Nekken Shiryo No. 66

Genetic Information in Rice

November 1984

Tropical Agriculture Research Center
Ministry of Agriculture, Forestry and Fisheries
Japan

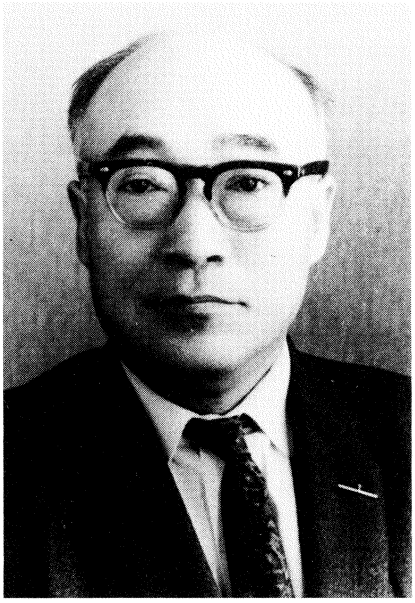
DEDICATION

The first volume of the Rice Genetics Newsletter is dedicated to: the late Dr. T. Morinaga, Dr. K. Ramiah, Dr. N. E. Jodon, and Dr. S. Nagao

T. Morinaga

K. Ramiah

N. E. Jodon



S. Nagao

The late Dr. T. Morinaga

Toshitaro Morinaga was born on September 13, 1985 at Uozu, Toyama, Japan, and graduated from the College of Agriculture, Tokyo Imperial University in 1919. He was an outstanding geneticist and plant breeder who made major contributions to our understanding of rice genetics, cytogenetics, cytotaxonomy and origin of cultivated rice. The first haploid plant of rice was obtained by him in 1930. He obtained rice triploids from the reciprocal crosses between diploid and tetraploid plants and isolated 8 trisomics from the progenies of triploids.

On the basis of cytogenetic analysis of interspecific hybrids, Dr. Morinaga proposed that *Oryza sativa, O. officinalis, O. minuta* and *O. latifolia* be assigned the genome symbols of AA, CC, BBCC and CCDD, respectively. He also concluded that although *O. sativa* and *O. glaberrima* have the same genome, they should be considered as distinct species. He classified the tropical rice cultivars into five ecotypes, namely, aus, aman, boro, bulu and tjereh, and hypothesized that the primary center of diversity of rice is in the region immediately to the southeast of Himalayas.

Dr. Morinaga detected linkage between genes for purple leaf color and liguleless condition in 1933 and studied linkage relations between many rice markers in subsequent years.

Dr. Morinaga trained numerous agricultural scientists when he was Professor at Kyushu University (1940—1951) and provided leadership in developing agricultural sciences as director of Central Agricultural Experiment Station (1946—1950) and as director of National Institute of Agricultural Sciences (1954—1961). He was a member of Japan Academy and the first president of the Japanese Society of Breeding. He passed in 1980 at Tokyo.

(T. Omura)

Dr. K. Ramiah

Dr. Krishnaswami Ramiah, a world renowned rice breeder and geneticist was born in 1892. He began his scientific career in 1914 when he joined the staff of the Paddy Breeding Station, Coimbatore. He was the first scientist in India to begin the systematic hybridization programme in rice. Prior the first crosses he made in 1917—18, rice varietal improvement in India was mainly limited to pureline selection. Dr. Ramiah was deeply interested not only in varietal improvement but in understanding the genetic basis of yield, pest resistance and grain quality. Soon after the discovery of Muller and Stadler of the mutation-inducing properties of X-rays, he initiated studies on X-ray induced variability in rice.

Dr. Ramiah was the founder director of the Central Rice Research Institute (CRRI), Cuttack. In 1949, he led the FAO-sponsored International Rice Commission. He inspired the FAO sponsored program on indica-japonica hybridization for developing nitrogen responsive varieties of rice for the tropics which resulted in the release of Mahsuri and Malinja in Malaysia and ADT 27 in India.

As FAO expert with headquarters in Bangkok, Dr. Ramiah travelled widely in rice growing countries and stimulated Government support for rice research and development. Dr. Ramiah is the author of *Rice in Madras* and *Rice Breeding and Genetics*. He was the first geneticist who advocated the standardization of gene symbols in rice. Dr. Ramiah has received numerous honors nationally and internationally.

(M. S. Swaminathan)

Dr. Nelson E. Jodon

Nelson Edgar Jodon was born on March 2, 1903 in a farmhouse near Sandusky, Ohio, USA. He graduated with honors from the College of Agriculture, University of Nebraska in 1929. He was appointed USDA Junior Agronomist and stationed at North Platte, Nebraska. He received the M. Sc. degree from the University of Nebraska in 1932 and later studied at Cornell University and University of Minnesota. In 1933, he was transferred to Rice Research Station, Crowley, Louisiana, where he engaged in rice varietal improvement work for over half a century. He developed several important rice varieties such as Magnolia, Lacrosse, Nato, Saturn, Della, Toro, LA110 and Toro 2.

Concurrently, he pioneered the studies on linkage groups of rice. He collected very useful marker gene stocks which included a set of linkage testers for 11 of the 12 linkage groups. His was the only collection of rice mutants in the USA. He also pioneered the studies on inheritance of disease resistance in rice.

At the invitation of FAO and in collaboration with R. Seethraman, M. Takahashi and others he prepared a proposal for the standardization of gene symbols in rice. This proposal was discussed and accepted at the 1959 meeting of the Working Party on Rice Production and Protection of the International Rice Commission held at Peradeniya, Sri Lanka. He also participated in the symposium on Rice Genetics held in 1963 at IRRI and the XIIth International Congress of Genetics held in 1968 at Tokyo and coordinated efforts to standardize the rice gene symbols internationally. In recognition of his outstanding contributions to rice genetics and breeding, Louisiana State University honored Dr. Jodon by conferring upon him the Doctor of Science degree. He is also the recipient of 11 other awards.

(M. Takahashi)

Dr. S. Nagao

Seijin Nagao was born in 1901 in Tokyo. He was the eldest son of Uzan Nagao, an eminent scholar of Chinese classics. He majored in plant breeding at the Agricultural College of Hokkaido Imperial University, and continued research in cytogenetics at Kyoto University as a graduate student. He obtained doctor's degree in science in 1933 with a thesis on polyploidy in narcissus which was considered a pioneering work on cytology of triploids.

Dr. Nagao joined the faculty of the Hokkaido University in 1935 as associate professor and was promoted to professor in 1939. He published "Genetics and Breeding in Rice" in 1935 which was the first book on rice genetics in Japan. In this book he proposed standardization of gene symbols. He started investigations on gene analysis in rice as one of main research activities of the Plant Breeding Institute. His earlier work was published in Advances in Genetics, Vol. 4 (1951), entitled "Genic analysis and linkage relationship of characters in rice". The genes for coloration of organs reported by him became widely known and were often used as markers in linkage studies. In 1963 he co-authored "Trial construction of twelve linkage groups in Japanese rice" with Manemon Takahashi. Dr. Nagao received the Japan Academy Prize in 1965, and was elected to be academician in 1973.

(M. Takahashi)

FOREWORD

The standardization of gene symbols for rice was first proposed by Dr. K. Ramiah during the World War II. The sixth meeting of the International Rice Commission (IRC), held in 1955, strongly recommended standardizing gene symbols and appointed a committee consisting of Mr. N. E. Jodon, Dr. N. Parthasarathy, and Dr. S. Nagao to formulate rules for gene symbolization in rice. Later, the 10th International Congress of Genetics held in 1958 at Montreal, Canada published the *Report of the International Committe on Gene Symbols and Nomenclature*. Following the ground rules of this report, the IRC Committee, assisted by Drs. M. Takahashi and R. Seetharaman, prepared rules and gene symbols for rice. Their report was published by the International Rice Commission in 1959 (*IRC Newsletter* 1959). Dr. C. Roy Adair had the report printed in the U. S. Department of Agriculture, Agric. Research Service Report Series (ARS34-28). Standardized gene symbols for rice were also discussed at the Symposium on Rice Genetics and Cytogenetics held at the International Rice Resarch Institute (IRRI) in February 1963 (*Rice Genetics and Cytogenetics*, Elsevier, Amsterdam, 1964, 274p.).

The rapid generation of new information on the rice genetics in recent years has resulted in the use of different symbols for the same genes and the same symbols for different genes. To promote cooperation and adoption of uniform gene symbols for rice in Japan, Dr. H. I. Oka organized an interim committee of Japanese scientists in 1979. The committee has met two or three times a year and promoted the adoption of rules of gene nomenclature suggested earlier. The gene symbols assigned since the publication of the IRC-recommended rules have been reviewed. In April 1981, the interim committee was named the Japanese Committee on Rice Gene Nomenclature and Linkage groups; it was supported by the Japanese Society of Breeding, and its meeting expenses were subsidized by the Institute of Physical and Chemical Research. In April 1984, the committee was renamed the Japanese Rice Genetics Information Committee. Its members are T. Matsuo, Chairman; Y. Futsuhara, Secretary; active members N. Iwata, F. Kikuchi, T. Kinoshita, H. Morishima, M. Nakagahra, and K. Takeda; and coordinating members S. Iyama, T. Kawai, T. Nakajima, H. I. Oka, T. Omura, M. Takahashi, K. Toriyama, and H. Yamagata.

At a committee meeting held at Kurashiki in June 1982, it was suggested by Dr. Ryuhei Takahashi to publish a rice genetics newsletter, like that for barley, on an international basis. In parallel, Dr. G. S. Khush wrote to Dr. T. Kinoshita in September 1982 about the need for an international organization to bring about uniformity of gene symbolization in rice, and proposed that an international workshop on rice genetics be held and that a rice genetics newsletter be published annually. These proposals were discussed and acknowledged at the October meeting of the Japanese committee. In January 1983, Dr. M. Takahashi asked a number of rice geneticists abroad for their opinions on the desirability of publishing a rice genetics newsletter and holding a rice genetics symposium. The responses were favorable and the matter was further discussed between Drs. M. S. Swaminathan, G. S. Khush, and H. I. Oka during Dr. Oka's visit to IRRI in April 1983. They agreed to publish the first issue of the *Rice Genetics Newsletter* (RGN) in 1984 under the editorship of Drs. Oka and Khush and to hold the International Rice Genetics Symposium (IRGS) in May 1985. Drs. Y. Futsuhara and T. Kinoshita visited IRRI in October 1983 and reviewed the agreements for RGN and IRGS with Drs. Swaminathan and Khush.

The first issue of the RGN is now put in print. On behalf of the Japanese Rice Genetics Information Committee, I express our sincere thanks to Dr. Sho-ichiro Nakagawa, Director General, Tropical Agriculture Research Center, Ministry of Agriculture, Forestry and Fisheries, Japan, for his generous help in printing the first issue as "Genetic information in rice".

For the future management and publication of RGN, however, an international organization is needed. It is hoped that during the IRGS, world rice geneticists will establish a formal Rice Genetics Cooperative to assume the responsibility for publishing RGN; monitoring rice gene symbolization; maintaining and exchanging gene stocks, and chromosomal mapping.

The publication of the first issue of RGN is a landmark in rice genetics, and I hope the RGN will be published annually. Each issue should report new findings on rice genes, linkage relations, and other aspects of rice genetics; and contain lists of available gene stocks, up-to-date linkage maps, and information on rice genetic resources. I hope the first issue of RGN will open the doors for cooperation among world rice geneticists.

T. Matsuo
Chairman, Japanese Rice Genetics
Information Committee

MESSAGE

Rice is the principal food of nearly half of mankind. Yet our knowledge of rice genetics lags behind that of other major food crops such as wheat, maize, barley and tomato. In the genetically well known species, newsletters are published annually for the informal exchange of preliminary information on findings of interest to geneticists. Availability of seed stocks of interest for genetic research is reported and such newsletters serve the useful purpose of regular communication between the research workers having a common interest in the genetics of the crop. I am very happy that rice geneticists have decided to publish an International Rice Genetics Newsletter (IRGN) annually. I hope that a formal organization for the publication of the IRGN will be established during the International Rice Genetics Symposium to be held at IRRI in May 1985. Understanding of basic genetics of rice will be imperative for utilizing emerging techniques of genetic engineering for rice improvement.

April 30, 1984

M. S. Swaminathan
Director General, IRRI
Independent Chairman, FAO Council

RICE GENETICS NEWSLETTER, VOL. 1

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E. RESEARCH NOTES

A. NOTICE AND ANNOUCEMENT

1. The Aim and Scope of the Rice Genetics Newsletter

The general aim of this Newsletter is to promote cooperation and exchange of information and material among rice geneticists. Emphasis will be laid on standardization of gene symbols and presentation of current linkage maps, in addition to reporting new findings in the genetic study of rice.

Each issue will contain A) Special notices, B) Current linkage maps, C) List of genes and genetic stocks, D) List of recent publications, E) Research notes, and F) Mailing list. The Newsletter will be published annually.

The research notes should consist of short articles (abstracts with necessary tables and references). Each article should contain information that might otherwise not be available to interested workers, even if it does not merit formal publication. Particular strains used as experimental material should be briefly described. New findings on genes, linkage relations, and useful techniques can logically be reported here. Articles are published primarily for the benefit of members, and none of information may be used in publications by others without the consent of the respective authors. Recent papers on rice genetics published in languages other than English can be summarized as a research note. Preliminary reports of research projects that might be published later as journal articles are also welcome.

Manuscripts should be sent to the Editor: Dr. H. I. Oka, National Institute of Genetics, Misima City, 411 Japan, or Dr. G. S. Khush, International Rice Research Institute, P.O. Box 933, Manila, Philippines.

For subscription, please write to the Secretary: Professor Y. Futsuhara, Faculty of Agriculture, Nagoya University, Chigusa-ku, Nagoya, 464 Japan.

(Y. Futsuhara)

2. Announcement: The International Rice Genetics Symposium

The Symposium will be held at the International Rice Research Institute (IRRI, Los Banõs, Philippines) in May 1985, and will be jointly sponsored by the IRRI and Japanese Rice Genetics Information Committee. The following topics will be discussed: 1) Systematics and evolution, 2) varietal differentiation and reproductive barriers, 3) rice karyotype, polyploids, aneuploids, and translocations, 4) genetic markers and linkage maps, 5) geographical distribution of genes, 6) genetics of physiological traits, 7) genetics of endosperm traits, 8) genetics of disease and insect resistance, 9) quantitative genetics, 10) cytoplasmic male sterility and restoration, 11) mutagenesis, 12) tissue and cell culture, 13) gene library for genetic engineering research, and 14) gene transfer techniques.

The proceedings will be published in a book form. An international committee for preparing the program has been established with the following membership: Co-chairmen: Drs. M. S. Swaminathan and M. Takahashi, Secretaries: Drs. G. S. Khush and H. I. Oka, Members: Drs. A. Abifarin (Liberia), T. T. Chang (IRRI), R. C. Chaudhary (India), M. H. Heu (Korea), M. Jacquot (France), Min Shao Kai (China), T. Kinoshita (Japan), M. Van Montagu (Belgium), J. N. Rutger (USA), B. H. Siwi (Indonesia), Ray Wu (USA), and S. M. H. Zaman (Bangladesh).

If you are interested in attending the Symposium, please write to one of the Secretaries at your earliest convenience. (G. S. Khush)

3. Proposal for Rules of Gene Symbolization

In 1963, the Committee on rice gene symbolization and linkage groups (chairman Dr. N. E. Jodon), appointed by the 6th (1955) Meeting of the Working Party on Rice Breeding, FAO International Rice Commission (IRC), proposed the general rules and standardized symbols for known genes. Since then, this report has served as a guide for rice geneticists. On account of new developments in rice genetics during the last two decades, however, the need for examination and revision of gene symbols and linkage groups has been progressively felt. Realizing this, the Japanese Rice Genetics Information Committee initiated the examination of the gene symbols and linkage groups. First, the Committee discussed the principles and accepted the following:

1) The symbols used should follow the above-mentioned international rules (given in Table 1) and the gene symbols thereby recommended (cf. Publ. List 1, 10, 19 & 32).

2) The symbols commonly used by many workers can be retained even if they do not fit the rules completely.

3) When a new gene is identified but its allelic relationships with previously reported mimic genes are not known, it is denoted by adding (t) to its symbol; (t means tentative). For examples, *d-50*(t).

4) Non-allelic loci (mimics, polymeric genes, etc.) are distinguished by a suffix letter or arabic numeral either on the same line after a hyphen or as a subscript.

5) When two or more different gene symbols have been used for the same gene, the one appearing in the earlier report is adopted so far as it satisfies the rules.

6) For revision of gene symbols used by an author, contact with the author to seek approval is necessary.

7) The list of genes includes those for which seed stocks are maintained.

The gene symbols recommended by the Committee are shown in the List that follows.

(T. Kinoshita)

Table 1. International rules adopted by ICG and the comments for application to rice genetics added by IRC

(quoted from Crop Research 1963)

1. In naming hereditary factors, the use of languages of higher internationality should be given preference. (English is and probably will continue to be the language most commonly used by rice geneticists.)

