STUDIES ON RESISTANCE TO YELLOW MOSAIC DISEASE IN BARLEY. I.*

Tests for Varietal Reactions and Genetic Analysis of Resistance to the Disease.

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INTRODUCTION

Barley yellow mosaic is a disease caused by a soil-borne virus. It was first found in Okayama Prefecture, western Japan, by Ikata and Kawai in 1940 and now is known to be prevalent all over Japan, except Hokkaido. It does not occur outside Japan. Before about 1950 its damage to the barley crop was not serious, but it has recently become one of the pressing problems, especially in production of two-rowed malting barley. Japanese farmers and maltsters eagerly await the breeding of malting-barley varieties highly resistant to this disease, because there is no other effective and economical measure of control.

Several authors have already shown that there is considerable variation among barley varieties with respect to disease symptoms, damage and resistance to the disease. Native six-rowed covered and naked barleys are said to include much more tolerant varieties than the two-rowed forms, which are in general highly susceptible. This suggests the possibility of developing a resistant two-rowed form by breeding.

Needless to say, resistance breeding requires some immune or highly resistant genetic stocks and knowledge about the mode of inheritance of the resistance. Differentiation in virulence of the causal virus is another important problem. For the past eight years we have been attempting to solve these questions. This paper outlines the results of screening for resistance, tests for varietal reactions to the virus from different sources, and genetic studies of the resistance to the disease.

NATURE, SYMPTOMS AND RATING OF THE DISEASE

Inouye (1964, 1968) has confirmed by electron microscopy that slightly flexible rod-shaped particles are always associated with barley yellow mosaic. The particles are about 13–14 mμ in width, and in their length distribution have two peaks at about 275 and 550 mμ. The barley yellow mosaic virus resembles in morphology two other distinct soil-borne viruses of Japan: wheat yellow mosaic virus and rice necrosis mosaic

* This paper was compiled in an abridged form from the following three papers published in Japanese in the Journal of our Institute, "Nôgaku Kenkyu", Vol. 51 (1966) 135–152, Vol. 52 (1968) 65–78 and Vol. 53 (1970) 153–165.

virus. Although the mechanism of infection with barley yellow mosaic virus is not yet fully proved, Kusaba and Tôyama (1970) and Tôyama and Kusaba (1970) have recently obtained some evidence that a lower fungus, *Polymyxa graminis* Led., is possibly the vector of the virus.

For the screening and genetical studies it is desirable to establish a highly reliable method of testing the reaction to the disease. A high rate of infection has sometimes been observed by means of artificial inoculation with expressed sap obtained from a heavily infected plant, but in some other cases only a low rate of infection has been obtained by the same method, owing to some unknown causes. Moreover, this method requires too much labor for use in tests of a large number of varieties and genetical materials. Consequently, all of our experiments and tests were made by growing plants in a field heavily infected with the virus under consideration.

It is widely accepted that sowing seed one week to ten days earlier than the usual or optimum seeding date for barley in the particular location in late autumn results in heavier infection. In this case the temperature prevailing during the seedling growth stage seems to be the most important factor: According to Kusaba et al. (1969a) and others, optimum temperature for infection lies between 13° and 16°C. and some of the incubation period should also be at a rather low temperature for the proper development of the disease symptoms.

When a highly susceptible variety was seeded in early November at Kurashiki, characteristic mottling symptoms began to be recognizable in late December to early January, followed by some necrosis and yellowing of lower leaves. Growth was markedly suppressed, tillering was reduced, and the plants eventually died in April to May. In more or less tolerant varieties the first appearance of the disease symptom was delayed to some extent. Their symptoms were most markedly expressed in late March to early April, but as the temperature rose above 20°C in April, the plants tended to recover their vigor to some extent.

In this series of experiments disease symptoms were observed and recorded three or more times from early March to mid-May, mostly on a single plant basis for both mottling and yellowing of leaves. The criteria for the classification of reactions used were as follows:

Leaf mottling		Yellowing of leaves	
Class	Degree of reaction	Class	Degree of reaction
0	None	o	None
1	Slight	a	Slight
2	Moderate	b	Moderate
3	Severe	c	Considerable with some necrosis
4	Very striking	d	Plant heavily stunted, dying or dead, with severe yellowing and necrosis

Generally there is some variation in symptom expression among individuals of the same variety. A tolerant variety sometimes includes a few seemingly healthy plants, which might be due to minor differences in concentration of virus-infected soil in the field or to differences in time of germination and growth of seedlings. Yearly variation is also considerable. Therefore, the screening tests of varieties were repeated at least twice for each variety. The genetical data, on the other hand, were admitted as reliable only when all plants of a susceptible parent and a susceptible control variety expressed symptoms almost typical of the variety.

