELATIONSHIPS OF GENES FOR TUNGRO RESISTANCE IN CR94-13 AND IR833-6-2-1 LINES," prepared and submitted by Mr. Mansur Lande in partial fulfillment of the requirements for the degree of Master of Science (Agronomy) is hereby accepted.

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BIOGRAPHICAL SKETCH

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ABSTRACT

Mansur Lande, University of the Philippines at Los Baños, February 1975. The Inheritance of tungro resistance in rice selection CR94-13 and allelic relationships of genes for tungro resistance in CR94-13 and IR833-6-2-1 lines. Major Professor: Dr. G. S. Khush.

The inheritance of resistance to tungro disease in rice selection CR94-13 was investigated, and allelic relationships of genes for tungro resistance in CR94-13 and IR833-6-2-1 lines were studied at Lanrang Experimental Farm, Maros Research Institute for Agriculture, South Sulawesi, Indonesia. The results indicated that two pairs of genes having cumulative effect conveyed resistance to tungro in CR94-13. Resistance is incompletely dominant over susceptibility. In addition to the two pairs of incompletely dominant genes, modifiers with minor effect might also be involved in these crosses.

The reactions of F_1, F_2, and F_3 populations of the cross between CR94-13 and IR833-6-2-1 showed that the resistance in these two selections was governed by allelic genes.

INTRODUCTION

Tungro virus is one of the most destructive and widely distributed rice disease in Southeast Asia. A recent outbreak of the disease infected about 10,000 hectares in West Malaysia in 1964. During the same year the yellow-orange leaf disease was first noticed in Thailand and it spread to 660,000 hectares in 1966. Northeastern India and Bangladesh were severely hit by tungro in 1968 and 1969. In 1971, tungro suddenly broke out in Luzon and other islands of the Philippines and severely affected some 100,000 hectares. During the 1969 to 1971 crop seasons in the Indonesian provinces of South Kalimantan, South Sumatra and Lampung, approximately 21,000 hectares of rice were attacked by a disease having tungro like symptoms and caused a severe loss. The disease is likely to become more important with increasing emphasis on the greater use of fertilizer, closer plant spacing, and year-round cultivation with the new improved early maturing varieties which lack field resistance to tungro. The extensive plantings of a few susceptible varieties in large areas to replace hundreds of local varieties will increase the danger of future epidemics.

The disease is generally acknowledged to have been first identified as a virus disease by Rivera and Ou (1965). Mentek disease of Indonesia which has been considered a physiological disorder since 1859 is probably tungro, too. Penyakit merah in Malaysia which has been known since 1934 was identified to be tungro in 1965. Same disease probably affected rice crop in the Philippines in 1940 but was called as "stunt" or "dwarf" disease.

The virus is principally transmitted by the rice green leafhopper, Nephotettix virescens (Distant). Other insect vectors are N. nigropictus, N. parvus, and Recilia dorsalis, but these insect species are poor transmitters of the virus. The insect can acquire the virus if it feeds on diseased plants for at least 30 minutes and it can infect healthy plants by feeding on them for at least 15 minutes. The latent period for the virus in the plant is six to nine days.

Symptoms of the diseased plants differ with variety, environmental conditions, age of plants, and strains of the virus. Stunting is severe on susceptible varieties, but slight on more resistant ones. The leaves of the diseased plants show yellowing or reddish-brown discoloration from the tip of the leaf and may extend to the lower part of the leaf blade. Often only the upper portion of the leaf is discolored. The panicles produced by diseased plants are fewer in number, shorter in size, imperfectly exserted and often have a high percentage of sterile or imperfectly developed grains with brown hulls. A heavy and frequent infestation on susceptible varieties will cause a total loss of the crop.

One way of controlling the disease is through vector control with insecticide, but this method has several limitations at the farmers level. Attempts to control the vector in South Sulawesi during an outbreak in 1972 were disappointing. Two major contributing factors were a shortage of effective insecticides in sufficient quantity at critical time and a need to make extensive applications over the vectors' natural habitat as well as in rice field, which make insecticide costs prohibitive to most farmers. Moreover,

intensive applications of insecticides over several years may lead to the build-up of insect tolerance to commonly used chemicals as well as to an increase in the rise of environmental pollution.

The other alternative of controlling the disease is the planting of varieties resistant to tungro virus. It is an ideal method of protecting the crop from the virus. The initial cost of developing a resistant variety should amount to little in most cases in comparison with the cost of other control measures or with losses that would be experienced if no efforts were made to control the disease. Also, the logistics, extension and credit activities associated with an effective chemical control program are woefully lacking in most of the affected areas. These considerations have provided a strong stimulus for breeding resistant varieties.

Several resistant varieties have been used as source of resistance in the breeding programs but the mode of inheritance of resistance to virus is not known. A knowledge of the mode of inheritance of disease resistance is helpful in selecting the most efficient and effective breeding method for developing resistant varieties. Moreover, it is important to identify diverse genes for resistance so that rice breeders may not unconsciously incorporate the same gene for resistance even though they may be using several different parents as a source of resistance.

The objectives of this study therefore are the following:

1. To determine the mode of inheritance of tungro resistance in a resistant selection, CR94-13.

2. To determine whether tungro resistance of IR833-6-2 and CR94-13 is governed by allelic or non-allelic genes. These two selections are being used as sources of resistance at the International Rice Research Institute.