2. Symbols of hereditary factors, derived from their original names, should be written in Roman letters of distinctive type, preferably in italics, and be as short as possible. (Symbols may be based on a key word or on an adjective-noun combination.)

3. Whenever unambiguous, the name and symbol of a dominant begin with a capital letter and those of a recessive with a small letter. (Non-controversial.)

4. Literal or numeral superscripts are used to represent the different members of an allelic series. (Same as 'convention' number 1 of Kadam and Ramiah [6].)

5. Standard or wild type alleles are designated by the gene symbols with + as a superscript or by + with the gene symbol as a superscript. In formulae the + alone may be used. (It hardly could be said that there is either a standard or a wild type in rice, and therefore the first part of this rule does not seem to apply. The + sign could be used in formulae if desired.)

6. Two or more genes having phenotypically similar effects are designated by a common basic symbol. Non-allelic loci (mimics, polymeric genes, etc.) are distinguished by an additional letter or Arabic numeral either on the same line after a hyphen or as a subscript. Alleles of independent mutational origin may be indicated by a superscript. (Here rice geneticists might follow 'convention' numbers 2 and 3 of Kadam and Ramiah [6] in using literal subscripts for complementary genes and numeral subscripts for duplicate genes.)

7. Inhibitors, suppressors and enhancers are designated by the symbols *I, Su,* and *En,* or by *i, su,* and *en* if they are recessive, followed by a hyphen and the symbol of the allele affected. (This appears non-controversial.)

8. Whenever convenient, lethals should be designated by the letter *l* or *L,* and sterility and incompatability genes by *s* or *S.* (Would not be needed for albinos, which are always lethal.)

9. Linkage groups and corresponding chromosomes are preferably designated by Arabic numerals. ([In the past] Roman numerals have been used, but in the future this rule should be complied with.)

10. The letter X and Y are recommended to designate the sex chromosomes. (Does not apply.)

11. Genic formulae are written as fractions with the maternal alleles given first or above. Each fraction corresponds to a single linkage group. Different linkage groups written in numerical sequence are separated by semicolons. Symbols of unlocated genes are placed within parentheses at the end of the formula. In euploids and aneuploids the gene symbols are repeated as many times as there are homologous loci. (Non-controversial.)

12. Chromosomal aberrations should be indicated by the abbreviations: *Df* for deficiency, *Dp* for duplication, *In* for inversion, *T* for translocation, *Tp* for transposition. (Cytologists and cytogeneticists will have use for these symbols in future work with rice.)

13. The zygotic number of chromosomes is indicated by 2n, the gametic number by n and the basic number by x. (Non-controversial usage.)

14. Symbols of extra-chromosomal factors should be enclosed within brackets and precede the genic formulae. (Non-controversial usage.)

4. List of gene symbols recommended for rice

A		
	A^S, A^E, A, A^d, A^m	Anthocyanin activator (complementary action with *C*)
	$Acp\text{-}1^{-17}, Acp\text{-}1^{-9}, Acp\text{-}1^{-4}, Acp\text{-}1^{+4}, Acp\text{-}1^{+9}, Acp\text{-}1^{+12}, Acp\text{-}1^{+24}, Acp\text{-}1^{Nul}$ (*Acp-B*)	Acid phosphatase-1
	$Acp\text{-}2^{Fa}, Acp\text{-}2^{Sa}, Acp\text{-}2^{Nul}$ (*Acp-C*)	Acid phosphatase-2
	$Acp\text{-}3^{B}, Acp\text{-}3^{Nul}$	Acid phosphatase-3
	al-1 (al-K-1)	albino- 1
	al-2 (al-K-2)	albino- 2
	al-3 (al-K-3)	albino- 3
	al-4 (al-K-4)	albino- 4
	al-5 (al-K-5)	albino- 5
	al-6(t) (al-K-6)	albino- 6
	al-7(t) (al-K-7)	albino- 7
	al-8 (al-K-8)	albino- 8
	al-9(t) (al-K-9)	albino- 9
	al-10 (al-K-10)	albino-10
	alk	alkali degeneration
	An-1	Awn-1 (triplicate genes)
	An-2	Awn-2 (ditto)
	An-3	Awn-3 (ditto)
	An-4(t)	Awn-4
	as	asynapsis
B		
	bc-1	brittle culm-1
	bc-2	brittle culm-2
	bc-3	brittle culm-3
	bd-1,2	beaked lemma (duplicate genes)
	Bf	Brown furrows of hull
	bgl	bright green leaf
	Bh-a,b,c (Bh-1,2,3)	Black hull (complementary genes)
	bk	big grain
	bl-1	brown leaf spot-1
	bl-2 (bl-m)	brown leaf spot-2
	bl-3	brown leaf spot-3
	bl-4	brown leaf spot-4
	bl-5	brown leaf spot-5

bl-6	brown leaf spot-6
Bp	Bulrush-like panicle
Bph-1	Brown planthopper resistance-1
bph-2	brown planthopper resistance-2
Bph-3	Brown planthopper resistance-3
bph-4	brown planthopper resistance-4
Bsv (Bs)	Black streaked dwarf virus resistance

C

C^{Bs}, C^{B}, C^{Bp}, C^{Bt}, C^{Br}, C^{Bd} C^{Bk}, C^{Bc}, C^{Bm}	Chromogen for anthocyanin (complementary action with *A*)
$Cat\text{-}1^{1}$, $Cat\text{-}2^{2}$ *(Cat-A)*	Catalase-1
Ce-1,2,3,4	*Cercospora oryzae* resistance (multiple genes)
chl-1 (ch-1)	chlorina-1
chl-2 (ch-2)	chlorina-2
chl-3 (ch-3)	chlorina-3
chl-4 (ch-4)	chlorina-4
chl-5 (ch-5)	chlorina-5
chl-6 (ch-6)	chlorina-6
chl-7(t)	chlorina-7
Cl	Clustered spikelets
clw	claw shaped spikelets
cps	compact panicle sterile

D

d-1	daikoku dwarf
d-2	ebisu dwarf
d-3	bunketsu-waito of tillering dwarf (duplicate or triplicate genes)
d-4	bunketsu-waito of tillering dwarf (ditto)
d-5	bunketsu-waito of tillering dwarf (ditto)
d-6 (d-34)	ebisumochi dwarf or tankan-shirasasa dwarf
d-7	heiei-daikoku or cleistogamous dwarf
d-9	chinese dwarf
d-10 (d-15,d-16)	kikeibanshinriki or toyohikari-bunwai of tillering dwarf
d-11 (d-8)	shinkane-aikoku or nōrin-28 dwarf
d-12	yūkara dwarf
d-13	short grained dwarf
d-14 (d-10)	kamikawabunwai of tillering dwarf

d-17(t)	slender dwarf
d-18^{h}	hosetsu-waisei or akibare dwarf (multiple alleles)
d-18^{k} (d-25)	kotaketamanishiki dwarf (ditto)
d-19(t)	kamikawa dwarf
d-20	hayayuki dwarf
d-21	aomorimochi-14 dwarf
d-22(t)	jokei 6549 dwarf
d-23(t)	ah-7 dwarf
d-24(t)	m-7 dwarf
d-26(t)	7237 dwarf
d-27 (d-t)	bunketsuto of tillering dwarf
d-28 (d-C)	chokeidaikoku or long stemmed dwarf
d-29 (d-K-1)	short uppermost internode dwarf
d-30 (d-W)	waisei-shirasasa dwarf
d-31	taichung-155-irradiated dwarf
d-32 (d-K-4,d-12)	dwarf Kyushu-4
d-33 (d-B)	bonsaito dwarf
d-35(t)	tanginbozu dwarf
d-42(t)	liguleless dwarf
d-49(t)	reimei dwarf
d-50(t)	fukei 71 dwarf
d-51 (d-K-8)	dwarf Kyushu-8
d-52 (d-K-2)	dwarf Kyushu-2
D-53 (D-K-3)	Dwarf Kyushu-3
d-54 (d-K-5)	dwarf Kyushu-5
d-55 (d-K-6)	dwarf Kyushu-6
d-56 (d-K-7)	dwarf Kyushu-7
d-57 [d(x)]	dwarf
D-a,b (D-1,2)	Complementary dominant lethal (complementary genes)
da	double awns
dl (lop)	drooping leaf
Dn-1 (Dn)	Dense panicle-1
Dn-2	Dense panicle-2
dn-3	dense panicle-3
dp-1	depressed palea-1
dp-2	depressed palea-2
drp-1	dripping-wet leaf-1
drp-2	dripping-wet leaf-2

drp-3	dripping-wet leaf-3
drp-4	dripping-wet leaf-4
drp-5(t)	dripping-wet leaf-5
drp-6(t)	dripping-wet leaf-6
drp-7(t)	dripping-wet leaf-7
ds	desynapsis
du	dull endosperm
dw-1,2 (fh)	deep water tolerance (duplicate genes)

E

E-1	Heading date-1
E-2	Heading date-2
E-3	Heading date-3
Ef-1^{a},Ef-1^{b},Ef-1^{g}, Ef-1^{X} (E)	Earliness-1
Ef-2	Earliness-2
eg	extra glume
er (o)	erect growth habit
Est-1,Est-1^{Nul} (Est-D)	Esterase-1
Est-2^{S},Est-2^{F},Est-2^{Nul} (Est-E)	Esterase-2
Est-3^{S},Est-3^{F} (Est-J)	Esterase-3
Est-4^{S},Est-4^{F},Est-4^{Nul} (Est-H)	Esterase-4
eui	elongated uppermost internode

F

fc-1	fine culm-1
fc-2(t)	fine culm-2
fes-1	female sterile-1
Fes-2	Female sterile-2
fgl (fl)	faded green leaf
Fgr	Fragrant flower
fs-1 (fs)	fine stripe-1
fs-2	fine stripe-2

G

g-1 (g)	long sterile lemmas-1
G-2 (Gm,Gl)	Long sterile lemmas-2
ga-1	gametophyte gene-1
ga-2	gametophyte gene-2
ga-3	gametophyte gene-3
ga-4 (ga-A)	gametophyte gene-4
ga-5 (ga-B)	gametophyte gene-5
ga-6	gametophyte gene-6

ga-7	gametophyte gene-7
ga-8	gametophyte gene-8
ga-9	gametophyte gene-9
ga-10(t)	gametophyte gene-10
ge	giant embryo
gf	gold furrows of hull
gh-1	gold hull and internode-1
gh-2	gold hull and internode-2
gh-3	gold hull and internode-3
gl-1,2	glabrous leaf and hull (duplicate genes)
Glh-1	Green leafhopper resistance-1
Glh-2	Green leafhopper resistance-2
Glh-3	Green leafhopper resistance-3
glh-4	green leafhopper resistance-4
Glh-5	Green leafhopper resistance-5
Glh-6	Green leafhopper resistance-6
Glh-7	Green leafhopper resistance-7
gm-1,2,3 (pd)	gall midge resistance (triplicate genes)
Grh-1,2	Green rice leafhopper resistance (duplicate genes)
Gsv (Gs)	Grassy stunt virus resistance

H

Hbv (Rhb)	*Hoja blanca* virus resistance
He	*Helminthosporium oryzae* resistance
Hg	Hairy glume
Hl-a,b	Hairy leaf (complementary genes)

I

I-Bf	Inhibitor for brown furrows
I-Bph-1	Inhibitor for brown planthopper resistance
I-gm-1	Inhibitor for susceptibility to gall midge
I-Pl-1	Inhibitor for purple leaf-1 (duplicate or triplicate genes)
I-Pl-2	Inhibitor for purple leaf-2 (ditto)
I-Pl-3	Inhibitor for purple leaf-3 (ditto)
I-Pl-4	Inhibitor for purple pericarp-4 (duplicate genes)

I-Pl-5	Inhibitor for purple pericarp-5 (ditto)
I-Pl-6	Inhibitor for purple leaf (Pl^i)-6
I-Ps-a,b (I-Ps-1,2)	Inhibitor for purple stigma (complementary genes)

L

L-1-a,b (L-1-1,2)	Complementary dominant lethal-1 (complementary genes)
L-2-a,b (Lr-1-1,2)	Complementary dominant lethal-2 (complementary genes)
la	'lazy' growth habit
Lap-1 (Lap-E)	Leucine amino peptidase-1
lax (lx)	lax panicle
lg	liguleless
lgt	long twisted grain
Lh-a,b	Heavy pubescence (complementary genes)
lhd	leafy hull sterile-1
lhs-1 (op)	leafy hull sterile-2
lhs-2 (lhs)	slender grain
lk	'Fusayoshi' long grain
Lk-f	long lemma
lmx	long palea (duplicate genes)
lp-1,2	

M

m^a-*Ef-1*,m^b-*Ef-1*	modifier for *Ef-1* (multiple alleles)
M-Pi-z (Rb-6)	*Pyricularia oryzae* resistance (modifier for *Pi-z*)
Mdh-1 (Mdh-A)	Malate dehydrogenase-1
me	multiple embryos
Mi	Minute grain
mls-1,2	malformed lemma (duplicate genes)
mp	multiple pistils
ms-1 (sf)	male sterile-1
ms-2 (ms-d)	male sterile-2
ms-3 (ms-1)	male sterile-3
ms-4 (ms-2)	male sterile-4
ms-5 (ms-3)	male sterile-5
ms-6 (ms-4)	male sterile-6
ms-7(t)	male sterile-7
ms-8(t)	male sterile-8
ms-9(t)	male sterile-9

ms-10(t)	male-sterile-10
ms-11(t)	male-sterile-11
ms-12(t)	male-sterile-12
ms-13(t)	male-sterile-13
ms-14(t)	male-sterile-14
ms-15(t)	male-sterile-15
ms-16(t)	male-sterile-16
ms-17(t)	male-sterile-17

N

nal-1	narrow leaf-1 (triplicate genes with *nal-2* and *nal-3*)
nal-2	narrow leaf-2 (ditto)
nal-3 (nal-2 or nal-3)	narrow leaf-3 (ditto)
nal-4 (nal)	narrow leaf-4
nal-5 (nal-1)	narrow leaf-5
nbs	non-bearing of spikelets
nl-1	neck leaf-1
nl-2	neck leaf-2

O

ops	open hull sterile

P

P, P^k*,* P^c	Colored apiculus (complementary action with *C* and *A*)
Pa	Purple apiculus
Pau-a,b	Purple auricle
Pc-1,2	Purple coleoptile
pcs (ops-2)	parthenocarpy sterile
Pd	Pendant panicle
Pg-1,2,3	Purple glume
Pgi-1^1*,Pgi-*1^2 *(Pgi-A)*	Phosphoglucose isomerase-1
Pgi-2^1*,Pgi-*2^2 *(Pgi-B)*	Phosphoglucose isomerase-2
pgl	pale green leaf
Ph (Po)	Phenol staining
Pi-a	*Pyricularia oryzae* resistance-a
Pi-b (Pi-s)	*Pyricularia oryzae* resistance-b
Pi-f	*Pyricularia oryzae* resistance-f
Pi-i	*Pyricularia oryzae* resistance-i
Pi-k,Pi-K^s*,Pi-*k^p*,Pi-*k^m *(=Pi-m),Pi-*k^h	*Pyricularia oryzae* resistance-k

Pi-t	*Pyricularia oryzae* resistance-t
Pi-ta, $Pi\text{-}ta^{2}$, $Pi\text{-}ta^{n}$	*Pyricularia oryzae* resistance-ta
Pi-z, $Pi\text{-}z^{t}$	*Pyricularia oryzae* resistance-z
Pi-se-1 (Rb-1)	*Pyricularia oryzae* resistance-se (additive effect with three genes)
Pi-se-2 (Rb-2)	*Pyricularia oryzae* resistance-se (ditto)
Pi-se-3 (Rb-3)	*Pyricularia oryzae* resistance-se (ditto)
Pi-is-1 (Rb-4)	*Pyricularia oryzae* resistance-is (cumulative effect with two genes)
Pi-is-2 (Rb-5)	*Pyricularia oryzae* restistance-is (ditto)
Pi(t)	*Pyricularia oryzae* restistance
Pin-1	Purple internode
Pj-a,b,c,d	Purple junctura
Pjb	Purple junctura back
Pl, Pl^{w}, Pl^{i} *(Pl')*	Purple leaf
Pla	Purple leaf apex
Plg	Purple ligule
Plm(Pla)	Purple leaf margin
Pm-a,b,c,d (Sp)	Purple septum
Pmr(Plm)	Purple midrib
Pn	Purple node
Pnr-1,2,3	Purple nodal ring
$Pox\text{-}1^{0C}$, $Pox\text{-}1^{2A}$, $Pox\text{-}1^{4A}$, $Pox\text{-}1^{Nul}$ *(Px,Pe)*	Peroxidase-1
$Pox\text{-}2^{4C}$, $Pox\text{-}2^{Nul}$	Peroxidase-2
$Pox\text{-}3^{3C}$, $Pox\text{-}3^{5C}$	Peroxidase-3
Pr	Purple hull
Prp-a(Pp)	Purple pericarp (complementary action with *Prp-b*)
Prp-b (Pb)	Purple pericarp (ditto with *Prp-a*)
Ps-1	Purple stigma-1
Ps-2	Purple stigma-2
Ps-3	Purple stigma-3
Psh	Purple sheath
Pu-a,b,c,d	Purple pulvinus
Px	Purple leaf axil