SCREENING OF VARIETIES FOR RESISTANCE

A total of 800 varieties or strains of barley, consisting of 300 two-rowed forms and 368 and 132 six-rowed covered and naked forms, respectively, were screened for resistance to barley yellow mosaic at two severely infected locations, Niimi and Nariwa, in Okayama Prefecture, for four years. All the varieties were grown each year at each of these locations in a randomized block design with two replications. As a result, they could be classified into five groups, RR (highly resistant), R (resistant), M (intermediate), S (susceptible) and SS (highly susceptible). Table 1 shows frequencies in per cent of the varieties in each reaction grade in each of the different groups of barley and also in each part of the world.

TABLE 1. Frequencies in per cent of the varieties showing different reactions to barley yellow mosaic in the three barley groups and in various parts of the world.

Barley group or region	Reactions					No. of vars tested
	RR	R	M	S	SS	
Two-rowed	1	4	9	64	22	300
Six-rowed, covered	4	10	18	50	18	368
Six-rowed, naked	1	6	20	58	15	132
Japan	2	8	20	50	20	167
Korea	7	17	28	44	4	46
China	2	4	12	63	19	57
Nepal & India		3	4	55	38	68
S.W. Asia	2	5	9	72	12	43
Turkey	7	15	25	46	7	92
Europe	4	8	8	64	16	133
U.S.S.R.	2	4	6	65	23	48
North Africa		2	16	51	31	67
Ethiopia		4	13	66	17	54
Others		4	4	76	16	25

It was found in this test that most Japanese and European two-rowed malting varieties with good quality were SS or S. The two-rowed forms from Turkey, on the contrary, involved about 40 per cent of more or less resistant ones; among 67 samples 29 were RR, R or M. However, they were all inferior in quality and agronomic characters.

As is seen in Table 1, the covered six-rowed barley is more tolerant in general than the two-rowed barley. About one-half of the varieties from Japan, Korea, Turkey and Europe are more or less tolerant to this disease. It is noteworthy that a Chinese variety, Mokusekko 3, proved to be most promising for resistance breeding, because it did not show any disease symptoms in this and other field tests nor in artificial inoculation tests. The six-rowed, naked barley seems to be intermediate between the two other groups.

Generally speaking, Japan, Korea and Turkey are rich in forms tolerant to this disease, while Nepal, India, North Africa and Ethiopia are poor in resistant forms and lacking entirely in highly resistant form.

RELATION BETWEEN DISEASE SYMPTOMS AND PLANT DAMAGE

Twenty-two barley varieties, which had been known from the previous screening test to be distinctly different in reaction to the disease, were used as the material. Twenty plants of each variety were grown with two replications in both the heavily infected and the virus-free fields in our institute at Kurashiki. The numbers of diseased plants and their symptoms were recorded 16 times at intervals of about one or two weeks from late December to mid-April. Stem length, number of heads per plant and number of dead plants per plot were recorded at the time of maturity. The experiment was performed twice, in 1965 and 1966.

Six of these 22 varieties did not show any disease symptoms in this test, whereas they had been found to show conspicuous disease symptoms at Niimi, north of Kurashiki. On the contrary, Mihori Hadaka 3 showed definite disease symptoms in late spring, whereas it was never diseased at Niimi. The 15 other varieties behaved almost the same in both locations, however. The problem of the inconsistent reactions between the two locations will be discussed in the next section.

Fig. 1 and Table 2 summarize the results obtained in 1965. As is apparent in Fig. 1, in all the highly susceptible varieties, numbered 18–22, the appearance of disease symptoms was much earlier and the rate of increase of diseased plants more abrupt than in the less susceptible and resistant varieties. It could also be recognized that there was a close relation between the severity of disease symptoms and plant damage as expressed by the reduction of stem length and number of heads per plant. Moreover, some of the highly susceptible plants died in late April