REVIEW OF LITERATURE

Past Occurrence of the Disease

The disease probably occurred in Java as early as 1840. In 1859, a disease later thought to be tungro caused severe damage in the fields (Vriese, 1859). Another report indicated that such a disease was serious in 1921 and 1934 and somewhat serious in other years in Bali, Sulawesi, Java, Madura, and Sumatra (Vecht, 1953). In early 1969 a moderately severe outbreak occurred in South Kalimantan, lasting up to 1971. At the same time an outbreak occurred in South Sumatra (Tantera and Oka, 1972). Likewise a similar disease was noticed in Central and South Sulawesi at the end of 1972 (Shagir Sama, 1972a and 1972b). Early in the following season, during January and March 1973, an estimated 40,000 hectares were seriously damaged.

In Malaysia, according to Singh (1969), the first mention of "penyakit merah" (red disease) appears to have been made by Coleman-Doscas in 1934. The viral nature of the disease was demonstrated by transmission with Nephotettix virescens by Ou and Goh (1966). Farmers in the Krian district, where "penyakit merah" is a serious problem in some season, believe that losses from the disease may be about 40 percent. During the 1966-67 season in

Kedah, the area affected was about 600 acres. Fifty acres were affected severely and no yield was obtained from them. Overall losses reported were between 50 and 56 percent (Singh, 1969).

Tungro may have occurred in the Philippines, for many years and at least since 1940. Serrano (1957) estimated the yield losses in 1940 were estimated at 30 percent or 1.4 million metric tons. An outbreak in 1971 affected thousands of hectares in Central Luzon, Bicol and other areas in the country (Reddy, 1973).

Tungro disease in Thailand was formerly called yellow-orange leaf. It was first observed in central Thailand in 1964. The affected area varies from year to year; in 1965 an estimated 16,000 hectares were affected severely (King, 1966). Lamey et al. (1967), estimating the outbreak in central Thailand, concluded that 660,000 hectares were affected moderately to severely, of which 350,000 hectares were in the latter category.

In India, the disease was first reported in 1967 (Raychaudhuri et al., 1967). A severe outbreak occurred in Bihar and U.P. States in 1969 (John, 1970).

Nature and Identification of the Disease

The disease has probably been present in Indonesia since 1840 or even earlier as reported by Vriese (1859). Due to the uncertainty of identification, the disease was often considered to be a physiological disease, a nematode problem, a virus disease or a combination of these. The disease is known as "mentek" in Java, "penyakit habang" in South Kalimantan, and "Cella pance" in South Sulawesi.

Rivera et al. (1968) indicated that, based on symptoms, virus-vector interaction, and the varietal reactions, the disease observed in South Sumatra and West Java was caused by the tungro virus. Their findings corroborate those on tungro in the Philippines.

Since the early 1940s, rice dwarf or stunt disease (Agati et al., 1941; Reyes, 1957; Reyes et al., 1959), 'accep na pula' (red disease) or stunt disease (Serrano, 1957) and rice 'cadang-cadang' (yellowing) (Agati and Peralta, 1939; Peralta and Agati, 1939) have been reported from the Philippines. These cases are now believed to have been due to tungro disease (Ou and Ling, 1966).

'Penyakit merah' has been known to occur in Malaysia since 1938. It was for a long time considered to be a physiological disorder and was suspected of being caused by metabolic deficiency of nitrogen (Lockard, 1959) or by hydrogen sulphide or organic acids originating from the anaerobic decomposition of weeds. Recent studies (Ou, 1965; Ou and Goh, 1966) have shown that "penyakit merah" is due to a virus transmitted by Nephotettix virescens. The symptoms, the non-persistent manner in which the virus is transmitted by the insect vector and the varietal reaction of the varieties tested are very similar to those of tungro virus, and indeed no distinction between the two virus diseases has been found.

A new virus disease was reported from Thailand in 1964 (Wathanakul, 1965) and was called yellow-orange leaf (Wathanakul and Weerapat, 1969). It was transmitted by Nephotettix virescens and was also found to be non-persistent. Similarities in symptoms and varietal reaction in general, together with the virus-vector interaction

and other factors suggest the virus is the same as, or is a strain of, tungro virus.

Another virus disease resembling tungro has been reported from India (Raychaudhuri et al., 1967). It has symptoms similar to tungro and is also transmitted by Nephotettix virescens. John (1970) has conclusively shown that the virus disease is identical to tungro.

Varietal Resistance to Virus and the Vector

The need to control tungro virus, potentially the most destructive disease of rice, is especially urgent. Introduction of rice varieties resistant to the virus as well as to the insect vector can directly affect the incident of virus disease. Siwi and Oka (1967) reported that tungro resistant varieties Intan and Mas were released for commercial production in Indonesia in 1940, and Bengawan and Peta in 1941. Within a short period these varieties spread all over Indonesia, especially in the regions where "mentek" disease was known to be prevalent. These resistant varieties gradually replaced the susceptible local varieties, and the crop failures by "mentek" were very much reduced.