R

R_{LB}^{4C}	Regulator gene for peroxidase
R_{LS}^{4C}	Regulator gene for peroxidase
Rc, Rc^{S}	Brown pericarp and seed coat
rcn	reduced culm number
Rcp^{2A}, Rcp^{4A}	Receptor gene for peroxidase
Rd	Red pericarp and seed coat (complementary action with *Rc*)

Reg-1^{2A}*,Reg-2*^{4A}*,Reg-3*^{2A}	Regulator gene for peroxidase
Rf-1	Pollen fertility restoration-1
Rf-2 (Rf-x)	Pollen fertility restoration-2
Rf-a,b,c	Pollen fertility restoration (complementary genes)
Rf-a',b',c',d'	Pollen fertility restoration (complementary genes)
Rf-j	Pollen fertility restoration-j
rfs	rolled fine striped leaf
ri	verticillate rachis
rk-1	round kernel-1
rk-2	round kernel-2
rl-1	rolled leaf-1
rl-2	rolled leaf-2
rl-3 (rl-1)	rolled leaf-3
rl-4 (rl-2)	rolled leaf-4
rl-5 (rl-3)	rolled leaf-5

S

s-a-1,2 (s-1,s-2)	hybrid sterility-a (duplicate genes)
s-b-1,2 (s-1,s-2)	hybrid sterility-b (duplicate genes)
s-c-1,2 (s-1,s-2)	hybrid sterility-c (duplicate genes)
s-d-1,2 (s-1,s-2)	hybrid sterility-d (duplicate genes)
s-e-1,2 (s-1,s-2)	hybrid sterility-e (duplicate genes)
S-1, S^{a}*-1*	Hybrid sterility-1 (one locus sporo-gametophytic lethal,multiple alleles)
S-2, S^{a}*-2*	Hybrid sterility-2 (one locus sporo-gametophytic lethal ,multiple alleles)
S-3, S^{a}*-3*	Hybrid sterility-3 (one locus sporo-gametophytic lethal,multiple alleles)
S-A-1,2 (A-1,2)	Hybrid sterility-A (duplicate fertility genes)
S-B-1,2 (B-1,2)	Hybrid sterility-B (duplicate feritlity genes)
Sb	Stem borer resistance
Sc-1,2	*Sclerotium oryzae* resistance (duplicate genes)
Scl	Superclustered spikelets
sd-1 (d-47)	dee-geo-woo-gen dwarf
sd-2	semidwarf-2
sd-3	semidwarf-3
sd-4	semidwarf-4

Sdr-a,b (Sd)	Seed dormancy (complementary genes)
Se-1^{e},Se-1^{n},Se-1^{t},Se-1^{s},Se-1^{u} (Lm,Lf,Rs)	Photosensitivity-1
se-2	photosensitivity-2
Sg	Permeability of testa to water
sh	shattering
Sh	Shattering
Shp (Ex)	Sheathed panicle
shr-1^{s}, shr-1^{a}	shrunken endosperm-1 (multiple alleles)
shr-2	shrunken endosperm-2
Sk	Scented kernel
sl	sekiguchi lesion
Sm (Rsm)	Stem maggot resistance
sn-1,2	sinuous neck (duplicate genes)
sp	short panicle
spr-1	spreading panicle-1
Spr-2 (E)	Spreading panicle-2
spl-1 (bl-7,bl-12)	spotted leaf-1
spl-2 (bl-13)	spotted leaf-2
spl-3 (bl-14)	spotted leaf-3
spl-4 (bl-15)	spotted leaf-4
spl-5 (bl-16)	spotted leaf-5
spl-6	spotted leaf-6
spl-7	spotted leaf-7
spl-8 (bl-8)	spotted leaf-8
st-1 (ws-1)	stripe-1
st-2 (gw)	stripe-2
st-3 (stl)	stripe-3
st-4 (ws-2)	stripe-4
Stv-a (St-1)	Stripe virus resistance (complementary genes)
Stv-b,Stv-b^{i} (St-2)	Stripe virus resistance (multiple alleles)
su	sugary endosperm
Su-g-1	Suppressor for long sterile lemmas

T

tri	triangular hull
ts-a,b	twisted stem (complementary genes)
Tw-a,b (Rtv)	Tungro virus resistance (complementary genes)

U

Ur-1 (Ur)	Undulate rachis-1
ur-2	undulate rachis-2
Un-a,b	Uneven grain (complementary genes)

V

v-1	virescent-1
v-1(t) (v-1)	virescent-1
v-2	virescent-2
v-3	virescent-3
v-4	virescent-4
v-5	virescent-5
v-6	virescent-6
v-7	virescent-7
v-8	virescent-8
v-9(t)	virescent-9
v-10(t)	virescent-10
v-11(t)	virescent-11

W

W-a,b (W-1,2)	Complementary dominant lethal-W (complementary genes)
Wh	White hull
Wph-1 (Wbph-1)	White-backed planthopper resistance-1
Wph-2 (Wbph-2)	White-backed planthopper resistance-2
Wph-3 (Wbph-3)	White-backed planthopper resistance-3
wph-4 (wbph-4)	white-backed planthopper resistance-4
Wph-5 (Wbph-5)	White-backed planthopper resistance-5
wx (cm)	glutinous endosperm

X

Xa-1,Xa-1^{h} (Xe-1)	*Xanthomonas oryzae* resistance-1 (multiple alleles)
Xa-2 (Xe-2)	*Xanthomonas oryzae* resistance-2
Xa-3 (Xa-w)	*Xanthomonas oryzae* resistance-3
Xa-4^{a},Xa-4^{b}	*Xanthomonas oryzae* resistance-4 (multiple alleles)
xa-5	*xanthomonas oryzae* resistance-5
Xa-6	*Xanthomonas oryzae* resistance-6
Xa-7	*Xanthomonas oryzae* resistance-7
xa-8	*xanthomonas oryzae* resistance-8
xa-9	*xanthomonas oryzae* resistance-9

Xa-10	*Xanthomonas oryzae* resistance-10
Xa-kg, *Xa-kg*^{h}	*Xanthomonas oryzae* resistance-kg (multiple alleles)

Y

Ydv (Ryd)	Yellow dwarf resistance
ylb	yellow banded leaf blade
ysl	yellow leaf spot

Z

z-1	zebra-1
z-2	zebra-2
z-3	zebra-3
z-4	zebra-4
z-5	zebra-5
zn	zebra necrosis

Cytoplasmic male sterility

[ms-bo]	Cytoplasm from 'Chinsurah boro II'
[ms-ld]	Cytoplasm from 'Lead rice'
[ms-TA]	Cytoplasm from 'TA 820'
[ms-CW]	Cytoplasm from Chinese wild rice
[ms-WA]	Cytoplasm, WA-group
[ms-HL]	Cytoplasm, HL-group
[ms-jp]	Cytoplasm from *japonica* cultivar 'Akebono'

B. CURRENT LINKAGE MAPS

A new linkage map of rice based on latest available information is presented in Fig. 1, with a list of genes of each linkage group.

In the review of rice genetics by Yamaguchi (1927), only four linkage groups, each consisting of two genes, were reported. Jodon (1948) reported on eight linkage groups involving nearly 50 genes. Since then, information on the location of different genes has accumulated in both the Indica and Japonica rices. In 1963, Nagao and Takahashi first proposed twelve linkage groups corresponding to the haploid number of chromosomes, on the basis of linkage data in Japonica varieties. Misro et al. (1966) also presented twelve linkage groups in the Indica rice. However, because of differences in the genic scheme for organ coloration and scarcity of identical genes involved in the two series of linkage groups at that time, it was difficult to establish twelve groups common to the Indica and Japonica types.

The cytological basis of linkage groups was first reported by Iwata and Omura (1971 a, b) from a study of relationships of gene loci with the points of interchange of reciprocal translocations. Ten linkage groups were then corresponded to different chromosome. Sato et al. (1973) detected the association of the 3rd linkage group with chromosomes 3, thus modifying the relationship established earlier. A series of primary trisomics were established by different workers (Hu 1968; Iwata et al. 1970, 1984; Watanabe and Koga 1975; Kawaguchi et al. 1982; Khush et al. 1984). The extra chromosomes of the trisomics were identified by examining the somatic karyotype of trisomics by Kurata et al. (1981) and at pachytene stage of meiosis by Khush et al. (1984). On the basis of trisomic analysis, the correspondence between linkage groups and chromosomes was partly revised by Iwata and Omura (1976b). The 5th and 7th linkage groups were associated with chromosome 1, and the 6th, 9th and 12th groups with chromosome 2. More recently Khush et al. (1984) established associations between twelve linkage groups and cytologically identifiable chromosomes through the trisomic technique.

The linkage map presented in Fig. 1 includes amendments to the Takahashi-Kinoshita's (1977) map made on the basis of recent findings. The 6th and 9th groups have been combined since the points of interchange connecting the two groups were detected by Sato et al. (1982). However, there is no definite map combining the markers of the 5th and 7th groups. The 12th group was retained except that *gl-1* and *An-2* were shifted to the 6+9th group. *Hg* and *d-20* belonging to the 12th group have not been subjected to trisomic analysis.

Different systems of numbering the chromosomes and linkage groups have been employed by different authors. The relationships among linkage groups, chromosomes and trisomics as determined by different worker are presented in Table 1, Khush et al. (Res. note no. 32) and Table 1, Iwata et al. (no. 34). The chromosome numbering system in the table still follows that of Nishimura (1961).

With the establishment of induced translocation homozygotes, the translocation analysis has also progressed, and chromosome maps have been proposed for ten chromosomes (Sato et al. 1980). The relative positions of breakage points, centromeres and several marker genes in each chromosome are shown in Fig. 2.

Following matters in relation to rice linkage maps need further attention.

1. There is no consistency in the numbering of linkage groups, chromosomes, and the trisomics. We propose to revise the numbering system for chromosomes and the linkage groups on the

basis of discussions among rice geneticists during the forthcoming Rice Genetic symposium.

2. The allelism tests between marker genes with similar effects are most important. For this purpose, exchange of gene stocks and information among rice workers must be promoted.

3. Multiple marker stocks, induced mutants and cytogenetic materials are being developed by different workers. Easily identifiable mutants, even though they are of no immediate economic value, are useful in linkage studies.

These useful materials are sometimes lost on account of difficulty of seed maintenance. It is hoped that arrangement will be made to preserve these stocks in certain centers of germplasm conservation.

4. A complete set of primary trisomics has already been established in both the Indica and Japonica rices (Khush et al. 1984; Iwata et al. 1984). Rice karyotypes have been described by Hu (1958), Shastry et al. (1960), Kurata and Omura (1978), Kurata et al. (1981), Chen and Wu (1984), Chen et al. (1982), and Khush et al. (1984). The extensive use of cytological mutants such as telo and tertiary trisomics and induced deficiencies should be encouraged to locate the gene loci on the respective arms of chromosomes.

5. The linkage information should be utilized in the breeding programs. Some of the genes for short stature, heading date, disease and insect resistances, and grain chracters have been mapped. Linkage between some marker characters and cold tolerance and germinability at low temperatures has been detected (Futsuhara and Toriyama, 1966; Takahashi, 1977).

I wish to acknowledge the assistance of Drs. N. Iwata and S. Sato in preparing the linkage maps. References used are listed in Publication List, 1. Genic Analysis.

(Toshiro Kinoshita)

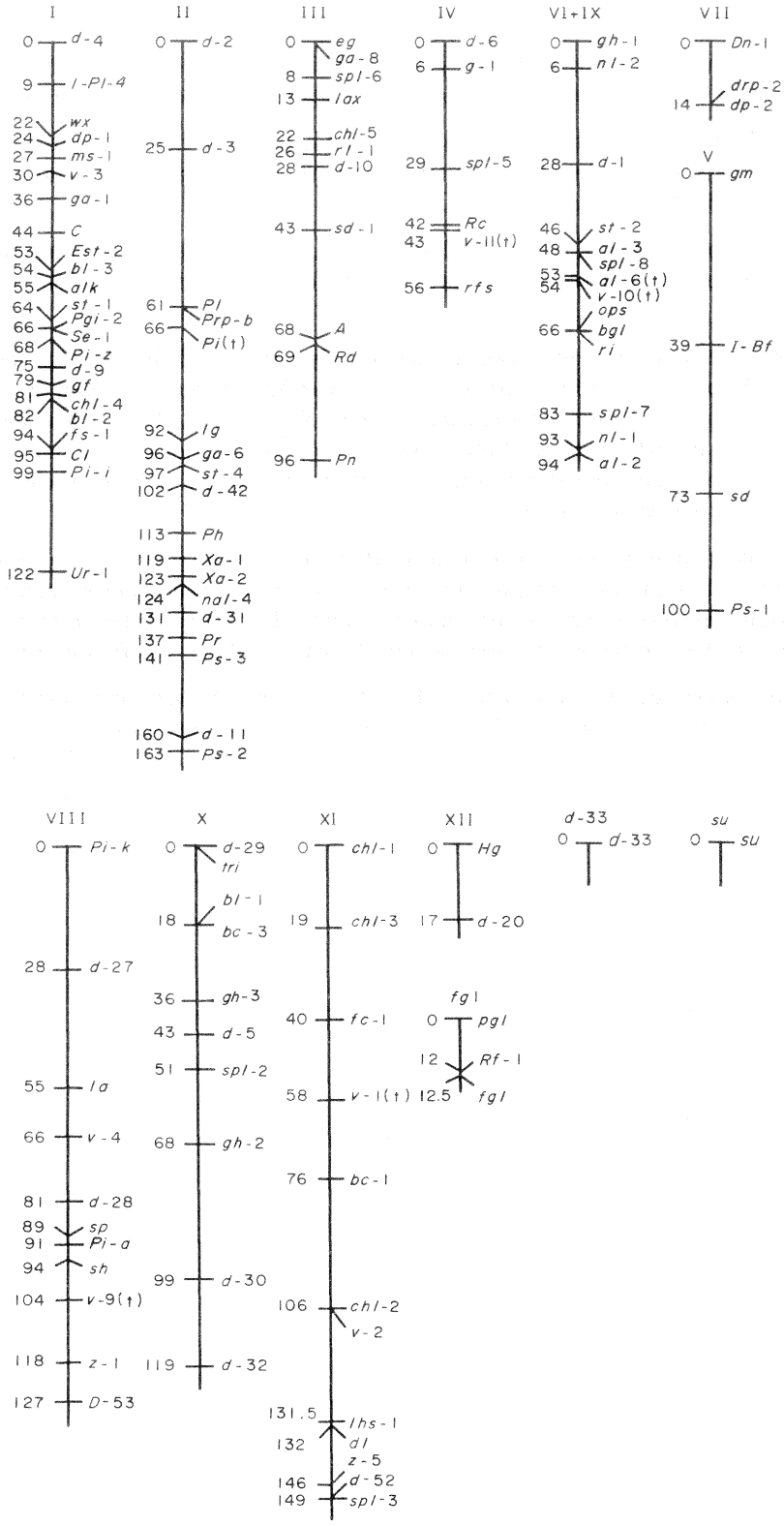

Fig. 1. 1984 linkage map of rice.