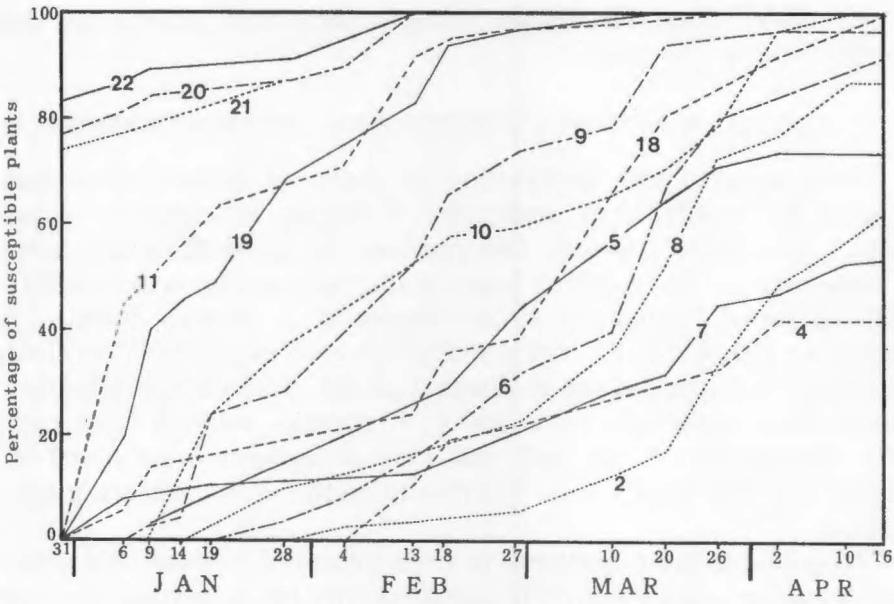

Fig. 1. Increase in per cent of diseased plants in the following 14 varieties from January to mid-April in 1955.

2. Mihori Hadaka 3	8. Turkey 78	19. Tochigi Golden
4. Turkey 582	9. Turkey 7	20. Nikaku Chevalier
5. Turkey 666	10. Hozoroi	21. Natsudaikon-mugi
6. Kasaura Naked	11. Peatland	22. B.E. 20
7. Short Head	18. Kô A	

TABLE 2. Relation between disease symptoms and plant damage.

Variety	Symptom		Diseased plants %	Dead plants %	Reduction of *		Rank
					Stem length %	Head no. %	
1. Mokusekko 3	0	o	0	0	3	+30	15
2. Mihori Hadaka 3	1	o	63	0	8	23	14
3. Bavarian	0–1	b	12	0	16	46	12
4. Turkey 582	1	ab	42	0	22	46	10
5. Turkey 666	2	b	74	0	32	37	8
6. Kasaura Naked	2	a	92	0	26	34	11
7. Short Head	2	a	53	0	10	27	13
8. Turkey 78	2	b	87	0	24	49	9
9. Turkey 7	2–3	b	97	0	28	42	9
10. Hozoroi 1	3	a	100	0	35	53	7
11. Peatland	3	b	100	0	46	64	3
18. Kô A	3	c	100	25	33	59	6
19. Tochigi Golden	3	d	100	71	65	91	1
20. Nikaku Chevalier	3	c	100	55	57	40	5
21. Natsudaikon-mugi	4	c	100	26	63	72	2
22. B.E. 20 (Ymer)	4	c	100	0	46	53	4

* Rate of reduction in infested plot against the virus-free plot

to early May. Thus the disease strongly affects plant growth and consequently reduces the yield.

VARIETAL REACTIONS TO VIRUSES FROM DIFFERENT SOURCES

Wide geographical distribution in Japan of barley yellow mosaic suggests the possibility of occurrence of viruses with different pathogenicity. In order to approach this problem, the same 22 barley varieties as were used in the previous investigation were grown in both 1965 and 1966 and tested for reaction to the disease at 11 severely infested locations from Tôhoku to Kyushu districts. In addition, artificial inoculation tests with the expressed sap of diseased plants obtained from six different sources were repeatedly made using 10 varieties selected from the 22. Since the results of the field and inoculation tests were found to be substantially the same, only the results of the latter test are given in Table 3.

According to their reactions to virus infection in these two kinds of tests, varieties could roughly be classified into the following five types:

a) Completely resistant.... Mokusekko 3, which showed no appreciable symptoms in the tests made at different locations nor in the inoculation test.

b) Highly susceptible.... Nikaku Chevalier, Natsudaikon-mugi, Kô A, etc. The symptoms were invariably very marked at all places and years. Plants were severely stunted and some of them almost dead at last.

c) Intermediately susceptible with little variability in reactions.... Hozoroi 1, Kasaura Naked, Peatland, etc. There was, however, some variation among varieties in severity of disease symptoms.

d) Highly resistant in most of the locations and years, but in some cases susceptible. One is Mihori Hadaka 3, as mentioned before, and the other Bavarian. The latter variety rarely showed disease symptoms in the field tests, but as seen in Table 3, was almost always infected by artificial inoculations.

e) Reaction varying markedly with location and year; in some places or years these varieties were susceptible, being ranked S, but in other places or years showed no symptoms. As seen in Table 3, quite similar behavior was observed in the artificial inoculation tests. Varieties belonging to this group are Svanhals, Satsuki Nijô, Asahi 5, Asahi 19, Avanguardia and Revil, which were not listed in Table 2 because disease symptoms were not observed at all at Kurashiki, though very marked at Niimi.