Varietal evaluation for resistance to tungro was initiated soon after the identification of the virus nature of disease. Thousands of varieties and hybrid lines have been tested for resistant to tungro at IRRI and in different countries and several resistant varieties have been identified (Ling and Aguiero, 1970). In India, Pankhari 203, Latisail, Ambemohar 102, Kataribhog and Kamod 25-3 were found resistant to tungro. Pankhari 203, HR-21,

PTB 18, Gam Pai-15, TKM-6 and Habiganj DW 8 are also being used as sources of resistance to tungro in IRRI breeding program (Khush, 1972).

The abundance of insect population may sometimes be associated with the release of a new host crop variety. According to Pathak (1968), if the newly released variety happened to be more susceptible to the insect pest than other existing varieties, a phenomenal increase in the insect population and damage may be encountered. On the other hand, if it was less susceptible, its wide cultivation may lead to the general decline of the pest population. Studies conducted by Jennings and Pineda (1970) to determine the effect of resistant rice plants on the multiplication of the planthopper, Sogatodes oryzicola (Muir) indicated that the number of eggs deposited per day by each female was about twice as great on the susceptible as on the resistant variety. Resistance of the host variety reduced the number of nymphs hatched, lower nymphal survival, prolonged the nymphal period, and reduced adult longevity. Because of more eggs deposited per day, greater egg fertility and increased nymphal survival, the susceptible plants had about 10 to 12 times as many nymphs as did resistant plants. Of these, about 3 to 5 times as many nymphs reached adult stage on susceptible rice as on resistant one. Cheng and Pathak (1972) obtained similar results in the study of resistance to Nephotettix virescens (Distant) in rice varieties. The results indicated that besides higher mortality, nymphs developed slower on resistant than on susceptible varieties. Resistant varieties generally were not preferred by adults and nymphs. Adults had 4 to 8 times longer life span and laid about 15 times more eggs on susceptible than on

resistant varieties. The population declined rapidly on resistant varieties whereas it increased cumulatively on susceptible varieties. Since rice virus diseases are transmitted by the insect vectors, the incidence of the virus is directly related to the insect population. Therefore varietal resistance to the insect indirectly contributes to the control of virus diseases.

Inheritance of Resistance to Virus and its Vector

Studies on the inheritance of tungro resistance at IRRI revealed that 22 F_1 seedlings from the cross, Pankhari 203 x Taichung Native 1, showed completely resistant reactions. Out of about 520 F_2 plants tested, the healthy and diseased plants segregated at a ratio of 9:7 indicating that the resistance in Pankhari 203 is governed by two complementary dominant genes (International Rice Research Institute, 1967). The F_1 hybrid from the cross of IR8 x Latisail showed a resistant reaction. In the F_2 populations, 339 plants were resistant while 299 were susceptible, a ratio that fits the digenic, complementary-dominant ratio of 9:7 (Shastry et al., 1972). They observed that some of the plants originally classified as susceptible recovered while the plants originally classified as resistant remained resistant. Thus, later scoring indicated that 529 plants were resistant while 39 were susceptible. This conforms to the duplicate gene ratio of 15:1.

According to Shastry et al. (1972), the resistance is basically governed by two complementary dominant genes and the recovery from the initial susceptible condition is controlled by the presence of either one of these two dominant genes. Plants remained susceptible, i.e., did not recover only when both genes controlling the resistance were absent.

The study of the backcross progenies of Pankhari 203 x Taichung Native 1 and Pankhari 203 x IR8 at IRRI showed that certain plants identified as resistant became diseased at a later growth stage and vice versa. It was therefore concluded in this study that resistance at the seedling stage and at adult stage should be considered separately in the genetic analysis of tungro resistance (International Rice Research Institute, 1968).

In studying the inheritance of resistance to leafhoppers in rice varieties, Athwal et al. (1970, 1971) concluded that dominant alleles at three different independent loci convey resistance to the green leafhopper. They found that the F_1 plants from crosses between leafhopper resistant cultivars (Pankhari 203, ASD 7 and IR8) and susceptible parent (Mudgo) were resistant. Random sample of F_2 population of the crosses Mudgo x Pankhari 203, Taichung Native 1 x ASD 7, and Taichung Native 1 x IR8 were screened for reaction to the green leafhopper. The F_2 segregation in all three crosses resulted in a 3 resistant: 1 susceptible ratio. The monogenic segregation was confirmed by the F_3 breeding behavior, which in all crosses, agreed with the expected 1 homozygous resistant: 2 segregating: 1 homozygous susceptible ratio. It was concluded that Pankhari 203, ASD 7, and IR8 are homozygous resistant for one major dominant gene. These genes, were designated as Glh 1 in Pankhari 203, Glh 2 in ASD 7, and Glh 3 in IR8.

MATERIALS AND METHODS

The study was conducted at Lanrang Experimental Farm of Maros Research Institute for Agriculture, South Sulawesi, Indonesia. The

Farm is 25 meters above sea level. The area and the vicinity were severely infected with tungro disease four seasons previously. The experimental materials were planted on January 12, 1974. The planting time was selected to obtain a good infestation of the disease.

Three improved plant type selections and the hybrid populations from the crosses between them were used in the study. The selection CR94-13, a tungro and rice green leafhopper resistant line developed at CRRI (India), is from a cross of (PTB 21 x PTB 18) x IR8. PTB 18 and PTB 21 are tungro resistant varieties from India, while IR8 is susceptible. The tungro susceptible line IR1416-131-5 is from a cross of IR400-28-4-5 x Tetep. Tetep is resistant to blast but susceptible to tungro and rice green leafhopper.