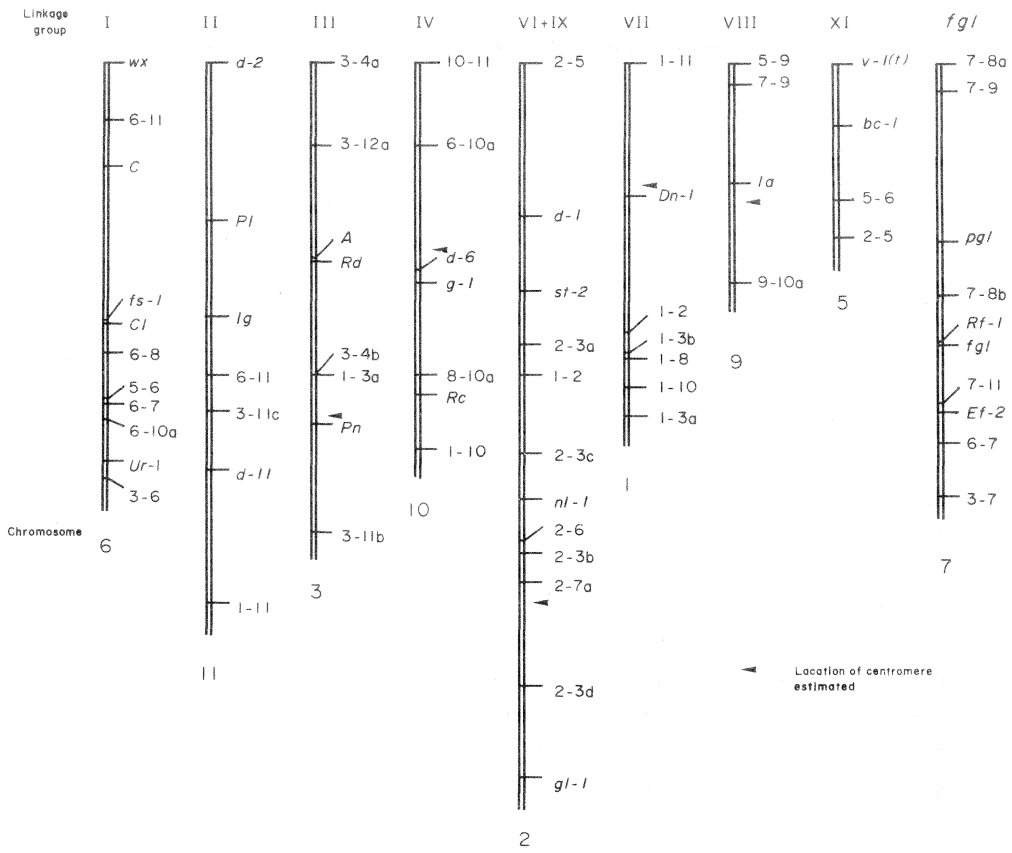

Fig. 2. Cytological map of rice based on pachytene analysis of translocation heterozygotes.

List of marker genes belonging to the linkage groups

Gene	Name	Gene locus
Group I (*wx* Group), chromosome 6		
d-4	bunketsu-waito of tillering dwarf	0
I-Pl-4	Inhibitor for purple pericarp-4	9
wx (am)	glutinous endosperm	22
dp-1	depressed palea-1	24
ms-1 (sf)	male sterile-1	27
v-3	virescent-3	30
ga-1	gametophyte gene-1	36
C	Chromogen for anthocyanin	44
Est-2	Esterase-2	53
bl-3	brown leaf spot-3	54
alk	alkali degeneration	55
st-1 (ws)	stripe-1	64
Pgi-2	Phosphoglucose isomerase-2	66
Se-1 (Lf,Lm,Rs)	Photosensitivity-1	66
Pi-z	*Pyricularia oryzae* resistance-1	68
d-9	chinese dwarf	75
gf	gold furrows of glume	79
chl-4 (ch-4)	chlorina-4	81
bl-2	brown leaf spot-2	82
fs-1	fine stripe-1	94
Cl	Clustered spikelets	95
Pi-i	*Pyricularia oryzae* resistance-i	99
Ur-1	Undulated rachis-1	122
Unlocated genes		
al-1 (al-K-1)	albino-1	7.1%-*wx*
al-9(t) (al-K-9)	albino-9	trisomic B
chl-7(t)	chlorina-7	27%-*Pi-z*
d-21	aomorimochi-14 dwarf	8.3%-*wx*
ga-4 (ga-A)	gametophyte gene-4	34%-*wx*
ga-5 (ga-B)	gametophyte gene-5	27%-*wx*
Hl-a	Hairy leaf	21%-*fs-1*
I-Pl-2	Inhibitor for purple leaf-2	10%-*I-Pl-4*
rcn	reduced culm number	32%-*C*
S-1	Hydrid sterility (one locus sporo-gametophytic lethal)	close to *C*

s-a-1 (s_1,x)	hybrid sterility-a (duplicate gametophytic lethal)	21%-*wx*
s-b-1	hybrid sterility-b	18%-*wx*
s-c-1	hybrid sterility-c	8.6%-*C*
s-d-1	hybrid sterility-d	33%-*wx*
S-A-1 (A-1)	Hybrid sterility-A (duplicate fertility genes)	9.5%-*C*
S-B-2 (B-2)	Hybrid sterility-B	28%-*wx*
spl-4 (bl-15)	spotted leaf-4	2.5%-*dp-1*
Stv-a (St-1)	Stripe virus resistance	38%-*wx*
Un-a	Uneven grain	22%-*Cl-a*
v-1	virescent-1	25%-*C*
zn	zebra necrosis	20%-*C*

Group II (*Pl* group), chromosome 11

d-2	ebisu dwarf	0
d-3	bunketsu-waito of tillering dwarf	25
Pl (Pl-1)	Purple leaf	61
Prp-b (Pb)	Purple pericarp	61
Pi(t)	*Pyricularia oryzae* resistance	66
lg	liguleless	92
ga-6	gametophyte gene-6	96
st-4 (ws-2)	stripe-4	97
d-42	liguleless dwarf	102
Ph (Po)	Phenol staining	113
Xa-1 (Xe-1)	*Xanthomonas oryzae* resistance-1	119
Xa-2 (Xe-2)	*Xanthomonas oryzae* resistance-2	123
nal-4 (nal)	narrow leaf-4	124
d-31	taichung-155 irradiated dwarf	131
Pr	Purple hull	137
Ps-3	Purple stigma-3	141
d-11 (d-8)	sinkane-aikoku or norin-28 dwarf	160
Ps-2	Purple stigma-2	163

Unlocated genes

al-5 (al-K-5)	albino-5	34%-*lg*
al-7 (t) (al-K-7)	albino-7	31%-*lg*
An-1	Awn-1	5.4%-*d-11*
Bph-1	Brown planthopper resistance-1	trisomic E
bph-2	brown planthopper resistance-2	39%-*d-2*

drp-1	dripping-wet leaf-1	39%-*d-2*
drp-5(t)	dripping-wet leaf-5	17%-*lg*
ga-10(t)	gemetophyte gene-10	27%-*lg*
nal-1	narrow leaf-1	25%-*d-2*
nal-5 (nal-1)	narrow leaf-5	9.5%-*lg*
P	Purple apiculus	2.7%-*Pl*
Pin-1	Purple internode-1	31%-*Pl*
rk-1	round kernel-1	35%-*lg*
rl-2	rolled leaf-2	35%-*d-2*
s-c-2	hybrid sterility-c	31%-*Ph*
s-e-2	hybrid sterility-e	15%-*lg*
Sc-1	*Sclerotium oryzae* resistance	26%-*lg*
ssk (sk)	malformed semi-sterile	6.8%-*Pl*
Wh	White hull	8.0%-*lg*
Xa-kg	*Xanthomas oryzae* resistance-kg	2.1%-*Xa-1*
ylm	yellow leaf margin	10%-*lg*
z-5	zebra-5	11%-*lg*

Group III (*A* group), chromosome 3

eg	extra glume	0
ga-8	gametophyte gene-8	0
spl-6	spotted leaf-6	8
lax (lx)	lax panicle	13
chl-5 (ch-5)	chlorina-5	22
rl-1	rolled leaf-1	26
d-10 (d-15,d-16)	kikeibanshinriki or toyohikari-bunwai of tillering dwarf	28
sd-1 (d-47)	dee-geo-woo-gen dwarf	43
A	Anthocyanin activator	68
Rd	Red pericarp	69
Pn	Purple node	96

Unlocated genes

al-4 (al-K-4)	albino-4	13%-*lax*
al-8 (al-K-8)	albino-8	11%-*d-18*
bph-4	brown planthopper resistance-4	close to *Bph-3*
chl-6 (ch-6)	chlorina-6	31%-*lax*
d-18 (d-25)	hosetsu dwarf and kotaketamanishiki dwarf	0.6%-TR3-8b
d-26(t)	7237 dwarf	37%-*A*

d-54 (d-K-5)	dwarf-Kyushu-5	30%-*rl-4*
d-55 (d-K-6)	dwarf-Kyushu-6	12%-*eg*
fs-2	fine stripe-2	13%-*d-18*
ga-7	gametophyte gene-7	29%-*A*
ga-9	gametophyte gene-9	0.6%-*d-18*
Glh-3	Green leafhopper resistance-3	34%-*bph-4*
I-Ps-b	Inhibitor for purple stigma	linked with *A*
lgt	long twisted grain	16%-*d-26*
Prp-a (Pp)	Purple pericarp	7.3%-*A*
rl-4 (rl-2)	rolled leaf-4	20%-*A*
shr-1	shrunken endosperm	24%-*rl-4*
ts-a	twisted stem	23%-*A*
v-6	virescent-6	27%-*lax*

Group IV (*g-1* group), chromosome-10

d-6	ebisumoshi or tankanshirasasa dwarf	0
g-1 (g)	long strile lemmas-1	6
spl-5 (bl-6)	spotted leaf-5	29
Rc	Brown pericarp and seed coat	42
v-11(t)	virescent-11	43
rfs	rolled fine striped leaf	56

Unlocated genes

d-7	heieidaikoku or cleistogamons dwarf	39%-*d-6*
ge	giant embryo	trisomic-F
lp-1	long palea	12%-*Un-b*
m-Ef-1	modifier for *Ef-1*	23%-*Rc*
se-2	photosensitivity-2	23%-*g-1*
Un-b	Uneven grain	18%-*g-1*

Group VI+IX (*d-1* group), chromosome-2

gh-1	gold hull and internode-1	0
nl-2	neck leaf-2	6
d-1	daikoku dwarf	28
st-2 (gw)	stripe-2	46
al-3 (dl-K-3)	albino-3	48
spl-8 (bl-8)	spotted leaf-8	48
al-6(t) (al-K-6)	albino-6	53
v-10(t)	virescent-10	54
ops (ops-1)	open spikelet sterile	66

bgl	bright green leaf	66
ri	verticillate rachis	66
spl-7	spotted leaf-7	83
nl-1	neck leaf-1	93
al-2 (al-K-2)	albino-2	94

Unlocated genes

An-2	Awn-2	33%-*gl-1*
bd	beaked lemma	22%-*gl-1*
er (0)	erect growth habit	38%-*gh-1*
eui	elongated uppermost internode	27%-*nl-1*
gl-1	glabrous leaf blade-1	12%-RT2-3d
I-Pl-1	Inhibitor for purple leaf-1	31%-*gh-1*
v-10(t)	virescent-10	12%-*ri*
xa-5	*xanthomonas oryzae* resistance-5	trisomic L
ylb	yellow banded leaf blade	32%-*nl-1*

Group VII (*Dn-1* group), chromosome-1

Dn-1 (Dn)	Dense panicle-1	0
drp-2	dripping-wet leaf-2	14
dp-2	depressed palea-2	14

Unlocated genes

Bp	Burlush-like panicle	trisomic H
d-57 [d(x)]	dwarf	21%-*Dn-1*
Pi-ta	*Pyricularia oryzae* resistance-ta	4.5%-RT1-4
sl	sekiguchi lesion	10%-*Pi-ta*

Group V (*I-Bf* group), chromosome-1 *(I-Bf)*

gm (pd)	gall midge resistance	0
I-Bf	Inhibitor for brown furrows of glume	39
sd	semidwarf	73
Ps-1	Purple stigma-1	100

Group VIII (*la* group), chromosome 9

Pi-k	*Pyricularia oryzae* resistance-k	0
d-27 (d-t)	bunketsuto of tillering dwarf	28
la	'lazy' growth habit	55
v-4	virescent-4	66
d-28 (d-C)	chokeidaikoku or long stemmed dwarf	81
sp	short panicle	89

Pi-a	*Pyricularia oryzae* resistance-a	91
sh	shattering	94
v-9(t)	virescent-9	104
z-1	zebra-1	118
D-53 (D-K-3)	Dwarf Kyushu-3	127

Unlocated genes

drp-7(t)	dripping-wet leaf-7	trisomic G
Ef-1 (E)	Earliness-1	38%-*la*
nal-2	narrow leaf-2	36%-*la*
Pi-f	*Pyricularia oryzae* resistance-f	15%-*Pi-k*
Pi-se-1 (Rb-1)	*Pyricularia oryzae* resistance-se	9.5%-*la*
Pi-is-1 (Rb-4)	*Pyricularia oryzae* resistance-is	23%-*la*
S-3	Hybrid sterility-3	1%-*la*
z-2	zebra-2	5.9%-*d-27*

Group X (*bl-1* group), chromosome-8

d-29 (d-K-1)	short uppermost internode dwarf	0
tri	triangular hull	0
bl-1	brown leaf spot-1	18
bc-3	brittle culm-3	18
gh-3	gold hull and internode-3	36
d-5	bunketsu-waito of tillering dwarf	43
spl-2 (bl-13)	spotted leaf-2	51
gh-2	gold hull and internode-2	68
d-30 (d-W)	waisei-shirasasa dwarf	99
d-32 (d-K-4)	dwarf Kyushu-4	119

Unlocated genes

d-29 (d-K-1)	short uppermost internode dwarf	14%-*bl-1*
Pi-b (Pi-s)	*Pyricularia oryzae* resistance-b	5.8%-RT7-8

Group XI (*bc-1* group), chromosome 5

chl-1 (ch-1)	chlorina-1	0
chl-3 (ch-3)	chlorina-3	19
fc-1	fine culm-1	40
v-1(t) (v-1)	virescent-1	58
bc-1	brittle culm-1	76
chl-2 (ch-2)	chlorina-2	106
v-2	virescent-2	106
lhs-1 (op)	leafy hull sterile-1	131.5

dl (lop)	drooping leaf	132
z-3	zebra-3	146
d-52 (d-K-2)	dwarf Kyushu-2	149
spl-3 (bl-14)	spotted leaf-3	149

Unlocated genes

An-3	Awn-3	38%-*bc-1*
al-10 (al-K-10)	albino-10	22%-*dl*
bl-4	brown leaf spot-4	29%-*bc-1*
d-14 (d-10)	kamikawa bunwai of tillering dwarf	32%-*dl*
d-56 (d-K-7)	dwarf Kyushu-7	7.2%-*dl*
drp-3	dripping-wet leaf-3	22%-*dl*
drp-4	dripping-wet leaf-4	6.0%-*dl*
ga-2	gametophyte gene-2	11%-*dl*
ga-3	gametophyte gene-3	34%-*dl*
Lk-f	'Fusayoshi' long grain	19%-*bc-1*
Mi	Minute grain	24%-*Lk-f*
rl-5 (rl-3)	rolled leaf-5	13%-*chl-1*
s-e-1	hybrid sterility-e	16%-*bc-1*
st-3 (stl)	stripe-3	1.1%-*bc-1*
v-5	virescent-5	2.0%-*chl-1*
v-7	virescent-7	1.7%-*bc-1*

Group XII (*Hg*-group)

Hg	Hairy glume	0
d-20	hayayuki dwarf	17

Unlocated gene

lhs-2 (lhs)	leafy hull sterile-2	8.2%-*Hg*

fgl-group, chromosome-7

pgl	pale green leaf	0
Rf-1	pollen ferility restoration-1	12
fgl (fl)	faded green leaf	12.5

Unlocated genes

Bph-3	Brown planthopper resistance-3	trisomic C
bph-4	brown planthopper resistance-4	30%-*rk-2*
du	dull endosperm	trisomic C
Ef-2	Earliness-2	7.8%-RT3-7
Glh-3	Green planthopper resistance-3	34%-*bph-4*

rk-2	round kernel-2	2.5%-RT7-9
d-33 group, chromosome 4 (Unlocated genes)		
d-33 (d-B)	bonsaito dwarf	trisomic A
nal-3 (nal-2)	narrow leaf-3	19%-RT3-4b
rl-3 (rl-1)	rolled leaf	13%-RT4-12
spl-1 (bl-12)	spotted leaf-1	1.7%-RT3-4a
su-group, chromosome-12 (Unlocated genes)		
An-4(t)	Awn-4	5.0%-RT10-12b
d-51 (d-K-8)	dwarf Kyushu-8	trisomic D
Stv-b (St-2)	striped virus resistance	linked with RT3-12
su	sugary endosperm	trisomic D
ur-2	undulate rachis-2	trisomic D
v-8	virescent-8	trisomic D
z-4	zebra-4	trisomic D

C. LIST OF GENES AND GENETIC STOCKS

This list was compiled on the basis of survey made by several rice geneticists in Japan and information obtained from the International Rice Research Institute, Los Baños and National Chung Hsing University, Taichung. Therefore, the list is incomplete, and we hope more complete lists will be presented in the 2nd and later issues of RGN. The institutions maintaining the listed stocks are shown by codes which are explained below.

The genetic stocks listed are classified into four major categories: marker genes for 14 different character groups, primary trisomics, reciprocal translocation lines, and isogenic lines. Strains of wild species and many of induced mutants are not included in the present list, although a small list of induced mutants from *Oryza glaberrima* is added to the list of isogenic lines.