The variable reactions of the barley varieties belonging to the groups d and e seem to indicate differentiation of barley yellow mosaic viruses

TABLE 3. Susceptibility in per cent of ten varieties inoculated with juice from diseased plants from sources indicated.

Variety \ Source	Kurashiki			Niimi		Kagawa			Tottori			Yamaguchi			Kumamoto		
	A	B	C	A	B	A	B	C	A	B	C	A	B	C	A	B	C
1. Mokusekko 3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2. Mihori Hadaka 3	0	0	0	0	0	50	5	0	0	0	0	0	0	0	0	0	0
3. Bavarian	80	10	13	13	20	30	20	80	55	0	33	20	0	0	70	0	7
6. Kasaura Naked	100	35	60	100	100	95	45	93	80	5	53	90	5	7	90	5	20
12. Svanhals	0	0	0	90	87	40	5	0	0	0	0	90	10	20	0	0	0
13. Satsuki Nijô	0	0	0	—	47	30	0	0	0	0	0	75	33	7	0	0	0
15. Revil	0	0	0	80	80	35	0	0	0	0	0	100	10	0	0	5	0
16. Asahi 5	0	0	0	95	87	70	0	0	0	0	0	90	10	20	0	0	0
18. Kô A	75	35	33	75	87	95	45	60	60	25	17	80	15	21	70	5	33
21. Natsudaikon-mugi	95	45	33	60	87	95	35	60	75	30	17	95	20	13	100	45	53

A and B: tested in a glass house, and C in a growth chamber kept at 15°C in daytime and 12°C at night under 12 hours illumination.

with different pathogenicity, as was maintained by Saito and Okamoto (1964) and Kusaba et al. (1969b). However, it is not yet clear whether this anomalous behavior can wholly be attributed to differences in virus strains. In any case, the fact that Mokusekko 3 has never been infected by this disease in any location in Japan manifests that this problem is not serious for the present from the breeder's viewpoint, if the resistance gene or genes involved in this variety are used for breeding.

GENETICS OF RESISTANCE AND SUSCEPTIBILITY

A total of 32 crosses were dealt with in the genetic experiments. The crosses may be roughly classified into two groups: One is those made between the completely resistant variety Mokusekko 3 and 11 varieties varying in degree of susceptibility, and the other involves as the parents the highly resistant variety Mihori Hadaka 3 and six susceptible varieties. Almost all the experiments were carried out at Niimi during 1963 to 1969 and the rest at Kurashiki. Unless otherwise stated, all the data presented in the following are those obtained in the experiments in which all the plants of the "susceptible" parents expressed disease symptoms typical of the varieties.

a) Reaction of the F_1 Hybrids

The F_1 hybrids, together with their parents, were grown in both 1963 and 1966 and tested for reaction to the disease. Since the results obtained in both years were found to be almost the same, in Table 4

TABLE 4. Disease reactions of F_1 hybrids and their parents.

Parent & Symptom			Mokusekko 3		Kasaura Naked		Hozoroi 1		Natsudaikon-mugi	
			0	o	1	o	3	a–b	4	b–c
B.E. 20	3	b–c	2	a	3	o	3–4	b	4	b
Natsudai-kon-mugi	4	b–c	2	a	2–3	o	4	b		
Hozoroi 1	3	a–b	2	a	2	o				
Kasaura Naked	1	o	0–1	o						
Nikaku Chevalier	3	b	2	a						
Seijô 17	3–4	b	2–3	a						
Satsuki Nijô	3	b	2	a						
Tochigi Golden	4	c	2	a						

are given only the results in 1966. It is evident that all the F_1 hybrids, without exception, expressed reactions intermediate between their parents. This suggests no dominance or complicated interactions between the genes for resistance and susceptibility.

b) Inheritance and Linkage of the Resistance of Mokusekko 3

Table 5 shows the F_2 data for the segregation of non-diseased and diseased plants in the crosses between Mokusekko 3 and eight susceptible varieties. As is apparent in the table, the observed numbers of the two phenotypes fitted well to the calculated numbers on a 1 : 3 ratio in most cases. The crosses with Kasaura Naked and Seijô 17 are the exceptions, in which non-diseased plants were a little more numerous than expected for a 1 : 3 ratio. This may be explained by assuming that a few "susceptible" plants escaped from the infection or had symptoms too faint to be recognized. These results clearly indicate that a single major gene pair is involved in these crosses.

TABLE 5. Segregation of the resistant and susceptible plants in F_2 of the crosses of Mokusekko 3 with eight susceptible varieties in 1966 and 1969.