IR400-28-4-5, an experimental line from Peta[4] x Taichung Native 1 is likewise susceptible to tungro. The inheritance of resistance was investigated in the F_1, F_2 and F_3 generations of the cross between CR94-13 and IR1416-131-5.

IR833-6-2 is another tungro and rice green leafhopper resistant line from the cross of (Peta[3] x TN1) x Gam Pai 15. It inherits its resistance from Gam Pai 15. F_1, F_2, and F_3 progenies of the cross between CR94-13 and IR833-6-2 were studied to determine the allelic relationships of genes for resistance of these two selections.

All three lines have improved plant type and semidwarf stature. The crosses between these three lines were made at IRRI and the F_1 and F_2 populations were also grown at IRRI. Duplicate samples of each parent, F_1, F_2 population and F_3 lines were provided by IRRI.

Pelita I/1 was planted in three rows along both side of the experimental field parallel to the test plots. This variety is

susceptible to tungro disease as well as to the rice green leafhopper. It is a medium tall variety with vigorous vegetative growth and having a dark green leaf color. Due to these characters the infected plants of this variety show a very clear and bright yellow-orange leaf discoloration symptoms. Pelita I/1 was planted on the border rows to verify the distribution of the disease infection in the experimental field and also to serve as a source of disease inoculum.

A plot size of 3-row, 5 meters long was used, with a 25 x 25 cm spacing in the plot. One plant per hill was planted, resulting in 63 plants per lot. The number of plots and plants of each generation studied are shown in Table 1.

The plots planted to F_1 were flanked by the plots planted to two parents. The plantings of the F_3 progenies were arranged according to their consecutive number.

Immediately preceding transplanting, nitrogen and phosphorus fertilizers were applied at the rate of 40 kg N, and 60 kg P_2O_5 per hectare, respectively. Additional nitrogen fertilizer was applied at the rate of 40 kg N per hectare at 4 and 7 weeks after transplanting, respectively. No insecticide was used in the seedbed or in the field.

Seeds of the parents and of the F_2 and F_3 generations were sown directly in a wet seedbed. The F_1 seeds were germinated in a petri dish on the same date. The germinated F_1 seeds were placed in wooden box filled with fine soil after 5 days of germination and thereafter treated and managed as the seedlings in the seedbed. At 21 days after sowing, the seedlings were transplanted into the field.

Table 1. Number of plots and plants of parents and hybrid generations studied.

No.	Entries	No. of plots	No. of plants
1	P_1 - CR94-13	1	59
2	P_2 - IR1416-131-5	1	52
3	P_3 - IR833-6-2-1	1	56
4	C - Pelita I/1	1	56
5	F_1 - CR94-13 x IR1416-131-5	1	55
6	F_1 - IR833-6-2 x CR94-13	1	38
7	F_2 - CR94-13 x IR1416-131-5	9	535
8	F_2 - IR833-6-2 x CR94-13	10	550
9	F_3 - CR94-13 x IR1416-131-5	399 lines	
10	F_3 - IR833-6-2 x CR94-13	408 lines	

The scoring for tungro infection was done at 60 days after seeding or 39 days after transplanting. The scoring was mainly based on yellow-orange discoloration of the infected leaves and degree of stunting. At this stage the stunting of the diseased plants was quite obvious. Diseased and healthy plants were counted in the parental as well as in the F_1, F_2 and F_3 progenies. General observations on flowering and recovery were taken at later stages also.

RESULTS

Symptoms of Tungro Virus Disease

The infected plants could be identified about 3 to 4 weeks after transplanting. The primary symptoms which were observed were the discoloration of leaves in the shade of yellow or orange (Fig. 1). As the plants advanced in age, differences in height of the infected and healthy plants could be observed. The diseased plants were stunted and had somewhat reduced tillering. At heavier disease pressure even the apparently resistant plants showed some leaf yellowing symptoms especially near the leaf tips. No stunting was observed in such plants however.

Healthy plants flowered at normal time and had good seed set. Infected plants either failed to flower at all or produced small panicles with discolored grains (Fig. 2). Seed set in infected plants was very low and the kernels were chalky and otherwise of reduced size. Flowering of infected plants was delayed by several weeks. Number of panicle bearing tillers in infected plants was much reduced.

Figure 1. Symptoms of tungro virus disease on resistant and susceptible F_3 families of CR94-13 x IR1416-131-5 at 60 DAS showing yellow-orange leaf discoloration.

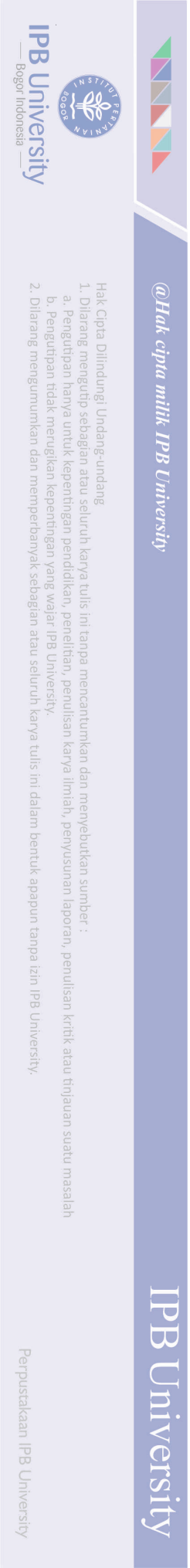

Figure 2. Symptoms of tungro virus disease on susceptible IR1416-131-5 parents at maturity.