Institution code:

CA: Chugoku National Agricultural Experiment Station, Fukuyama, Hiroshima-ken, 721 Japan

CH: Food Crops Research Institute, National Chung Hsing University, Taichung, Taiwan 400, ROC

GI: Genetic Stocks Center, National Institute of Genetics, Misima, 411 Japan

HK: Plant Breeding Institute, Faculty of Agriculture, Hokkaido University, Kita 9, Sapporo, 060 Japan

IR: Rice Germplasm Center, International Rice Research Institute, P.O. Box 933, Manila, Philippines

KA: Kyushu National Agricultural Experiment Station, Chikugo, Fukuoka-ken, 833 Japan

KT: Plant Breeding Laboratory, Faculty of Agriculture, Kyoto University, Kitashirakawa, Sakyo-ku, Kyoto, 606 Japan

KY: Plant Breeding Laboratory, Faculty of Agriculture, Kyushu University, Hakozaki, Fukuoka, 812 Japan

NA: National Institute of Agrobiological Resources, Tsukuba Science City, Ibaraki, 305 Japan

NG: Faculty of Agriculture, Nagoya University, Chikusa-ku, Nagoya, 464 Japan

OF: Genetics and Plant Breeding Laboratory, College of Agriculture, Osaka Prefecture University, Sakai, 591 Japan

OK: Institute for Agricultural and Biological Science, Okayama University, Kurashiki, 710 Japan

RY: Plant Breeding Laboratory, College of Agriculture, University of Ryukyus, Senbaru, Nishihara-cho, Okinawa, 903-01 Japan

TH: Plant Breeding Laboratory, Faculty of Agriculture, Tohoku University, Tsutsumidori, Sendai, 980 Japan

YA: College of Agriculture, Yamagata University, Tsuruoka, 997 Japan

(T. Kinoshita)

1. Genes for coloration

Gene symbol	Character	Linkage* group	Chromosome	Strain (Institution)	Reference
A^S	Anthocyanin activator (purple apiculus in the complementary action with *C* and *P*, multiple alleles)	III	3	I-33 Surjamukhi(HK)	66,157,172,248, 276,280
A^E				E 44 Pirurutong(HK)	
A				A-58 Kokushokutō-2	
A^d				A-83 Nōrin-20gō(HK)	
A^m				A-43 Hokkaimochi-1gō(HK)	
A^+					
Bf	Brown furrows of hull			Most of *japonica*	68.172,231
I-Bf	Inhibitor for brown furrows	V	1	A-5 Akamuro(HK)	
Bh-a	Black hull (complementary genes)			H-478 tester(HK)	109,144,151,170
Bh-b				H-478 do. (HK)	
Bh-c(Ph?)		II	11	H-478 do. (HK)	
C^{Bs}	Chromogen for anthocyanin (Tawny color apiculus in the complementary action with *P* and purple apiculus with *A* and P_y multiple alleles)	I	6	I-33 Surjamukhi(HK)	67,157,172,248, 276,280
C^B				A-13 Chabo(HK)	
C^{Bp}				A-1 Akage(HK)	
C^{Bt}				A-103 Tanpaku(HK)	
C^{Br}				A-5 Akamuro(HK)	
C^{Bd}				I-47 Dalashaita(HK)	
C^{Bk}				I-33 Karalath(HK)	
C^{Bc}				I-45 Charnock(HK)	
C^{Bm}				A-43 Hokkaimochi-1gō(HK)	
$C^+=C^{Bm}$					
gf	gold furrows of hull	I	6		90
gh-1	gold hull and internode-1	VI+IX (VI)	2	H-75 Ōkasshoku(HK)	88,170,172
gh-2	gold hull and internode-2	X	8	HO-550 Miyazaki No.3 (KY)	67,73
gh-3	gold hull and internode-3	X	8	M-93 Nōrin-8 mutant(KY)	71
P	Colored apiculus (purple apiculus in the complementary action with *C* and *A*, multiple alleles)	II	11	I-32 Karalath(HK)	157,172,248,276, 280
P^K				I-45 Charanock(HK)	
P^C				A-58 Kokushokutō-2(HK)	
P^+				H-61 Fusenshiro(HK)	

Pa	Purple apiculus	(III)		*indica type*	20, 154,216
Pau	Purple auricle	(III)		do.	20,23,25,26,154
Pc	Purple coleoptile	(III)		do.	20,22,26,154
Pg	Purple glume	(II,III,IV V,IX)		do.	20,21.23 24,154
Pin-1	Purple internode-1	II (III,IV)	11	I-33 Surjamukhi(HK) *indica type*	154,280,335
Pj	Purple junctura	(IV,V)		do.	22,23,25,26,154
Pjb	Purple junctura back	(X)		do.	154,334
Pl	Purple leaf	II	11	A-77 Shitō(HK), HO-725,729 Shitō(KY)	21,67,172 178,276,280
Pl^w	Purple leaf and pericarp			H-120(HK)	174,178,280
Pl^i*(Pl')*	Purple leaf			I-102 Fully purple(HK)	111,280
I-Pl-1	Inhibitor for purple leaf (triplicate genes)	VI+IX	2	H-97(HK)	172,174,276,280
I-Pl-2		I	6	E-44 Pirurutong(HK)	
I-Pl-3					
I-Pl-4	Inhibitor for purple pericarp (duplicate genes)	I	6	H-190(HK)	280,282
I-Pl-5				do.	
I-Pl-6	Inhibitor for purple leaf(Pl^i)			Most of *japonica*	111,280
Pla	Purple leaf apex	(IV)		*indica type*	154,155
Plg	Purple ligule	(II)		do.	21,23,26,154
Plm(Pla)	Purple leaf margin	(II)		do.	21,154
Pm (Sp)	Purple spetum	(III)		do.	20,26,154
Pmr(Plm)	Purple midrib	(II)		do.	21,154
Pn	Purple node	III	3	A-58 Kokushokutō-2(HK), HO-850 Kokutō(KY)	23,66,172,276,280
Pnr	Purple nodal ring			*indica* type	23
Pr	Purple hull	II	11	A-13 Chabo(HK) HO-850 Kokutō(KY)	23,160,276,280
Prp-a(Pp)	Purple pericarp (complementary genes)	III (V)	3	M-514 Hun-nou	54,154
Prp-b(Pb)		II	11		
Ps-1	Purple stigma-1	V (III,IV,IX)		E-39 Gaisenmochi(HK)	24,154,216,231, 277,280
Ps-2	Purple stigma-2	II	11	Taichung 65, A-58 Kokushokutō-2(HK)	50,51
Ps-3	Purple stigma-3	II	11		50,51
I-Ps-a	Inhibitor for purple stigma (complementary genes)	(VII)		Taichung 65, H-59(HK)	51
I-Ps-b		III	3		
Psh	Purple leaf sheath	(II,III,V)		*indica* type	20,26,97,154
Pu	Purple pulvinus	(III)		do.	25, 154
Px	Purple leaf axil	(II,III)		do.	25,26,154

Rc	Brown pericarp and seed coat (multiple alleles)	IV	10	A-5 Akamuro(HK) HO 745 Kuromoro(KY)	109,172,289
Rc^s				I-33 Surjamukhi(HK)	109,289
Rd	Red pericarp and seed coat (complementary action with *Rc*)	III	3	A-5 Akamuro(HK)	109,172
Wh	White hull	II	11	L-11 White hull(HK)	88,172

2. Chlorophyll aberration

al-1(al-K-1)	albino-1	I	6	Al 9 Norin 8 muant(KY)	72
al-2(al-K-2)	albino-2	VI+IX	2	Al 12 do. (KY)	72
al-3(al-K-3)	albino-3	VI+IX	2	Al 15 do. (KY)	72
al-4(al-K-4)	albino-4	III	3	Al 18 do. (KY)	72
al-5(al-K-5)	albino-5	II	11	Al 48 do. (KY)	72
al-6(t)(al-K-6)	albino-6	VI+IX	2	Al 50 do. (KY)	72,82
al-7(t)(al-K-7)	albino-7	II	11	Al 69 Taichung 65 mutant(KY)	72
al-8(al-K-8)	albino-8	III	3	Al 63 do. (KY)	72,80
al-9(t)(al-K-9)	albino-9	I	6	Al 168 Kinmaze mutant(KY)	
al-10(al-K-10)	albino-10	XI	5	Al 450 do. (KY)	81
chl-1(ch-1)	chlorina-1	XI	5	HO 718 Kishinriki(KY)	67,78,204
chl-2(ch-2)	chlorina-2	XI	5	LT 4 Nōrin 8 mutant(KY)	78,204
chl-3(ch-3)	chlorina-3	XI	5	HO 717 Ō-to(KY)	78,204
chl-4(ch-4)	chlorina-4	I	6	M 77 Nōrin 8 mutant(KY)	204
chl-5(ch-5)	chlorina-5	III	3	CM 62 Kinmaze mutant(KY)	80
chl-6(ch-6)	chlorina-6	III	3	CM 259 do. (KY)	80
chl-7(t)	chlorina-7	I	6	HM-1(NA)	202
fs-1(fs)	fine stripe-1	I	6	N-1 Akageshima(HK)	116,172
fs-2	fine stripe-2	III	3	M-31(HK)	80,287
bgl	bright green leaf	VI+IX	2	CM 2052 Kinmaze mutant (KY)	82
fgl(fl)	faded green leaf		7	HO 800 Hōki-asahihen(KY)	68,259,330
pgl	pale green leaf		7	HO 775 Okayamakibiho-2gō (KY)	68,259,330
rfs	rolled fine striped leaf	IV	10	M 85 Nōrin 8 mutant(KY)	82
st-1(ws-1)	stripe-1	I	6	HO 594-600 Shima-ine(KY), H-450(HK)	164,286
st-2(gw)	stripe-2	VI+IX	2	N-11 Hokkoshima(HK)	172

st-3(stl)	stripe-3	XI	5	CM 139 Kinmaze mutant (KY)	81
st-4(ws-2)	stripe-4	II	11	M-533 (HK)	150
v-1	virescent-1	I	6	L-8 virescent (HK)	84,172
v-1(t)(v-1)	virescent-1	XI	5	Jodon's tester (KY)	71,204
v-2	virescent-2	XI	5	HO 799 Yaehohen (KY)	78,204
v-3	virescent-3	I	6	CM 25 Kinmaze mutant (KY)	204
v-4	virescent-4	VIII	9	LT 3 Nōrin 8 mutant (KY)	78,204
v-5	virescent-5	XI	5	CM 23 Kinmaze mutant (KY)	204
v-6	virescent-6	III	3	CM 220 do. (KY)	74,80
v-7	virescent-7	XI	5	CM 262 do. (KY)	81
v-8	virescent-8		12	CM 24 do. (KY)	83
v-9(t)	virescent-9	VIII	9	CM 202 do. (KY)	242
v-10(t)	virescent-10	VI+IX	2	CM 285 do. (KY)	242
v-11(t)	virescent-11	VI	10	CM 577 do. (KY)	242
ylb	yellow banded leaf blade	VI+IX	2		51
ylm	yellow leaf margin	II	11	M 88 Nōrin 8 mutant (KY)	71
z-1	zebra-1	VIII	9	HO 613,612 Iyogasuri 1gō (KY)	68,78
z-2	zebra-2	VIII	9	M 36 Nōrin 8 mutant (KY)	71,78
z-3	zebra-3	XI	5	CM 2064 Kinmaze mutant (KY)	81
z-4	zebra-4		12	CM 306 do. (KY)	83
z-5	zebra-5	II	11	M-51 Dohoku 21gō mutant (HK)	113

3. Dwarfness

d-1	daikoku dwarf (small round grain)	VI+IX	2	A-23 Daikoku (HK), HO 532 Daikoku (KY)	2,66,166,172
d-2	ebisu dwarf	II	11	A-26 Ebisu (HK)	2,67,166,172
d-3	bunketsu-waitō (tillering dwarf, triplicate genes)	II	11	A-12 Bunketsu-waitō (HK)	166,172
d-4		I	6	do.	
d-5		X	8	do.	
d-6(d-34)	ebisumochi or tankanshirasasa dwarf (short second internode)	IV	10	A-25 Ebisumochi (HK), HO 540 Tankan-shirasasa (KY)	67,169,172
d-7	heiei-daikoku dwarf (cleistogamous)	IV	10	N-7 Heiei-daikoku (HK)	171,172
d-9	chinese dwarf	I	6	N-60 Chugokutō waisei (HK)	176
d-10(d-15,d-16)	kikeibanshinriki or toyohikari bunwai (tillering dwarf)	III	3	N-70 Toyohikari bunwai (HK) HO 548 Kikeibanshinriki (KY)	66,77,110,284

d-11(d-8)	shinkane-aikoku or nōrin-28 dwarf (small round grain)	II	11	N-58 Nōrin-28 wai(HK), HO 556 Shikane x Aikoku (KY)	67,110,172
d-12	yūkara dwarf (semidwarf)			N-26 Yūkara waisei(HK)	287
d-13	short grained dwarf			M-15 Nōrin 8 mutant(HK)	287
d-14(d-10)	kamikawa-bunwai (tillering dwarf)	XI	5	N-57 Kamikawa-bunwai	284
d-17(t)	slender dwarf			I-17 Slender dwarf(HK)	284
d-18^{h}	hōsetsu-waisei (extreme dwarf)			N-71 Hōsetsu waisei(HK)	117,255,256
d-18^{k}(d-25)	kotaketamanishiki (semidwarf)	III	3	HO 563 Kotake-tamanishiki (KY)	80,256,330
d-19(t)	Kamikawa dwarf (dense panicle)			N-56 Kamikawa waisei(HK)	117
d-20	hayayuki dwarf (sinuous rachis)	XII		M-48 Hayayuki waisei(HK)	117
d-21	aomorimochi-14 dwarf (narrow leaf)	I	6	J-14 Aomorimochi-14 waisei (HK)	117
d-22(t)	jōkei 6549 dwarf (semidwarf)			N-61 jōkei 6549 waisei(HK)	284
d-23(t)	ah-7 dwarf (slender culm)			AH-7(HK)	284
d-24(t)	m-7 dwarf (slender and sinuous culm)			M-7 Nōrin 8 mutant(HK)	110,284
d-26(t)	7237 dwarf	III	3	7237 (Jodon's marker)	50,53
d-27(d-t)	bunketsu-tō (tillering dwarf)	VIII	9	HO 568 Bunketsutō(KY)	71,78
d-28(d-C)	chōkeidaikoku (tall daikoku type)	VIII	9	HO 534 Chōkeidaikoku(KY)	78
d-29(d-K-1)	short uppermost internode dwarf	X	8	M 92 Nōrin 8 mutant(KY)	71
d-30(d-W)	waisei-shirasasa (twisted flag leaf)	X	8	HO 539 Waisei-shirasasa (KY)	67,71
d-31	taichung-155 irradiated dwarf	II	11	D-155-8	324
d-32(d-K-4,d-12)	dwarf Kyushu-4 (spreading tillers)	X	8	M 9, M 49 Nōrin 8 mutant (KY)	65
d-33(d-B)	bonsaitō dwarf (rolled leaf)		4	HO 565 Bonsaitō(KY)	68,330
d-35(t)	tanginbōzu dwarf (gibberellin responsive)			N-77 tanginbōzu(HK)	110,255,270
d-42(t)	liguleless dwarf (narrow leaf)	II	11	M-341 Nōrin 8 mutant(HK), H106	55,110

d-49(t)	reimei dwarf (high yield, lodging resistance)			Reimei (Fukei 70)(NG)	37
d-50(t)	fukei 71 dwarf (strong culm)			Fukei 71(NG)	37
d-51(d-K-8)	dwarf Kyushu-8		12	CM 1305 Kinmaze mutant(KY)	83
d-52(d-K-2)	dwarf Kyushu-2	XI	5	CM 45 do. (KY)	78,79
D-53(D-K-3)	Dwarf Kyushu-3	VIII	9	LT 15 Nōrin 8 mutant(KY)	78,79
d-54(d-K-5)	dwarf Kyushu-5	III	3	CM 719 Kinmaze mutant(KY)	80
d-55(d-K-6)	dwarf Kyushu-6	III	3	CM 296 do. (KY)	80
d-56(d-K-7)	dwarf Kyushu-7	XI	5	CM 298 do. (KY)	81
d-57[d(x)]	dwarf	VII	1		53,323
sd-1(d-47)	dee-geo-woo-gen dwarf	III	3	Taichung Native-1(HK), SC 2,3,4,5(NA)	5,147,271
sd-2	semidwarf-2			D 66	33,34
sd-3	semidwarf-3			CI 9858	33
sd-4	semidwarf-4			D 23, D 24, D 25	147