Crossed with		Resist.	Suscept.	Total	χ^2 for 1:3 ratio	P
Natsudaikon-mugi	1966*	62	205	267	0.4507	.7 ~.5
	1969	72	178	250	1.9253	.2 ~.1
Kasaura Naked	1966*	58	116	174	6.4444	.02~.01
Nikaku Chevalier	1966	25	66	91	0.2967	.7 ~.5
	1969	51	126	177	1.3729	.3 ~.2
Satsuki Nijô	1966	40	109	149	0.2212	.7 ~.5
Seijô 17	1966	44	78	122	7.9672	<.01
Nigrinudum	1969	62	205	267	0.4507	.7 ~.5
Orange lemma	1969	60	179	239	0.0013	>.95
Colsess IV	1969	71	179	250	1.5413	.3 ~.2

* Tested at Kurashiki

It must be noted here again that, as was shown in Table 4, the resistance is not completely recessive to, but partially dominant over, susceptibility. In fact, all the "susceptible" classes always showed wider variations in reaction than the susceptible parent. The symbol *Ym* will be assigned to the gene responsible for the resistance of Mokusekko 3.

An F_3 progeny test was made only for the cross between Mokusekko 3 and the highly susceptible variety Natsudaikon-mugi in 1967-68. A total of 230 out of 267 lines, each consisting of about 30 plants, were grown in the heavily infected field at Kurashiki, and their reactions to

the disease were recorded on a single-plant basis on February 26 and March 25, 1968. The result of the first investigation is given in Fig. 2,

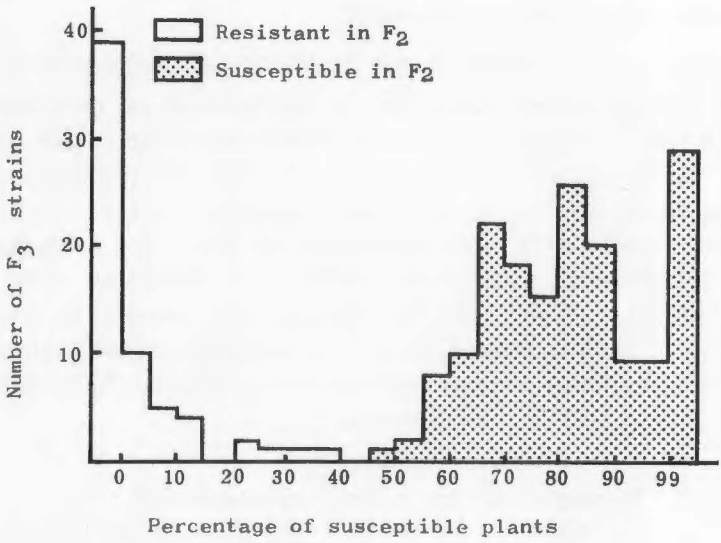

Fig. 2. Frequency distribution of the F_3 lines against percentages of susceptible plants involved within each line derived from the cross between Mokusekko 3 and Natsudaikon-mugi.

in which frequencies of F_3 lines having various precentages of diseased plants involved within each line are shown. It is immediately obvious that the F_3 lines derived from the resistant F_2 plants are either completely resistant or substantially lower in percentage of diseased plants than the F_3 lines derived from the susceptible F_2 plants, and the distribution is bimodal and discrete. Thus, a single pair of genes with strong effect was involved in the cross, as was inferred from the F_2 test. Nevertheless, segregation of some diseased plants within a line derived from a resistant F_2 plant can hardly be explained on the basis of a single-gene difference. According to the second assessment made on March 25, when diseased plants had increased much more, the number of F_3 line homozygous for resistance declined to about 1/16 of all the F_3 lines, and there was found also about the same number of F_3 lines which were all highly susceptible and uniformly stunted like Natsudaikon-mugi. This suggests the cross involved one more gene pair besides *Ymym*. If this be true, and if we assume that the genotype *Ymym aa* will be slightly susceptible, where *aa* is a gene for susceptibility present in Natsudaikon-mugi, the F_2 segregation of non-diseased and diseased plants should occur in a 3 : 13 ratio. In fact, the observed numbers fitted well to the calculated ones on this basis.

It may be of some interest in this connection to know the genetic behavior of the hybrid between a moderately susceptible variety and a highly susceptible one. Two crosses made between Kasaura Naked, which always expresses disease symptoms of grade 2, and two highly susceptible varieties, Natsudaikon-mugi and B.E. 20 (Ymer), were tested for their reactions. In Table 6 are given variations of disease symptoms of the F_2 plants. Although some difficulties were encountered in assessing the grade of disease symptoms exactly on a single-plant basis, the results

TABLE 6. Mosaic symptoms of the F_2 plants from the crosses of a moderately susceptible variety, Kasaura Naked, with two highly susceptible varieties, Natsudaikon-mugi and B.E. 20. Observed numbers of moderately susceptible (3–4 and 4) compared to the calculated numbers on a 3 : 1 ratio.