As the plants approached maturity the intensity of leaf yellowing decreased. However, the diseased plants could be easily identified by characteristic stunting and delayed flowering or absence of flowering.

Disease Reaction of Parents and Pelita I/1

Most of the plants of resistant parents showed good vigor and remained healthy throughout the life cycle. However, a small proportion of the plants of resistant parents became diseased and showed characteristic tungro symptoms. However, the number of diseased plants in the resistant parents was small (Table 2) and the symptoms were not so severe as in the susceptible parents. The diseased plants showed considerable recovery and produced good number of panicles. Occurrence of diseased plants in the resistant parents is not unusual. Ling (1968) reported that up to 10 percent plants of Pankhari 203 which is highly resistant to tungro become diseased after artificial inoculation. Similarly, Ling et al. (1970) observed that 6.9 percent plants of O. nivara became infected with grassy stunt although O. nivara is highly resistant to grassy stunt.

Susceptible parents showed high percentage of infected plants. In IR1416-131-5 for example, more than 90 percent plants were infected. In Pelita I/1, similarly, the proportion of infected plants was 82.1 percent. However, a few plants of susceptible parents escaped infection and appeared healthy.

Disease Reaction of F_1 Progenies

The proportion of diseased plants in the F_1 of CR94-13 x IR1416-131-5 was somewhat higher than that of resistant parent (Table 2). The infected plants were not as severely discolored and stunted as that of the susceptible parent, hence the F_1 is classified as having an intermediate reaction. The percentage of infected plants in the F_1 progenies is lower than the mean of infection of both resistant and susceptible parents. The resistance thus appeared to be incompletely dominant, and modifier genes with minor effect may be involved in the expression of resistance.

The F_1 progenies of IR833-6-2-1 x CR94-13 were all resistant when scored at 60 days after seeding. A few plants showed mild symptoms of infection at later stages. However, the infected plants flowered and produced quite normal panicles. A highly resistant reaction of the F_1 progenies in this cross compared to the reaction of resistant parent might also be due to the presence of modifier genes with minor effect. Thus the reaction of the F_1 between the two resistant parents was similar to both the resistant parents.

Disease Reaction of F_2 Populations

The proportion of diseased plants in F_2 populations of the cross CR94-13 x IR1416-131-5 is shown in Table 3. Approximately 40 percent of the plants showed disease symptoms. Chi-square test shows that the data failed to fit the ratio of 3 resistant: 1 susceptible, expected on the basis of monogenic control of resistance.

Table 2. Proportion of tungro-infected plants of Pelita I/1, parents, F_1 and F_2 populations at 60 days after seeding.

Parent or cross	No. of plants		%
	Total	Infected	Infected
CR94-13	59	4	6.78
IR833-6-2-1	56	4	7.14
IR1416-131-5	52	50	96.15
Pelita I/1 (check)	56	46	82.14
CR94-13 x IR1416-131-5 (F_1)	55	19	34.54
IR833-6-2-1 x CR94-13 (F_1)	38	0	0.0
CR94-13 x IR1416-131-5 (F_2)	535	215	40.19
IR833-6-2-1 x CR94-13 (F_2)	550	34	6.36

Test for a digenic model having complete dominance did not fit the ratio of 9 resistant: 6 intermediate: 1 susceptible, as the P value lies between .025 and .010. However, if we assume two genes having cumulative effect and partial dominance for resistance, as indicated by F_1 data, the F_2 data may segregate in the proportion of 5 resistant: 10 intermediate: 1 susceptible reaction to tungro disease. When the F_2 plants of this cross were classified according to the group means percentage of infected plants in F_3 families (Table 3), the expected proportions became 29 resistant, 180 intermediate and 29 susceptible. The X^2 value of 4.0384 gave a P value close to .050. This result suggests a digenic and incompletely dominance control of resistance.

In the F_2 of IR833-6-2-1 x CR94-13, 6.3 percent of the plants showed diseaae symptoms at 60 days after seeding. The proportion of diseased plants was not higher than that of resistant parents (Table 2). It thus appears that segregation for susceptibility did not occur in this cross, suggesting that the resistance in the two resistant parents may be goverend by allelic gene or genes.

Disease Reaction of F_3 Families of CR94-13 x IR1416-131-5

The number of infected plants in each family was counted. Some families had very few diseased plants. The diseased plants were not too stunted, however. These families were obviously homozygous for resistance. The percentage of plants showing the disease symptoms per family range from 4.8 percent to 98.4 percent. The frequency distribution of F_3 families of this cross is shown in

Table 3. Classification of F_2 plants of CR94-13 x IR1416-131-5 according to the group means (%) of infected plants in F_3 families of the same cross.