4. Spikelet or grain

alk	alkali degeneration (treated with 1.7% KOH)	I	6	most of *japonica*	140
An-1	Awn-1-3 (triplicate genes)	II	11	A-1 Akage(HK)	168,172,229, 280
An-2		VI+IX	2		
An-3		XI	5		
An-4(t)	Awn-4		12	T11-12(RY)	235
bd-1	beaked lemma (duplicate genes)	(XII)		AG507	154,230
bd-2					
bk	big kernel			Tairyuto (Tochigiwase mutant)	99,296
clw	claw shaped spikelet			M-8 Nōrin 8 mutant(HK)	287
da	double awns				166,275
dp-1	depressed palea-1 (underdeveloped palea)	I	6	HO 675 Hen-eitō(KY)	67,158,164
dp-2	depressed palea-2	VII	1	HO 672 Mikagetsutō(KY)	66,158
du	dull endosperm		7	EM 12,15,47 Kinmaze mutant(KY), 2035(NA)	203,240,321
eg	extra glume	III	3	HO 683 Henchoeitō(KY)	66,77,80
Fgr	Fragrant flower			C.I. 3794	85,87,98

g-1(g)	long sterile lemmas-1	IV	10	A-18 Chōgoeitō(HK), HO 680-682 Chōeitō(KY)	67,94,172,206
Su-g-1	Suppressor for *g-1*			E-41 Pappaku(HK)	172,173
G-2(Gl,Gm)	Long sterile lemmas-2 (incomplete dominance)			Early Prolific long glume	88
ge	giant embryo	IV	10	EM 40 Kinmaze mutant(KY)	240,321
Hg	Hairy glume	XII		E-45 Betong(HK)	172,173
lgt	long twisted grain	III	3	7237 (Jodon's marker)	50
lk	slender grain				89
Lk-f	'Fusayoshi' long grain	XI	5	Fusayoshi(OK,HK)	292,294,295
lmx	long lemma				87,88
lp-1 *lp-2*	long palea (duplicate genes)	IV	10	M 21	297 ,214
me	mutiple embryos				92,138,213
mp	multiple pistils	(IV)			154,210
Mi	Minute grain	XI	5	L-35 Minute, H-343(HK)	292,293
mls-1 *mls-2*	malformed lemma (duplicate genes)			H-166(HK)	287
Ph(Po)	Phenol staining (dark-violet grain with phenol solution)	II	11	A-58 Kokushokutō-2(HK) HO 755 Ōsugi(KY)	144,160,172 193,215
rk-1	round kernel-1	II	11	HO 637 Henpeitō(KY)	68,71
rk-2	round kernel-2		7	M 142 Taichung 65, mutant(KY)	69,330
Sdr-a(Sd) *Sdr-b*	Seed dormancy (complementary genes)			Surjamukhi(TH)	291
Sg	Permeability of testa to water			Ōu 195 gō(TH)	291
sh	shattering	VIII	9	H-21(HK)	87,172 ,214
shr-1^{s} *shr-1*^{a}	shrunken endosperm-1 (multiple alleles)	III	3	EM-20 Kinmaze mutant(KY) EM-6,27 do. (KY)	240,320,321, 322
shr-2	shrunken endosperm-2			Em-22,34,36 Kinmaze mutant(KY)	320,321,322
Sk	Scented kernel	(III,V)		Basmati-370, Kalabhat	87,218,301
su	sugary endosperm		12	EM-5 Kinmaze muatnt(KY)	60,240,322,
tri	triangular hull	X	8	HO 668 Sankakutō(KY)	67,166

Un-a *Un-b*	Uneven grain (complementary genes)	I IV	6 10	M.21	297
wx(am)	glutinous endosperm	I	6	A-43 Hokkaimochi-1gō(HK) HO 965 Hakugyokumochi(KY)	67,164,172, 316
5. Panicle					
Bp	Burlush-like panicle	VII	1	HO 551 Mansakuhen(KY)	68
Cl	Clusterd spikelets	I	6	L-16 Clustered(HK)	67,88,172
Scl	Superclustered				9,245
Dn-1(Dn)	Dense panicle-1	VII	1	M-53 Fūrenbōzu-mitsuryu (HK), HO 576,577 Koyabōzu (KY)	66,172
Dn-2	Dense panicle-2				95
dn-3	dense panicle-3			Akibare-missui(NG)	39
lax(lx)	lax panicle (very sparse setting of spikelets)	III	3	HO 616-618 Sodairyu, (KY), H-482(HK)	66,80,330
lhd	leafy head (absence of panicle)			Tayōtō,Ryushu X_3	1,56
nbs	non-bearing of spikelets			Akita 1gō mutant	186,307
nl-1(nl)	neck leaf-1	VI+IX	2	H-69(HK),HO 708,709,716 Hokamuri(KY)	66,88,172
nl-2	neck leaf-2	VI+IX	2	M 45 Nōrin 8 mutant(KY)	69,71,82
Pd	Pendant panicle	(XII)		W.137	154,216
ri	verticillate rachis (whorl arrangement of rachises)	VI+IX	2	H-68 Rinshimomigare(HK), HO 691 Rinshitō (KY)	66,82,172
Shp(Ex)	Sheathed panicle			T-131	95,247
sn-1 *sn-2*	sinuous neck (duplicate genes)			Niro Vialone	93
sp	short panicle	VIII	9	HO 547 Shinrikihen 8gō (KY), H-484(HK)	67,68,78
spr-1	spreading panicle-1				52
Spr-2-a(E) *Spr-2-b*	Spreading panicle-2 (complementary genes)			H-128(HK), Wild rice	156,166
Ur-1(Ur)	Undulate rachis-1	I	6	A-32 Fūrenbozu(HK)	172
ur-2	undulate rachis-2		12	Kinmaze mutant(KY)	83

6. Leaf and culm

bc-1	brittle culm-1	XI	5	H-9(HK), HO 702 Kamairazu(KY)	67,94,172
bc-2	brittle culm-2			M-5 Nōrin 8 muatnt(HK)	287
bc-3	brittle culm-3	X	8	M 11 do. (KY)	71
bl-1	brown leaf spot-1	X	8	A-73 Momigaredatsu(HK), HO 697 Momigarebyō(KY)	67,71,172
bl-2(bl-m)	brown leaf spot-2	I	6	L-7 black leaf Magnolia (HK)	88,177
bl-3	brown leaf spot-3	I	6	H-131(HK)	287
bl-4	brown leaf spot-4	XI	5	M-25 Nōrin 8 mutant(HK)	287
bl-5	brown leaf spot-5			M-21 do. (HK)	287
bl-6	brown leaf spot-6			M-26 do. (HK)	287
spl-1(bl-7,bl-12)	spotted leaf-1		4	HO 678 Banshinrikibyō-gata(KY)	68,77,330
spl-2(bl-13)	spotted leaf-2	X	8	HO 698 Katsumonbyō(KY)	77
spl-3(bl-14)	spotted leaf-3	XI	5	M 41 Nōrin 8 mutant(KY)	71,77
spl-4(bl-15)	spotted leaf-4	I	6	M 114 do. (KY)	71,77
spl-5(bl-16)	spotted leaf-5	IV	10	M 87 do. (KY)	71,82
spl-6	spotted leaf-6	III	3	CM 20 Kinmaze mutant(KY)	77,330
spl-7	spotted leaf-7	VI+IX	2	M 64 Nōrin 8 mutant(KY)	77,82
spl-8(bl-8)	spotted leaf-8	VI+IX	2	CM 207 Kinmaze mutant(KY)	77,82
spl-9	spotted leaf-9			LT 26 Nōrin 8 mutant(KY)	77
sl	'sekiguchi' lesion (yellowish brown leaf spot by chemicals and pathogens)	VII	1	Sekiguchi Asahi(NA)	128
zn	zebra necrosis	I	6	M-52 A-5 mutant(HK)	113
ysl	yellow leaf spot			M-30 Nōrin 8 mutant(HK)	287
dl(lop)	drooping leaf	XI	5	HO 788 Tareba(KY)	67,78,158
drp-1	dripping-wet leaf-1	II	11	D-47 Nureba(HK)	177
drp-2	dripping-wet leaf-2	VII	1	HO 700 Nurebatō(KY)	66
drp-3	dripping-wet leaf-3	XI	5	CM 1337 Kinmaze mutant (KY)	81
drp-4	dripping-wet leaf-4	XI	5	CM 198 do. (KY)	81
drp-5(t)	dripping-wet leaf-5	II	11	CM 1158 do. (KY)	241
drp-6(t)	dripping-wet leaf-6	III or I	3 or 6	CM 1776 do. (KY)	241
drp-7(t)	dripping-wet leaf-7	VIII	9	CM 2039 do. (KY)	241
dw-1(fh) *dw-2*	deep water tolerance (floating habit, duplicate genes)	(X)		Khama, Sai Bua	87,154,212

er(o)	erect growth habit	VI+IX	2	H-75(HK)	156,287
eui	elongated uppermost internode	VI+IX	2	N-171(HK),76:4512	149,219
fc-1	fine culm-1	XI	5	M 56 Norin 8 mutant(KY)	71,78
fc-2(t)	fine culm-2	I or III	6 or 3	CM 256 Kinmaze mutant(KY)	241
gl-1 *gl-2*	glabrous leaf and hull (duplicate genes	VI+IX	2	E-43 Garumbalay,H-96(HK)	70,172,193, 239
Hl-a *Hl-b*	Hairy leaf (complementary genes)	I	6	E-44 Pirurutong,H-120(HK)	172,173
la	'lazy' growth habit (prostrate habit)	VIII	9	A-75 Motsureto(HK), HO 571 Motsure(KY)	14,67,78, 96,172
lg	liguleless	II	11	A-78 Muyozetsu(HK), HO 706 Murasaki-muyozetsuto(KY)	67,94,172
Lh-a *Lh-b*	Heavy pubescence (complementary genes)				19
nal-1	narrow leaf-1 (duplicate or triplicate genes)	II	11	F1-63(KY),H-579(HK)	158
nal-2		VIII	9		
nal-3(nal-2)			4	HO 651 Murasaki-rokusuke-gawari(KY)	68,330
nal-4(nal)	narrow leaf-4	II	11	D-155-8	324
nal-5(nal-1)	narrow leaf-5	II	11	M 88 Norin 8 mutant(KY)	71
rcn	reduced culm number (one or two tillers)	I	6	N-133(HK)	112
rl-1	rolled leaf-1	III	3	L-29 Rolled leaf(HK)	95,177
rl-2	rolled leaf-2	II	11	F1-160(KY)	158
rl-3(rl-1)	rolled leaf-3		4	C54R9783(KY)	68,330
rl-4(rl-2)	rolled leaf-4	III	3	M 50 Norin 8 mutant(KY)	71,80
rl-5(rl-3)	rolled leaf-5	XI	5	CM 1993 Kinmaze mutant (KY)	81
ts-a *ts-b*	twisted stem (complementary genes)	III	3	7237 (Jodon's marker)	50

7. Heading date

E-1	Late heading-1			EG 1,4,6,7(KT)	106,201,273 274

E-2	Late heading-2			EG 2,4,5,7(KT)	106,274
E-3	Late heading-3			EG 3,5,6,7(KT)	106,274
Ef-1^{a}	Early flowering (compound locus consisting of five subloci)	VIII	9	T65E^{a}(A3)(CH)	302,303,304
Ef-1^{b}				T65E^{b}(B96)(CH)	
Ef-1$^{\gamma}$				T65E$^{\gamma}$(1123)(CH)	
Ef-1^{X}				T65E^{X}(1190)(CH)	
Ef-1^{+} (= *ef-1*)				T65e (Taichung 65)(CH)	
m^{a}-*Ef-1*$^{+}$(*m*^{a})	Emphatic effect when combined with *Ef-1*$^{+}$ (multiple alleles)	IV	10	T65e-*em*^{a}(A4-52)(CH)	304,305,306
m^{b}-*Ef-1*$^{+}$(*m*^{b})				T65e-*em*^{b}(B172)(CH)	
m-Ef-1^{a}	do. with *Ef-1*^{a}			T65E^{a}-*em*($A4_7$)(CH)	
m-Ef-1^{b}	do. with *Ef-1*^{b}			T65E^{b}-*em*(820)(CH)	
m-Ef-1$^{\gamma}$	do. with *Ef-1*$^{\gamma}$			T65E$^{\gamma}$-*em*(CH)	
m-Ef-1^{X}	do. with *Ef-1*^{X}			T65E^{X}-*em*(CH)	
Ef-2	Early flowering		7	T65Ef_2(RY)	234
Se-1^{e}*(Lm,Lf,Rs)*	Photosensitivity (earliness vs. lateness, multiple alleles)	I	6	ER(NA)	11, 84,140
Se-1^{n}				Nōrin 8 (NA)	328
Se-1^{t}					
Se-1^{s}				Shiranui(NA)	
Se-1^{u}				LR(NA)	
se-2	Photosensitivity	IV	10	P-150(Jodon No.7014)	332

8. Sterility

as	asynapsis				102,103,189
cps	compact panicle sterile			N-22 Mitsuryu-funen, H-66(HK)	166,281
ds	desynapsis			Kinmaze mutant(KY), IR 36 mutant(IR)	12,13,118, 267
fes-1	female sterility-1			Fujisaka 5 x Tjina	325
Fes-2	Female sterility-2			G2250	217
lhs-1(op)	leafy hull sterile-1	XI	5	CM 10(KY)	64,165
lhs-2(lhs)	leafy hull sterile-2	XII		N-76 Sorachi mutant(HK)	114
L-1-a(L_1)	Lethal-1 (complementary genes)			414 P.T.B.10(GI)	194
L-1-b(L_2)				419 P.T.B. 7(GI)	
L-2-a(L-r_1)	Lethal-2 (complementary genes)			Nōrin 8 (NA)	3
L-2-b.(L-r_2)				Jamaika(NA)	

ms-1(sf)	male sterile-1	I	6	Fukukame mutant	46
ms-2(ms-d)	partial male sterility (various interactions among *ms-2,3,4,5* and *6*, *ms-2* shows dwarfness and *ms-5* is responsible for cool tolerance)			md-strain	253,254
ms-3(ms-1)				Bufumochi-8	
ms-4(ms-2)				Fujinori	
ms-5(ms-3)				Ōtori	
ms-6(ms-4)				Bufumochi-8	
ms-7(t)	male sterile-7			K 1(KT)	136
ms-8(t)	male sterile-8			K 2, S 26(KT)	135,136
ms-9(t)	male sterile-9			E 1(KT)	136
ms-10(t)	male sterile-10			T 1(KT)	136
ms-11(t)	male sterile-11			T 2(KT)	136
ms-12(t)	male sterile-12			T 3(KT)	136
ms-13(t)	male sterile-13			S 32(KT)	135
ms-14(t)	male sterile-14			S 40(KT)	135
ms-15(t)	male sterile-15			S 55(KT)	135
ms-16(t)	male sterile-16			S 59(KT)	135
ms-17(t)	male sterile-17			S 81(KT)	135
ops(ops-1)	open hull sterile	VI+IX	2	HO 643 Funentō(KY)	66,82
pcs(ops-2)	parthenocarpy sterile			C-24 Kaieifunen(HK)	166,167
s-a-1(s_1,x_1)	hybrid sterility-a (gametophytic F_1-sterility, duplicate genes)	I	6	219 Garumbalay(GI)	192,196
s-a-2(s_2,x_2)				563 Kinoshita-mochi(GI)	
s-b-1(y_1)	hybrid sterility-b (duplicate genes)	I	6	219 Garumbalay(GI)	192,196
s-b-2(y_2)				563 Kinoshita-mochi(GI)	
s-c-1	hybrid sterility-c (duplicate genes)	I	6	414 P.T.B. 10(GI)	195
s-c-2		II	11	325 Kaniranga(GI)	
s-d-1	hybrid sterility-d (duplicate genes)	I	6	E1=T65 (Taichung 65) (GI)	196
s-d-2				E3=T65 Isogenic of T65 B_{13}(GI)	
s-e-1	Hybrid sterility-e (duplicate genes)	XI	5	E1=T65 (GI)	196
s-e-2		II	11	E2=T65A isogenic of T65 B_{13}(GI)	
S^a*-1*	F_1 sterility in heterozygote (*S^a-1/S-1*) (one locus sporo-gametophytic lethal)	I	6	E101=108 *O.sativa*(GI)	228
S-1				E103=(S)isogenic of 108 B_8(GI)	

S^a-2 S-2	F_1 sterility in heterozygote (S^a-2/S-2)			E102=W025 *O.glaberrima* (GI) E104=(G)isogenic of W025 B_8(GI)	228
S^a-3 S-3	F_1 sterility in heterozygote (S^a-3/S-3)	VIII	9	Taichung 65(GI) W025 *O.glaberrima*(GI)	225
S-A-1(A-1) S-A-2(A-2)	Hybrid sterility-A (duplicate seed fertility genes)	I	6	104 Peh-ku-tsao-tu, 563 Kinoshita-mochi, 537(GI)	199
S-B-1(B-1) S-B-2(B-2)	Hybrid sterility-B (duplicate pollen fertility genes)	I	6	S37,104,563(GI)	198,199
ssk(sk)	malformed semisterile	II	11	Kokurato mutant	46
w'-a(a_1) w'-b(a_2)	F_2 weak segregants, in the complementary action of w'-a and w'-b			451 Surjamukhi(GI) 521 Kisshin(GI)	194
D-a(D-1) D-b(D-2)	Complementary dominant lethal (F_1 lethal)			Af107,Af113(*barthii*)(GI) 563,T65 (*sativa*) (GI)	17
W-a(W-1) W-b(W-2)	Complementary dominant weakness (F_1 weakness)			W042(GI) W025(GI)	18