Kasaura Naked ×	Grade of mosaic symptoms							Total	χ^2	P
	0	1	2	2–3	3	3–4	4			
Natsudaikon-mugi	2	1	52	5	162	2	50	274	5.2992	.05~.02
B. E. 20			17		166	1	48	232	1.8620	.2 ~.1

indicate that segregation of slightly susceptible (grade 3 or lower) plants and highly susceptible (3–4 and 4 grades) plants occurred in a 3 : 1 ratio, which suggests that Kasaura Naked has a partially dominant gene for its slight resistance, while both Natsudaikon-mugi and B.E. 20 involve an allele conditioning susceptibility. No evidence is available to indicate whether the gene in Kasaura Naked and the second gene involved in Mokusekko 3 are the same or not.

Interrelationships between the gene *Ym* for resistance and several marker genes on different barley chromosomes were studied in F_2 of the four crosses of Mokusekko 3 with Nigrinudum, Orange lemma, Nikaku Chevalier and Colsess IV, the latter four being moderately susceptible to the disease.

The results shown in Table 7 indicate clearly that the resistance gene *Ym* is inherited independently of *n* for naked kernel, *V* for two-row, *B* for black kernel, *o* for orange lemma and *s* for short-haired rachilla, which are known to be on chromosomes 1, 2, 5, 6 and 7, respectively. Evidence of linkage of *Ym* with *K* for hooded appendage was recognized in the cross with Colsess IV, in which excessive numbers of parental combinations of characters, namely diseased-hooded and non-diseased-long awn were apparent. The results shown in Table 8 indicate that the observed frequencies of the four phenotypes did not fit to the calculated numbers with independent segregation, but fitted well on the basis of 29.37% recombination. It is safe to conclude therefore that the principal gene *Ym* for resistance in Mokusekko 3 is on chromosome 4.

TABLE 7. Independent inheritance of the resistance vs. susceptibility with several markers in four crosses with Mokusekko 3.

Mokusekko 3 ×	Characters X	Characters x	Chromosome	Suscept. X	Suscept. x	Resist. X	Resist. x	Total	χ^2_L	P
Nigrinudum	*N*	*n*	1	137	58	47	15	257	0.799	.5~.3
	V	*v*	2	152	43	45	17	257	0.727	.5~.3
	B	*b*	5	162	32	50	12	256	0.250	.7~.5
	S	*s*	7	135	60	46	16	257	0.658	.5~.3
Orange lemma	*O*	*o*	6	143	33	47	13	236	0.188	.7~.5
Nikaku Chevalier	*V*	*v*	2	106	20	40	11	177	0.392	.7~.5
Colsess IV	*S*	*s*	7	133	44	57	14	248	1.307	.3~.2

TABLE 8. Linkage of *Ym* for resistance of Mokusekko 3 with *K* for hooded lemma appendage.

Items	Suscept. Hooded	Suscept. Awned	Resist. Hooded	Resist. Awned	Total	χ^2	P
Observed number	148	29	36	35	248		
Calculated number on 9:3:3:1	139.5	46.5	46.5	15.5	248	34.0072	small
Calculated number with 29.37% recombination	154.9	31.1	31.1	30.9	248	1.7657	.7~.5

c) Genetics of the Resistance of Mihori Hadaka 3

Mihori Hadaka 3 is next to Mokusekko 3 in resistance and has never been infected at Niimi. Genetic study of its resistance was attempted during 1964 to 1969 using crosses with Kô A, Svanhals, Bavarian and Turkey 2. The variety Kô A is highly susceptible, Svanhals is moderately susceptible and both Bavarian and Turkey 2 are fairly tolerant, exhibiting only slight disease symptoms. About 300 F_2 plants each from these four crosses were grown in 1964 and 1965, but no reliable data were obtained as the expression of disease symptoms of the "susceptible" parents was incomplete in most cases. In the following year, 1966, therefore almost the same numbers of F_2 plants and F_3 lines, derived from the F_2 plants grown in 1965, were again grown at Niimi. The results are given in Table 9.

The data obtained in this series of experiments were not always consistent: As to the cross with Kô A only the F_3 data fit well to a 1 resistant vs. 3 susceptible segregation ratio, and as to the cross with Svanhals neither the F_2 nor the F_3 data in 1966 fit well to the 1:3 ratio. So, in 1969 about 250 F_2 plants from the latter cross were again tested for reaction, which gave a satifactory fit to the 1:3 ratio. It may therefore be assumed from the results of the first three crosses in the

TABLE 9. Segregation of resistant and susceptible plants or lines in F_2 or F_3 of the crosses with Mihori Hadaka 3, a highly resistant variety.