Group reaction	Group means (%)	Expected ratio	Expected number of plants		
			Total	Infected	Free
Resistant	17.50	5	167.2	29.26	137.94
Intermediate	53.90	10	334.4	180.24	154.16
Susceptible	85.75	1	33.4	28.64	4.76
		Expected		238.14	296.86
		Observed		215	320

X^2 value for 5:10:1 = 4.0384

P level = .050-.025

Table 4 and Figure 3. There were 126 or 31.83 percent of the F_3 families which had an infection range of from 3.9 percent to 31.1 percent and a mean of 17.5 percent. The F_3 families within this group were classified as resistant. This resistant group consisted of three genotypes of the doubly dominant type (AABB, AABb, AaBB). The double heterozygous portion (AaBb) is not included, because the disease reaction of double heterozygous F_1 was classified as intermediate (as indicated in F_1 and F_2). The mean of infection percentage in the resistant group is higher than the infection percentage of the resistant parent, partly because this group is not only made up of homozygous double dominant (AABB), but also of the other heterozygous dominants (AABb and AaBB) in one pair of the alleles.

The F_3 families of the same cross which had disease infection between 31.2 percent to 76.6 percent were classified as intermediate in reaction. This infection percentage range is equal to the mean infection percentage of 53.9 percent. The group had a larger infection range partly because it was made up of five different genotypes. In this intermediate reaction group, there were double heterozygous (AaBb), two genotypes with one pair homozygous dominant and the other pair of the homozygous recessive (AAbb and aaBB), and the other two genotypes with homozygous recessive for one factor pair and having heterozygous condition at the other locus (Aabb and aaBb). There were 213 or 53.38 percent of the F_3 families classified as intermediate.

The remaining 59 F_3 families or 14.79 percent with the range of infection from 76.7 percent to 98.4 percent and having a mean

Table 4. Frequency distribution and classification of F_3 families of CR94-13 x IR1416-131-5 according to the percentage of infected plants.

Range of infection (%)	Number of families	% of total	Group means (%)	Group reaction	Expected ratio	Number of families	
						Expected	Observed
3.9-12.9	11	2.76					
13.0-22.0	46	11.53	17.50	Resistant	21	130.9	127
22.1-31.1	70	17.54					
31.2-40.2	52	13.03					
40.3-49.3	58	14.54	53.90	Intermediate	34	212.0	213
49.4-58.4	41	10.28					
58.5-67.5	32	8.02					
67.6-76.6	30	7.52					
76.7-85.7	31	7.77	85.75	Susceptible	9	56.1	59
85.8-94.9	28	7.02					