9. Gametophyte genes (Low fertilization capacity of male gametes)

ga-1	gametophyte gene-1	I	6	Atomic bombed rice(KY)	75
ga-2	gametophyte gene-2	XI	5	Most of *japonica*(NA)	179,184
ga-3	gametophyte gene-3	XI	5	Nan jing hsien dao(NA)	179,184
ga-4(ga-A)	gametophyte gene-4	I	6	Most of *japonica*(KY,NA, HK)	159,185
ga-5(ga-B)	gametophyte gene-5	I	6	do. (HK)	159
ga-6	gametophyte gene-6	II	11	M-533 Nōrin 8 mutant(HK)	150
ga-7	gametophyte gene-7	III	3	H-50(HK)	152
ga-8	gametophyte gene-8	III	3	*indica* cultivars(NA)	182
ga-9	gametophyte gene-9	III	3	H-50(HK)	152
ga-10(t)	gametophyte gene-10	II	11	M-51 Dohoku 21 mutant (HK)	113

10. Cytoplasmic male sterility

Cytoplasm					
[ms-bo]	'Chinsurah boro II' cytoplasm			Chinsurah boro II(RY)	257,258,259

[ms-ld]	'Lead rice' cytoplasm			Lead rice (NA)	311,313
[ms-TA]	'TA820' cytoplasm			TA820 (NA)	119,120,121
[ms-CW]	Chinese wild rice cytoplasm			WI (TH)	104
[ms-WA]	WA-group cytoplasm			MS wild rice	15
[ms-HL]	HL-group cytoplasm			Wild red-awned rice x Lien-Tong-Tsao	15
[ms-jp]	'Akebono' cytoplasm			Akebono (OF)	315
Fertility restorer					
Rf-1	Pollen fertility restoration-1 (gametophytic)		7	Chinsurah boro II (RY)	115,257,259
Rf-2 (Rf-x)	do. -2			Fukuyama (NA)	260,311
Rf-a	Pollen fertility restoration (gametophytic, complementary action of *Rf-a* or *Rf-b* and *Rf-c*)			H-406 (HK)	115,148
Rf-b				do.	
Rf-c				do.	
Rf-a'	Pollen fertility restoration (complementary action of *Rf-a'* and *Rf-b'* or *Rf-C'* and *Rf-d'*			H-103 (HK)	115,148
Rf-b'				do.	
Rf-c'				do.	
Rf-d'				do.	
Rf-j	Pollen fertility restoration (sporophytic, derived from 'Akebono')			Akebono (OF)	315
11. Fungal and bacterial diseases resistance					
Ce	Narrow leaf spot resistance			Blue Rose 41, C.I.3794	85,91,220,221
He	*Helminthosporium* leaf spot resistance			*he:* Kattorube	163
Pi-a	Blast resistance-a	VIII	9	Aichi-asashi, Nōrin 41 (NA)	45,261,290, 319
Pi-b (Pi-s)	Blast resistance-b	X	8	BL-1 (NA)	129,131,261
Pi-f	Blast resistance-f (field resistance)	VIII	9	Chūgoku 31, ST No.1 (CA)	261,333
Pi-i	Blast resistance-i	I	6	Fujisaka 5 (NA)	36,45,319
Pi-k	Blast resistance-k (multiple alleles)	VIII	9	Kusabue (NA)	45,124,261, 319
$Pi\text{-}k^s$				Shin 2, Nōrin 6-k^s (NA)	125
$Pi\text{-}k^p$				K 60 (NA)	126,134
$Pi\text{-}k^m$ *(Pi-m)*				Tsuyuake (NA)	132

Pi-k^h				K 3(NA)	133
Pi-t	Blast resistance-t			K 59(NA)	129,131
Pi-ta	Blast resistance-ta (multiple alleles)	VII	1	K 1(NA)	122,127,261
Pi-ta^2				Pi No.4(NA)	123,127
Pi-ta^n				Nakei 212(CA)	261
Pi-z	Blast resistance-z (multiple alleles)	I	6	Fukunishiki(NA)	45,130,261
Pi-z^t				Toride 1(NA)	327
Pi-se-1(Rb-1)	Blast resistance-se (additive effects by three genes)	VIII	9	lazy-Sensho(YA)	40,42,43,44
Pi-se-2(Rb-2)				do. (YA)	
Pi-se-3(Rb-3)				do. (YA)	
Pi-is-1(Rb-4)	Blast resistance-is (cumulative effects by two genes)	VIII	9	Ishikarishiroke(YA)	40
Pi-is-2(Rb-5)					
M-Pi-z(Rb-6)	Modifier for *Pi-z*	VIII	9	Zenith(YA)	41
Pi(t)	Blast resistance	II	11		51,53
Sc-1	*Sclerotium* disease resistance (duplicate genes)			Boera Rope	47
Sc-2					
Xa-1	Bacterial blight resistance-1 (multiple alleles)	II	11	Kōgyoku(CA)	191,222,317
Xa-1^h				IR 28,29,30(NA)	
Xa-2	Bacterial blight resistance-2	II	11	Rantai Emas 2, Te tep (CA)	31,222
Xa-3(Xa-w)	Bacterial blight resistance-3			Wase-aikoku 3, Java 14(NA),Saikai PL1(KA)	31.191.268
Xa-4^a	Bacterial blight resistance-4 (multiple alleles)			IR 22(IR)	146,211,266
Xa-4^b				Semora Mangga(IR)	
xa-5	bacterial blight resistance-5	VI+IX	2	Aus 32, BJI, DZ92(IR)	146,211,266, 268,331
Xa-6	Bacterial blight resistance-6			DV 85, D 278(IR)	262,266,268
Xa-7	Bacterial blight resistance-7			DZ 78(IR)	266.268
xa-8	bacterial blight resistance-8			PI 231129(IR)	266,268
xa-9	bacterial blight resistance-9			Khao lay Nbay, Sateng (IR)	268

Xa-10	Bacterial blight resistance-10				
Xa-kg *Xa-kg*^{h}	Bacterial blight resistance-kg	II	11	Kōgyoku, Java 14(NA) IR28, 29,30(NA)	191,317
12. Virus and mycoplasma disease resistance					
Bsv(Bs)	Black streaked dwarf resistance			Te-tep(CA)	161,299
Gsv(Gs)	Grassy stunt resistance			*O. nivara*, IR 2061-464-6(IR)	107,272
Hbv(Rhb)	*Hoja blanca* resistance				8,298
Stv-a(St-1)	Stripe disease resistance (complementary genes, *Stv-b*^{i}; incompletely dominant)	I	6	Kuroboku, Zenith(CA)	300,308,309 310
Stv-b(St-2)				do. (CA)	
Stv-b^{i}*(St-2*^{i}*)*			12	Surjamukhi, Charnock Mineyutaka(CA)	
Tuv-a(Rtv) *Tuv-b*	Tungro resistance (complementary or duplicate genes)			Pankhari 203, Latisail	251,298
Ydv(Ryd)	Yellow dwarf resistance			Saitamamochi 10gō(NA)	162,298,299
13. Insect resistance					
Bph-1	Brown planthopper resistance-1	II	11	Saikai PL 3,4 (KA) IR1539-823 (IR)	7,58,59,263
bph-2	brown planthopper resistance-2	II	11	IR1154-243 (IR)	7,58,59,263
Bph-3	Brown planthopper resistance-3		7	IR17491-5-4-3-3-1 (IR)	58,145,263
bph-4	brown planthopper resistance-4		7	IR17488-3-3-2-2 (IR)	58,145,263
I-Bph-1	Inhibitor of *Bph-1*			TKM 6(IR)	153
Glh-1	Green leafhopper resistance-1			IR5491 (IR)	6,7
Glh-2	Green leafhopper resistance-2			IR5492 (IR)	7,269
Glh-3	Green leafhopper resistance-3		7	IR8 (IR)	7,264,269
glh-4	green leafhopper resistance-4			Ptb 8(IR)	269
Glh-5	Green leafhopper resistance-5			ASD 8(IR)	269
Glh-6	Green leafhopper resistance-6			IR36 (IR)	100

Glh-7	Green leafhopper resistance-7	Maddai Karuppan(IR)	100
gm-1(pd-a) *gm-2(pd-b)* *gm-3(pd-c)*	gall midge resistance V (triplicate or complementary genes)	W1263, Ptb 21 CR.94-MR.1624 4	231,252
I-Gm-1	Inhibitor for *Gm-1*	W1263, Ptb 21	252
Grh-1 *Grh-2*	Green rice leafhopper resistance (duplicate or complementary genes)	Te tep,Pebihun(NA) Saikai PL2(KA)	137,246
Sb	Stem borer resistance	TKM 6	6,27,139
Sm	Stem maggot resistance (incomplete dominance)	Nōrin 22 ,Ou 188(NA)	6,35,101
Wph-1(Wbph-1)	Whitebacked planthopper resistance-1	IR13475-7-3-2 (IR)	4,265
Wph-2(Wbph-2)	Whitebacked planthopper resistance-2	IR30659-1-59-6 (IR)	4
Wph-3(Wbph-3)	Whitebacked planthopper resistance-3	IR42646-8-90 (IR)	48
wph-4(wbph-4)	whitebacked planthopper resistance-4	IR42667-2-31 (IR)	48
Wph-5(Wbph-5)	Whitebacked planthopper resistance-5	N'Diang Marie(IR)	314
14. Isozymes			
$Acp\text{-}1^{-17}$ *(Acp-B)*	Acid phosphatase-1 AMC band-group((-17mm)	W1236 (GI)	29,30,207, 209,227,243
$Acp\text{-}1^{-9}$	do. (- 9mm)	W169 (GI)	
$Acp\text{-}1^{-4}$	do. (-4mm)	108 & W107 (GI,CH)	
$Acp\text{-}1^{4}$	do. (4mm)	W120, W149 (GI,CH)	
$Acp\text{-}1^{9}$	do. (9mm)	322(T65) & W 593(GI,CH)	
$Acp\text{-}1^{12}$	do. (12mm)	W036 (GI)	
$Acp\text{-}1^{24}$	do. (24mm)	W648 (GI)	
$Acp\text{-}1^{Nul}$	do. (null form)	1707(CH)	
$Acp\text{-}2^{Fa}$ *(Acp-C)*	Acid phosphatase-2 (Fa/Sa test moving) (fast band)	108, W107 GI,CH)	30,209
$Acp\text{-}2^{Sa}$	do. (slow band)	W120-12(GI,CH)	
$Acp\text{-}2^{Nul}$	do. (null form)	322(T65),563(GI,CH)	

Acp-3^{B}	Acid phosphatase-3 (B band-group) (presence)			W648, W1421 (GI,CH)	209
Acp-3^{Nul}	do. (null form)			108(GI,CH)	
Cat-1^{1} *(Cat-A)*	Catalase-1(slow band)			130, C5444 (GI)	227.243,244
Cat-1^{2}	do. (fast band)			T 65 & 221(GI)	
Est-1(Est-1^{S}, *Est-D)*	Esterase-1(slow band)			most of *japonica* and *indica*	180,181,243 249
Est-1^{Nul}	do. (absence)			some hill rice(NA)	
Est-2^{S} *(Est-E, Est-2*^{1}*)*	Esterase-2(slow band)	I	6	221 & W106 (GI), most of Indian cult. (NA)	180,181,183 227,243
Est-2^{F} *(Est-2*^{2}*)*	do. (fast band)			108 & 868(GI) most of Hsien(NA)	
Est-2^{Nul} *(Est-2*^{0}*)*	do. (null form)			T 65(GI) most of *japonica* (NA)	
Est-3^{S} *(Est-J)*	Esterase-3(slow band)			most of *japonica*(NA)	181,243
Est-3^{F}	do. (fast band)			most of *indica* (NA)	
Est-4^{S} *(Est-H)*	Esterase-4(slow band)				243
Est-4^{F}	do. (fast band)				
Est-4^{Nul}	do. (null form)				
Lap-1(Lap-E)	Leucine amino peptidase				243
Mdh-1(Mdh-A)	Malate dehydrogenase				243
Pgi-1(Pgi-A)	Phosphoglucose isomerase-1 (anodal band-group) (slow)			130(GI)	227,243,244
Pgi-1^{2}	do. (fast)			221(GI)	
Pgi-2(Pgi-B)	Phosphoglucose isomerase-2 (anodal band-group) (slow)	I	6	221(GI)	227,243,244
Pgi-2^{2}	do. (fast)			130(GI)	
Pox-1^{0C} *(Px,Pe)*	Peroxidase-1 (0C band)			W593, C8216 (GI,CH)	28,207,208 249
Pox-1^{2A}	do. (2A band)			322(T65) & 108(GI,CH)	
Pox-1^{4A}	do. (4A band)			W120 & W1294 (GI,CH)	
Pox-1^{Nul}	do. (absence)			4650(CH)	
Pox-2^{4C}	Peroxidase-2 (4C band)			108(GI,CH)	208,227, 249
Pox-2^{Nul}	do. (absence)			322(T65)(GI,CH)	

Pox-3^{3C}	Peroxidase-3 (3C band)	322(CH)	207
Pox-3^{5C}	do. (5C band)	4650(CH)	
R_{LB}^{4C}	Regulator gene acting in leaf blade	W120-5 (CH)	208
r_{LB}^{4C}	alternative allele of R_{LB}^{4C}	W120-4 (CH)	
R_{LS}^{4C}	Regulator gene acting in leaf sheath	W120-4 (CH)	208
r_{LS}^{4C}	alernative allele of R_{LS}^{4C}	W120-5 (CH)	
Rcp^{2A}	Receptor gene for peroxidase	T 65(GI)	28
Rcp^{4A}	Receptor gene for peroxidase	W648 (GI)	28
Reg-1^{2A}	Regulator gene for peroxidase	W648 (GI)	28
Reg-2^{4A}	Regulator gene for peroxidase	W648 (GI)	28
Reg-3^{2A}	Regulator gene for peroxidase		

*Parentheses mean the linkage group assigned by Misro (1981).

List of primary trisomics

Extra chromosome	Type	Name	Strain	Original cultivar
1	H	Large grain	T15, T16	Asakaze
			NT8314, NT8315	Nipponbare
			KT8301	Kinmaze
2	L	Short panicle	T23	Norin 8
			NT8316	Nipponbare
			KT8302	Kinmaze
3	O	Grassy	NT8321	Nipponbare
			KT8303	Kinmaze
4	A	Pale	T1	Aikoku
			T2	Asakaze
			NT831,NT832	Nipponbare
			KT8304	Kinmaze
5	M	Sterile	$P_1B_1$832, $P_1B_1$833	Nipponbare
			KT8305	Kinmaze
6	B	Awned	T3	Aikoku
			T4	Asakaze
			NT833, NT834	Nipponbare
			KT8306	Kinmaze
7	C	Small grain	T5, T6	Aikoku
			NT835, NT836	Nipponbare
			KT8307	Kinmaze
8	N	Smooth glume	$P_1B_1$831	Nipponbare
			KT8308	Kinmaze
9	G	Coarse	T13	Aikoku
			T14	Asakaze
	I	Late heading	T17, T18	Aikoku
	J	Spotted leaf	T19, T20	Aikoku
	K	Pseudo normal	T21, T22	Aikoku
	G	Pseudo normal	NT8312, NT8313	Nipponbare
	G	Pseudo normal	KT8309	Kinmaze
10	F	Rolled leaf	T11	Aikoku
			T12	Asakaze
			NT8311	Nipponbare
			KT8310	Kinmaze
11	E	Spreading	T9, T10	Aikoku
			NT8310	Nipponbare
			KT8311	Kinmaze
12	D	Erectoides	T7, T8	Aikoku
			NT837, NT838	Nipponbare
			KT8312	Kinmaze

Reference, Iwata et. al. (1984)

Institute: Laboratory of Plant Breeding,
Faculty of Agriculture, Kyushu University