Mihori Hadaka 3 ×	Generation and year	Resist.	Suscept.	Total	χ^2	P
Kô A	F_2(1966)	75	149	224	8.5952	small
	F_3(1966)	80	219	299	0.4916	.5~.2
Svanhals	F_2(1966)	90	188	278	8.0624	small
	F_3(1966)	49	251	300	12.0178	small
	F_2(1969)	50	191	241	2.3250	.2~.1
Bavarian	F_2(1966)	60	140	200	2.6667	.2~.1
	F_3(1966)	58	230	288	3.6296	.1~.05
Turkey 2	F_2(1966)	108	182	290	23.1772	small
	F_3(1966)	146	154	300	89.6176	small

table that a single major gene is responsible for the high resistance of Mihori Hadaka 3. The gene is tentatively named *Ym*$_2$. The genetic behavior of the cross with Turkey 2 was somewhat different from those of the three other crosses; many more resistant plants or lines appeared than were expected from a 1 : 3 segregation.

Interrelationships of the resistance gene *Ym*$_2$ with a few marker genes were studied in F_2 of the crosses of Mihori Hadaka 3 with Kô A and Svanhals in 1966 and 1969, respectively. It was found that *Ym*$_2$ was independent of *v* for six-row and *Hs* for hairy sheath on chromosomes 2 and 4, respectively, but was linked with the gene *n* for naked kernel on chromosome 1. As seen in Table 10, parental character combinations

TABLE 10. Linkage of the resistance gene *Ym*$_2$ in Mihori Hadaka 3 with the gene *n* for naked kernel.

Mihori Hadaka 3 ×	Item	Susceptible		Resistant		Total	χ^2	P
		Covered	Naked	Covered	Naked			
Kô A	Observed number	122	27	36	39	224		
	Calculated no. with independ. assortment	126	42	42	14	224	50.984	small
	Calculated no. with 29.5% recombination	139.8	23.2	28.2	27.8	224	8.984	small
Svanhals	Observed number	158	33	29	21	241		
	Calculated no. with independ. assortment	135.5	45.2	45.2	15.1	241	15.104	small
	Calculated no. with 33.3% recombination	147.2	33.6	33.6	26.6	241	2.612	0.5~0.3

Weighted average value of p=31.4±2.6936 (%)

are apparently in excess for the independent assortment of both gene pairs, and the fit of the observed frequencies to the calculated numbers

on this basis is very poor in both crosses. The recombination values calculated by Immer's product method were 29.5% and 33.5%, respectively. The χ^2 test made on the basis of the respective linkage intensities indicated that the fit was good only for the cross with Svanhals. Since χ^2 for the cross with Kô A proved to be as large as 8.984, the cause was investigated by partitioning the total χ^2 into its components, the results of which were as follows:

	χ^2	d.f.
Segregation for covered vs. naked kernel	2.381	1
Segregation for resistance vs. susceptibility	8.595	1
Linkage	40.008	1
Total	50.984	3

It became evident from the results that the large discrepancy mainly resulted from the distorted segregation of resistant vs. susceptible plants. At the same time, the very large χ^2 for linkage indicated clearly that Ym_2 and n are linked. Consequently, a weighted average value of p was calculated from the two data and an estimate of 31.4 ± 2.6936(%) arrived at.

d) Genetic Behavior of the Highly Susceptible Variety Kô A

Kô A is one of the highly susceptible varieties. It expresses conspicuous mosaic symptom from its very early growth stage, which is sooner or later followed by necrosis and yellowing of the lower leaves. When severely infected, it is markedly stunted and often dies in early spring. Mode of inheritance of the severe disease symptom expressed by Kô A was studied using crosses with Mihori Hadaka 3, Svanhals, Bavarian and Turkey 2, which differed from each other in disease reaction. For this, about 150 to 300 F_2 plants each from these crosses were grown at Niimi in 1964, and almost all of the F_2 plants were carried to F_3 test in the following year. About 300 F_2 plants each from these crosses were again grown together. However, as typical disease symptom of Kô A as well as those of the other susceptible parents were not observed in 1965, the same tests with almost the same numbers of F_2 plants and F_3 lines were repeated in 1966. The results obtained in 1964 and 1966 are given in Table 11.