X^2 for 21:34:9 = 0.2708

P value = .900-.750

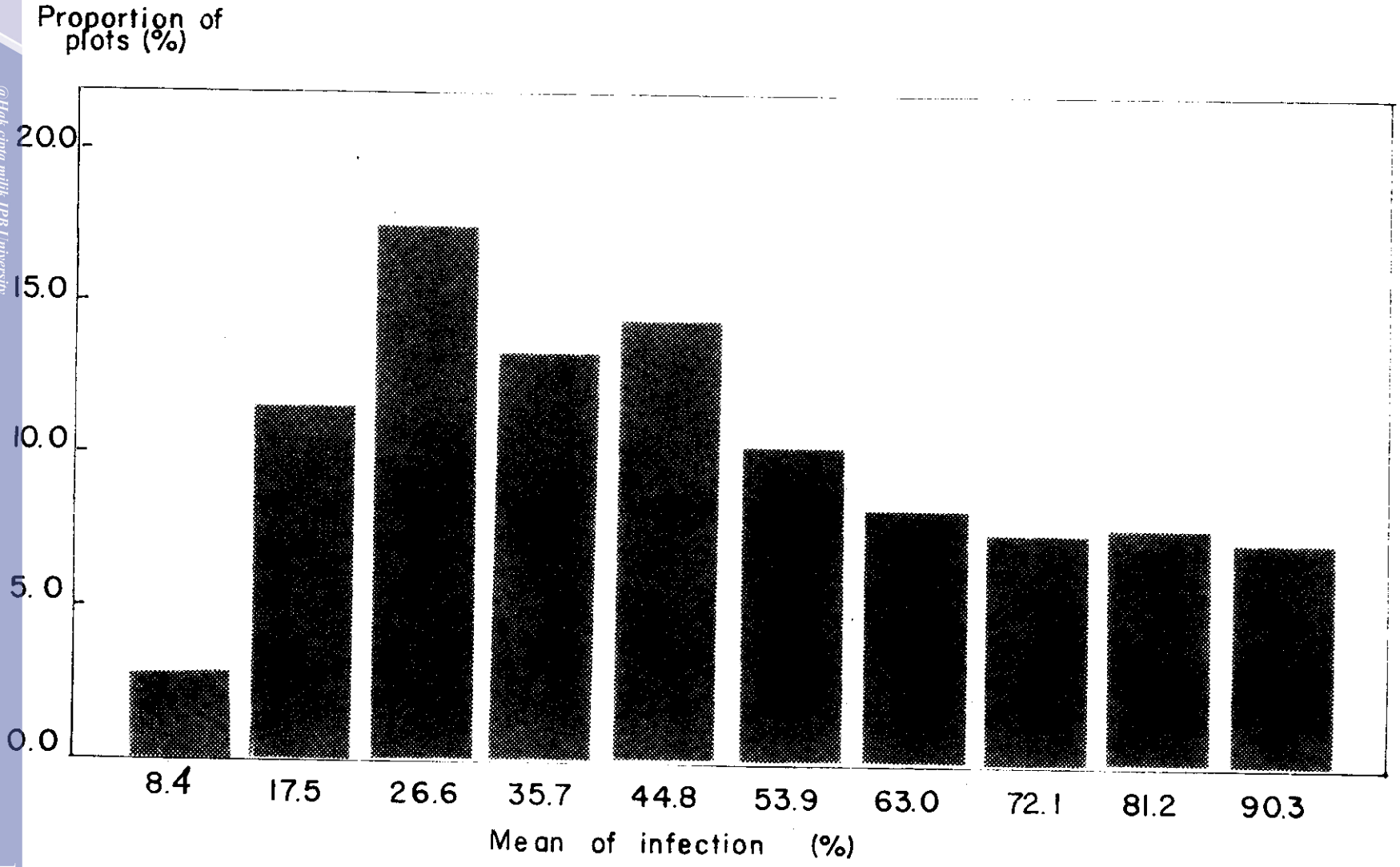

Fig. 3. Frequency distribution of disease infection of F_3 families of CR94-13 x IR1416-131-5 based on (%) infected plants.

infection of 85.75 percent were obviously of the double recessive genotype (aabb). This group would be susceptible. The proportion of susceptible F_3 families was very close to the expected value of about 14.06 percent.

Thus of the 399 F_3 families of the cross CR94-13 x IR1416-131-5, 127 were classified as resistant, 213 as intermediate, and 59 as susceptible. This classification is very close to the expected value for a digenic model with incomplete dominance. Chi-square test gave a good fit to the ratio of 21 resistant: 34 intermediate: 9 susceptible (X^2 = 0.2708, P = .750-.900).

Disease Reaction of F_3 Families of IR833-6-2-1 x CR94-13

A very high proportion of F_3 families of this cross were highly resistant. Out of 408 families 392 or 96.08 percent had mean percentage of infection which was equal to the mean percentage of both resistant parents (Table 5 and Figure 4). Among this group there were 91 families or 22.3 percent which had not a single infected plant at 60 days after seeding. These families showed a highly resistant reaction to the tungro disease.

On the other hand, 16 families gave a higher mean infection percentage (20.72%) than either one of resistant parents (6.78% and 7.14%). The highest infection within a family was 26.00 percent. But genetic segregation for truly susceptible progenies did not appear to occur in this cross. This transgressive segregation suggests that in addition to the major genes for resistance, modifier genes with minor effect may also be involved in this cross.

Table 5. Frequency distribution of F_3 families of IR833-6-2-1 x CR94-13 according to the percentage of infected plants.

Range of infection (%)	No. of families	% of total
0.0	91	22.30
0.1-13.81	301	73.78
13.82-27.62	16	3.92