Among the F_2 populations and F_3 lines derived from the cross with a moderately susceptible variety Svanhals, it was not difficult to distinguish the heavily stunted plants or lines from those exhibiting disease symptoms graded 2 or 3, and as seen in Table 11, segregation of these two types occurred in a 1:3 ratio in both years. Thus, it is possible to conclude that these two varieties differ from each other by a single gene pair, with the Svanhals allele conditioning slight resistance.

TABLE 11. Segregation of the normal and markedly stunted or dead plants or lines in the F_2 or F_3 generations of the four crosses with Kô A, a highly susceptible variety.

Kô A ×	Generation and year	Normal or moderately diseased	Stunted or dead	Total	Stunted %	χ^2 for 3:1	χ^2 for 15:1
Svanhals	F_2 1964	112	46	158	29.11	1.43*	
	F_2 1966	219	66	285	23.16	0.52*	
	F_3 1966	227	73	300	24.33	0.07*	
Bavarian	F_2 1964	291	23	314	7.32	52.32	0.62*
	F_2 1966	252	27	279	9.60	34.94	5.59
	F_3 1966	261	39	300	13.00	23.04	23.33
Turkey 2	F_2 1964	271	23	294	7.82	46.26	1.24*
	F_2 1966	240	39	279	13.98	18.08	28.43
	F_3 1966	298	1	299	—	—	—
Mihori Hadaka 3	F_2 1964	252	49	301	16.28	12.21	51.68
	F_2 1966	188	36	224	16.07	9.52	36.89
	F_3 1966	255	44	299	14.72	16.87	21.90

* $p > 0.05$

Reactions of the hybrids between Kô A and two tolerant varieties, Bavarian and Turkey 2, were somewhat different: A much lower number of stunted F_2 plants and F_3 lines were found in the F_2 and F_3 generations of both crosses, and the observed numbers in the F_2 tests made in 1964 fit well to a 1:15 segregation ratio. Thus, two gene-pair differences from Kô A were suggested. However, this could not be confirmed in the tests made in 1966, where the stunted type was slightly more than the expected number on the basis of a two-gene difference.

The behavior of the hybrid with Mihori Hadaka 3 differed again from either of the hybrids stated above. This cross segregated, besides one-fourth of the resistant F_2 plants or F_3 lines as shown in Table 9, about 16 per cent of plants or lines which were assessed as "stunted", though the degree of dwarfing of these plants or lines were mostly not so marked as in Kô A.

It is noted further that in the F_2 and F_3 generations of the cross between two highly susceptible varieties, Kô A and Tochigi Golden, almost all of the hybrid plants were severely infected by the disease and died out almost completely in early spring. This doubtless indicates that these two varieties have the same genetic constitution for disease reaction.

SUMMARY

Barley yellow mosaic is a disease caused by a soil-borne virus. It has recently become one of the most serious diseases, especially for two-rowed barleys, in Japan. This study aimed to contribute to the breeding

of barley varieties highly resistant to this mosaic. Screening for resistance, tests of the relation between disease symptoms and damage, varietal reactions to the viruses of different sources, and in particular genetic studies of resistance and susceptibility to the disease were carried out for eight years. The results obtained may be summerized as follows:

1. Japanese and European two-rowed barleys with good malting quality were all highly susceptible. But, a number of highly tolerant forms could be found among the six-rowed barleys originating from Japan, Korea, China and Turkey. A Chinese variety, Mokusekko 3, among others, proved to be the most promising for resistance breeding.
2. The higher the susceptibility of the variety, the more evident were reduction of plant height and number of tillers per plants.
3. A selected group of varieties exhibited differential reactions to the viruses of different locations, which suggested differentiation of viruses with different pathogenicity; but the majority of the varieties tested, including the highly resistant forms, showed little variation in reaction.
4. Mokusekko 3 expressed complete resistance at all locations and years. Its resistance is chiefly controlled by a partially dominant gene, *Ym*, which is linked with *K* for hooded awn on chromosome 4 with 29.37% recombination. This variety possesses, besides *Ym*, another gene which has a slight resistance effect.
5. Mihori Hadaka 3 has another partially dominant gene Ym_2 for its high resistance. The gene is located on chromosome 1 at a distance of 31.4 crossover units from *n* for naked kernel.
6. Kasaura Naked and Svanhals, both of which show moderate mosaic symptoms, were each found to have a gene for slight resistance. The minor resistance genes seemed to be responsible for the considerable tolerance of Bavarian and Turkey 2.
7. Kô A and Tochigi Golden, both of which are highly susceptible and often severely stunted and killed, proved to have each at least two alleles for susceptibility.

ACKNOWLEDGEMENT

The writers wish to express their sincere appreciation to Dr. E. R. Sears, University of Missouri, U. S. A. for his kindness in reading the manuscript and making valuable suggestions.

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