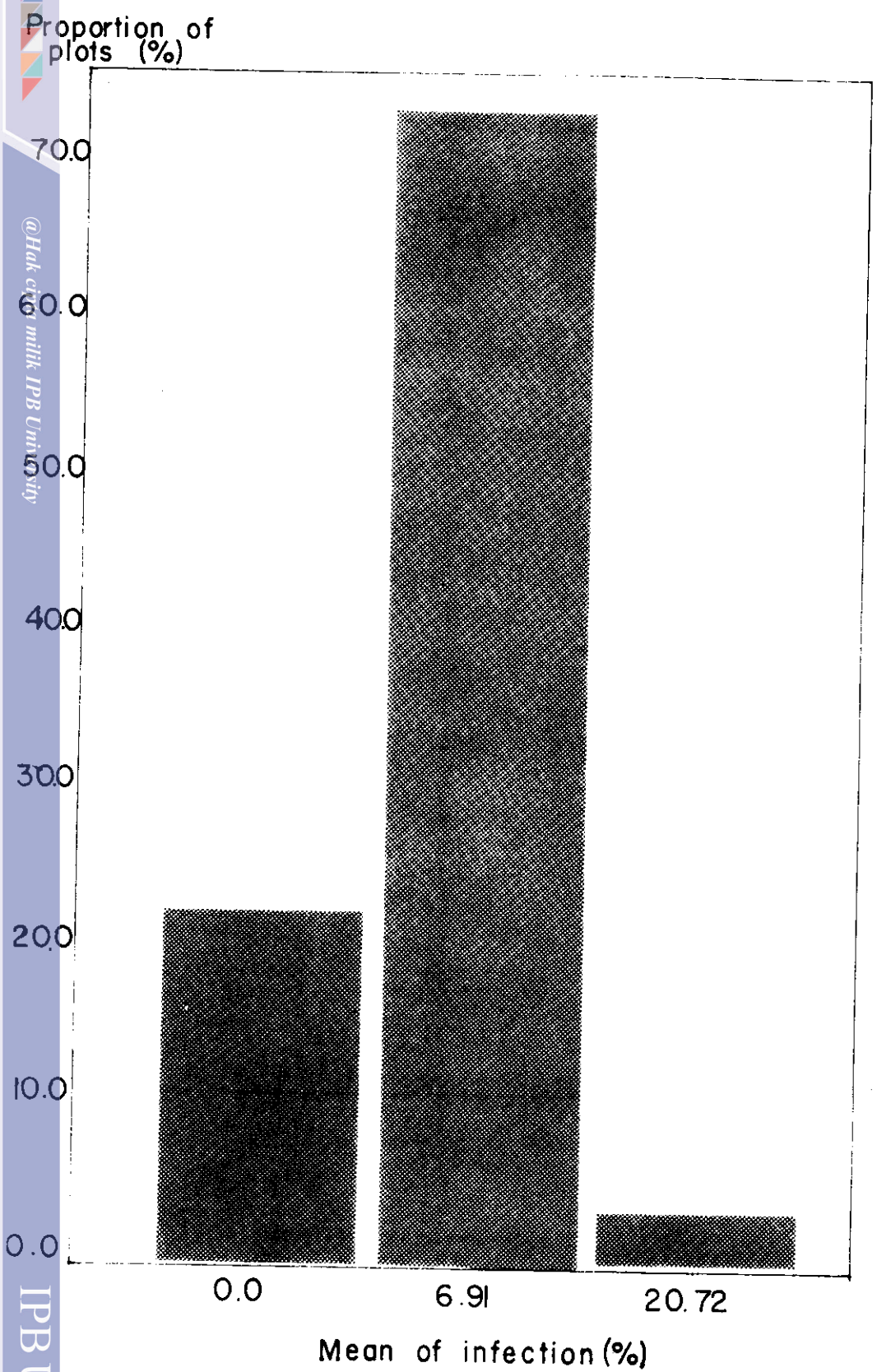

Fig. 4. Frequency distribution of disease infection of F_3 families of IR833-6-2-1 x CR94-13 based on (%) infected plants.

The two parents must have allelic genes for resistance. These results confirm the conclusions drawn earlier from the study of the F_2 population.

DISCUSSION

Very few critical studies have been carried out on the inheritance of resistance to tungro virus. Shastry et al. (1972) investigated the inheritance of resistance in IR8 x Latisail cross using the screenhouse technique. The F_1 showed a resistant reaction. The F_2 population segregated, 339 resistant and 229 susceptible seedlings approximating a digenic complementary ratio of 9:7. They transplanted the inoculated seedlings and re-scored them 40 days after transplanting. Some plants which were earlier scored as susceptible, recovered and were classified as resistant. According to the second scoring 529 plants were resistant and 39 susceptible, conforming to the duplicate gene ratio of 15:1. The authors concluded that the resistance is basically goverend by two dominant complementary genes. Recovery from an initial susceptible condition occured when only one of the two dominant genes was presented in the plant. Plants remained susceptible only when both genes controlling resistance were absent. However, the authors ignored the fact that in the screenhouse method of inoculation which they employed, the seedlings were inoculated only once and then grown under disease- and insect-free conditions. Some of the seedlings might recover as the amount of virus transferred to the plant during single inoculation period may not be sufficient. High fertility level of the field might also help the plant recover from initial infection.

Under the field screening method employed in this study, the seedlings were continuously re-inoculated throughout their life cycle because the disease source and the vectors were abundant and there was very little recovery from the severely diseased condition. Moreover, under moderate and heavy infestation of virus-free green leafhopper on an insect-susceptible variety, the plants suffered serious damage and showing leaf yellowing discoloration (Hsieh, 1972). Later, the plants had very few panicles and were stunted. Due to these factors, the F_2 data alone are not sufficient to draw conclusions about the inheritance of resistance to virus diseases. It is well known that a proportion of plants escape the disease infection even in the susceptible parents. A proportion of the plants of the resistant parent may also get diseased. Thus in the F_2 population there is a possibility that some susceptible genotypes are classified as resistant whereas some resistant genotypes are classified as susceptible. Therefore F_2 data on disease reaction is generally not considered as entirely reliable in studying the patterns of inheritance. Data on the disease reaction of F_3 families on the other hand, is more critical in determining the genotype of each F_2 individual.

Present study is the first attempt to analyze the inheritance of resistance to tungro from field screening and from F_3 data. The data indicated that two pairs of incompletely dominant genes confer the resistance to tungro in CR94-13. IR833-6-2-1 also has the same genes for resistance. In addition to the two pairs of incompletely dominant genes, modifiers with minor effect may also be involved in these crosses.

Several varieties with tungro resistance have been identified and are being used in the breeding program. However, it is not known whether they have the same genes or different genes for resistance.

This is the first attempt to analyze the allelic relationships of genes for resistance from two different sources. Studies of this nature need to be intensively pursued to identify diverse genes for tungro resistance. As soon as different genetic sources of resistance are identified, they should be incorporated into the high yielding background.

SUMMARY

Inheritance of resistance to tungro virus was investigated in rice selection CR94-13 which is highly resistant to tungro. Allelic relationships of genes for resistance in CR94-13 and IR833-6-2-1 another resistant selection were also studied. F_1, F_2, F_3 populations from the crosses CR94-13 x IR1416-131-5 and IR833-6-2-1 x CR94-13 were screened under heavy disease pressure under field conditions in South Sulawesi, Indonesia. The results indicated that two pairs of incompletely dominant genes conveyed resistance to tungro in CR94-13. In addition to the two pairs of incomplete dominant genes, modifiers with minor effect might also be involved in these crosses.

The reactions of F_1, F_2, and F_3 populations of the cross between CR94-13 and IR833-6-2-1 showed that the resistance in these two selections was governed by allelic gene or genes.

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