List of translocation lines

Interchanged chromosome	Strain No.	Institution	Source	Reference
1 - 2	RT1	KY	Okute-Asashi. X-15	190
"	RT1-2, T65	RY	"	
1 - 3a	RT2	KY	Nōrin 8. 1-15.0	190
"	RT1-3a, T65	RY	"	
1 - 3b	RT3	KY	Nōrin 8. 1581	190
"	RT1-3b, T65	RY	"	
1 - 3c	RT1-3c, T65	RY	Atom. bomb. rice	62
1 - 4a	RT4	KY	Okute-Asahi. A2-3	190
1 - 4b	RT1-4b, T65	RY	A-5 Akamuro. 293	237
1 - 8	RT5	KY	Nōrin 8. 1288	190
"	RT1-8, T65	RY	"	
1 - 10	RT6	KY	Nōrin 8. 1533	190
"	RT1-10, T65	RY	"	
1 - 11	RT7	KY	Nōrin 8. 1272	190
"	RT1-11. T65	RY	"	
2 - 3a	RT55	KY	Atom. bomb. rice. AP80	62
"	RT2-3a. T65	RY	"	
2 - 3b	E22, RT61	KY	Taichung 65. Tr1	200,226 233,329
"	RT2-3b. T65	RY	"	
2 - 3c	E-23, RT65	GI, KY	Taichung 65. Tr8	200,226 233,329
"	RT2-3c. T65	RY	"	
2 - 3d	E-24, RT70,80	GI, KY	Taichung 65. Tr17, Tr34	200,226 233,329
"	RT2-3d, T65	RY	"	
2 - 5	RT40	KY	Atom. bomb. rice. AP25	62
"	RT2-5, T65	RY	"	
2 - 6	E-25, RT85	GI, KY	Taichung 65. Tr52	200,226, 233,329
"	RT2-6a, T65	RY	"	
2 - 7a	E-26, RT68	GI, KY	Taichung 65. Tr14	200,226, 233,329
"	RT2-7a, T65	RY	"	
2 - 7b	E-27	GI	Taichung 65. Tr16	200,226
2 - 10a	E-28, RT77	GI, KY	Taichung 65. Tr31	200,226, 233,329
"	RT2-10a. T65	RY	"	
2 - 10b	E-29	GI	Taichung 65. Tr32	200,226
2 - 10c	E-30	GI	Taichung 65. Tr38	200,226

3 - 4a	RT8	KY	Okute-Asahi. X-61	190
"	RT3-4a, T65	RY	"	
3 - 4b	RT9	KY	Nōrin 8. 4,15-0	190
"	RT3-4b, T65	RY	"	
3 - 5a	RT40	KY	Atom. bomb. rice. AP21	62
"	RT3-5a, T65	RY	"	
3 - 5b	RT87	KY	Taichung 65. Tr54	200,226 233,329
"	RT3-5b, T65	RY	"	
3 - 5c	E-31	GI	Taichung 65. Tr55	200,226
3 - 6	RT10	KY	Okute-Asahi. X-120	190
"	RT3-6, T65	RY	"	
3 - 7	RT3-7, T65	RY	A-58 Kokushokutō-2. 208	237
3 - 8a	RT11	KY	Okute-Asahi. A_2-2	190
"	RT3-8a, T65	RY	"	
3 - 8b	RT12	KY	Okute-Asahi. A_2-4	190
"	RT3-8b, T65	RY	"	
3 - 8c	RT3-8c, T65	RY	Taichung 65. Tr16	200,226
3 - 8d	E-32, RT76	GI, KY	Taichung 65. Tr30	200,226,329
3 - 8e	E-33	GI	Taichung 65. Tr45	200,226
3 - 11a	RT13	KY	Okute-Asahi. X_2-4	190
"	RT3-11a, T65	RY	"	
3 - 11b	RT14	KY	Nōrin 8. 44	190
"	RT3-11b, T65	RY	"	
3 - 11c	RT15	KY	Nōrin 8. 1267	190
"	RT3-11c, T65	RY	"	
3 - 11d	ET34, RT82	GI, KY	Taichung 65. Tr39	200,226 233,329
"	RT3-11d, T65	RY	"	
3 - 12a	RT16	KY	Nōrin 8. 1509	190
"	RT3-12a, T65	RY	"	
3 - 12b	RT3-12b, T65	RY	Atom. bomb. rice	62
3 - 12c	E35, RT72	GI, KY	Taichung 65. Tr20	200,226 233,329
"	RT3-12c, T65	RY	"	
4 - 5a	RT17	KY	Nōrin 8. 1403	190
"	RT4-5a, T65	RY	"	
4 - 5b	RT4-5b, T65	RY	A-5 Akamuro 80	237
4 - 5c	RT4-5c, T65	RY	A-58 Kokushokutō-2. 279	237
4 - 12	RT32	KY	Atom. bomb. rice. AP5	62
5 - 6	RT18	KY	Okute-Asahi. X-120	190
"	RT5-6, T65	RY	"	

5 - 9	RT19	KY	Okute-Asahi. X-69	190
"	RT5-9, T65	RY	"	
5 - 10a	RT5-10a, T65	RY	A-58 Kokushokutō-2. 204	237
5 - 10b	E36, RT83	GI, KY	Taichung 65. Tr44	200,226 233,329
"	RT5-10b. T65	RY	"	
6 - 7	RT49	KY	Atom bomb. rice. AP39	62
"	RT6-7. T65	RY	"	
6 - 8	RT38	KY	Atom. bomb. rice. AP15	62
"	RT6-8. T65	RY	"	
6 - 10a	RT20	KY	Nōrin 8. 1470	190
"	RT6-10a, T65	RY	"	
6 - 10b	E37, RT79	GI, KY	Taichung 65, Tr33	200,226,329
6 - 10c	E38	GI	Taichung 65, Tr57	200.226
"	RT6-10b,T65	RY	"	
6 - 11	RT21	KY	Okute-Asahi. X-204	190
"	RT6-11, T65	RY	"	
6 - 12	E39, RT75	GI, KY	Taichung 65. Tr28	200,226 233,329
"	RT6-12a, T65	RY	"	
7 - 8a	RT22	KY	Okute Asahi. AC13	190
"	RT7-8a, T65	RY	"	
7 - 8b	RT23	KY	Okute Asahi. X-205	190
"	RT7-8b, T65	RY	"	
7 - 9	RT24	KY	Okute Asahi. A_1-7	190
"	RT7-9, T65	RY	"	
7 - 10	RT46	KY	Atom. bomb. rice. AP32	62
7 - 11	RT7-11, T65	RY	Atom. bomb. rice	62
7 - 12	E40, RT63	GI, KY	Taichung 65. Tr4	200,226 233,329
"	RT7-12, T65	RY	"	
8 - 10a	RT25	KY	Nōrin 8. 1244	190
"	RT8-10a, T65	RY	"	
8 - 10b	RT36	KY	Atom. bomb. rice. AP9	62
8 - 11	E41, RT66	GI, KY	Taichung 65. Tr10	200,226 233,329
"	RT8-11a, T65	RY	"	
8 - 12a	RT8-12a, T65	RY	Okute-Asahi. X-84	190
8 - 12b	RT27	KY	Nōrin 8. 13	190
"	RT8-12b, T65	RY	"	
9 - 10a	RT28	KY	Tōsan 19. B-7-1	190
"	RT9-10a, T65	RY	"	
9 - 10b	RT9-10b, T65	RY	A-5 Akamuro. 178	237

10 - 11	RT29	KY	Okute Asahi. X-141	190
"	RT10-11, T65	RY	"	
10 - 12	RT31	KY	Atom. bomb. rice. API	62
"	RT10-12, T65	RY	"	
11 - 12	RT11-12, T65	RY	A-58 Kokushokutō-2. 197	237

List of isogenic lines

1. Isogenic lines of 'Shiokari' for dwarf genes

Gene symbol	Name of dwarf	linkage groups	Chromo-some	Strain and Backcrosses	Dwarf donor
d-1	daikoku dwarf	VI+IX	2	ID-1,B_{11}	H-86 (HK)
d-2	ebisu dwarf	II	11	ID-2,B_9	H-85 (HK)
d-3,4,5	bunketsu-waito dwarf	II,I,X	11,6,8	ID-3,B_5	H-2 (HK)
d-6	ebisumochi dwarf	IV	10	ID-6,B_7	H-127(HK)
d-7	heiei-daikoku dwarf	IV	10	ID-7,B_9	N-7 (HK)
d-10	toyohikari-bunwai dwarf	III	3	ID-10,B_9	N-70 (HK)
d-11	norin-28 dwarf	II	11	ID-11,B_9	M-17 (HK)
d-12	yūkara dwarf			ID-12, B_7	N-62 (HK)
d-13	short grained dwarf			ID-13, B_7	M-15 (HK)
d-14	kamikawa-bunwai dwarf	XI	5	ID-14,B_{10}	H-147(HK)
d-17(t)	slender dwarf			ID-17 ,B_8	M-52 (HK)
d-18^h	hosetsu-waisei dwarf	III	3	ID-18^h,B_{10}	N-71 (HK)
d-18^k	kotake-tamanishiki dwarf	III	3	ID-18^k,B_{11}	F1-26(KY)
d-19	kamikawa dwarf			ID-19,B_9	N-56 (HK)
d-27	bunketsuto dwarf	VIII	9	ID-27,B_9	F1-86(KY)
d-30	waisei-shirasasa dwarf	X	8	ID-30,B_7	F1-3 (KY)
d-35(t)	tanginbozu dwarf			ID-35,B_{10}	N-77 (HK)
d-42(t)	liguleless dwarf	II	11	ID-42,B_6	M-341(HK)
sd-1	dee-geo-woo-gen dwarf	III	3	ID-47,B_7	I-120(IR)

Reference; Kinoshita and Shinbashi (1982)

Institution; Plant Breeding Institute, Faculty of Agriculture
Hokkaido University, Sapporo, 060 Japan.

Isogenic lines for semidwarf-1 (Dee-geo-woo-gen dwarf)

Strains: SC 2,3,4,5

Reference: Itakura et al. (1983).

Institution: National Agricultural Research Center, Yatabe, Tsukuba, 305 Japan.

3. Isogenic lines of Taichung 65 for marker genes

Gene symbol	Character	Linkage group	Chromo-some	Strain and backcrossings	Donor
wx	glutinous endosperm	I	6	E7=T65*wx*, B_{14}(GI,CH), B_{27}(RY)	563(GI), Kinoshitamochi (CH)
fs-1	fine stripe-1	I	6	T65*fs-1*, B_7(RY)	
Cl	Clustered spikelets	I	6	T65*Cl*, B_9(RY)	
Ur-1	Undulated rachis-1	I	6	T65*Ur-1*, B_3(RY)	
d-2	ebisu dwarf	II	11	E16=T65*d-2*, B_8(RY) (GI,CH), B_9(RY)	H-79(HK)
Pl	Purple leaf	II	11	T65*Pl*, B_6(RY)	
lg	liguleless	II	11	E8=T65*lg*, B_8 (GI,CH), B_{11}(RY)	H-79(HK)
Ph	Phenol staining	II	11	E15=T65*Ph*, B_8(GI,CH)	414(GI), P.T.B-10(CH)
d-11(d-8)	norin-28 dwarf	II	11	T65*d-11*, B_3(RY)	
sd-1(d-47)	dee-geo-woo-gen dwarf	III	3	T65*d-47*,(CH), B_6(RY)	Taichung-native-1(CH)
A	Anthocyanin activator	III	3	T65*A* B_{11}(RY)	
Rd	Red pericarp	III	3	T65*Rd*,(CH), B_{10}(RY)	P.T.B.-10(CH)
Pn	Purple node	III	3	T65*Pn*, B_{11}(RY)	
g-1	long sterile lemmas-1	IV	10	E12=T65*g-1*, B_8(GI,CH), B_{14}(RY)	868(GI)
Rc	Brown pericarp	IV	10	E14=T65*Rc*, B_8(GI,CH,RY)	414(GI)
gh-1	gold hull and internode-1	VI+IX	2	T65*gh-1*, B_7(CH,RY)	
d-1	daikoku dwarf	VI+IX	2	E-17=T65*d-1*, B_7(GI) B_6(RY)	H-80(HK)
ri	verticillate rachis	VI+IX	2	T65*ri*, B_5(RY)	
nl-1	neck leaf-1	VI+IX	2	E11=T65*nl-1*, B_8 (GI,CH,RY)	H-69(HK)
gl-1	glabrous leaf blade	VI+IX	2	E10=T65*gl-1*, B_8(GI) B_{10}(RY)	H-90(HK)
Dn-1	Dense panicle-1	VII	1	T65 *Dn-1*, B_8(RY)	
I-Bf	Inhibitor for brown furrows	V	1	T65*I-Bf*, B_3(RY)	
d-27(d-t)	bunketsuto dwarf	VIII	9	T65*d-27*,(CH)	
la	'lazy' growth habit	VIII	9	E13=T65*la*, B_8(GI,CH). B_6(RY)	H-79(HK)
Ef-1^b	Earliness-1	VIII	9	T65*E*^{b}, B_{14}(CH,RY)	
bc-1	brittle culm-1	XI	5	E9:T65 *bc-1*, B_8(GI) B_{10}(RY)	H-79(HK)
bl-1	brown leaf spot-1	X	8	T65 *bl-1*, B_3(RY)	

dl(lop)	drooping leaf	XI	5	T65*dl*, B_5(RY)
Hg	Hairy glume	XII		T65 *Hg*, B_8(RY)
pgl	pale green leaf		7	T65*pgl*, B_8(RY)
fgl(fl)	faded green leaf		7	T65*fgl*, B_{10}(RY)
Ef-2	Earliness-2		7	T65*Ef-2*, B_{10}(RY)
An-4(t)	Awn-4		12	T65*An-4*, B_{10}(RY)
wx & Rc	glutinous endosperm and Red pericarp	I & IV	6 & 10	E18=T65*wx,Rc* ($B_{12}F_3$ x B_7F_3)F_3 (GI,CH)
lg & g-1	liguleless and long sterile lemmas-1	II & IV	11 & 10	E21=T65*lg,g-1*, (B_7F_3 x B_7F_3)F_3 (GI,CH)
gl-1 & la	glabrous leaf and hull and 'lazy' growth habit	VI+IX & VIII	2 & 9	E20=T65*gl,la* (B_7F_3 x B_7F_3)F_3 (GI,CH)
nl-1 & bc-1	neck leaf-1 and brittle culm-1	VI+IX & XI	2 & 5	E19=T65*nl,bc-1*(B_7F_3 x B_7F_3)F_3 (GI,CH)

Reference; Oka and Morishima (1974), Sano and Oka (1977), Tsai (1973),

Institution: National Institute of Genetics, Mishima, 411 Japan.

4. Isogenic lines of Taichung 65 for earliness

Gene symbol	Character	Linkage group	Chromo some	Strain	Donor
$Ef\text{-}1^+$(=*ef-1*)	Recessive allele	VIII	9	T65e	Taichung 65
$Ef\text{-}1^a$	Early flowering Ca.10 days			T65E^a(A3)	Tatung-tsailai
$Ef\text{-}1^b$	do.			T65E^b(B96)	Bozu 5gō
$Ef\text{-}1^\gamma$	do.			T65E^γ(I123)	γ-rayed to T65e
$Ef\text{-}1^X$	do.			T65E^X(I190)	X-rayed to T65e
$m^a\text{-}Ef\text{-}1^+$	Emphatic gene of *E*-locus	IV	10	T65*e-em*^{a}(A4-58)	Tatung-tsailai
$m^b\text{-}Ef\text{-}1^+$	do.			T65*e-em*^{b}(B172)	Bozu 5gō
$m^*\text{-}Ef\text{-}1^a$	do.			T65E^a-*em*($A4_7$)	E^a X *em*
$m^*\text{-}Ef\text{-}1^b$	do.			T65E^b-*em*(B20)	E^b X *em*
$m^*\text{-}Ef\text{-}1^\gamma$	do.			T65E^γ-*em*	E^γ X *em*
$m^*\text{-}Ef\text{-}1^X$	do.			T65E^X-*em*	E^X X *em*

* Including m^a and m^b.

Reference: Tsai and Oka (1970), Tsai (1973, 1980, 1984).

Institution: Department of Agronomy, National Chung-Hsing University, Taichung, Republic of China.

5. Isogenic translocation lines of T65 with marker genes

Gene symbol	Character	Chromosomes involved	Strain
gl-1	glabrous leaf blade	2-3	E42=T65*Tr34.gl*

nl-1	neck leaf-1	2-6	E43 =T65*Tr*52.*nl* (T65*nl.bc* X T65*Tr*52)F_3
g-1	long sterile lemmas-1	5-10	E44=T65*Tr*44.*g-1* (T65*lg.g* X T65*Tr*44)F_3

Reference: Sano and Oka (1977)

Institution: National Institute of Genetics, Mishima, 411 Japan.

6. Isogenic lines for blast resistance

A. Isogenic lines of Fujisaka 5gō

Strains: ZTR (113905-020906)... *Pi-z*^{t}, *Pi-i*

ZTS (113905-020907)... *Pi-i*

Backcross and Donor: B_4, Morak Sepilai (*Pi-z*^{t})

Reference: Yokoo (1983)

Institution: National Institute of Agrobiological Resources, Yatabe, Tsukuba, 305 Japan.

B. Isogenic lines of Nipponbare

Gene symbol	Linkage group	Chromosome	Strain	Backcrosses	Donor
Pi-i	I	6	Kanto-IL6,IL7,IL13	B_4	Todoroki-wase
Pi-z	I	6	Kanto-IL4,IL5	B_4	Ōu-287 gō
Pi-z^t			Kanto-IL18,IL19	B_4	T3B205
Pi-ta^2	VII	1	Kanto-IL10,IL11	B_4	Etsuman-109 gō
Pi-k	VIII	9	Kanto-IL2,IL3,IL12	B_4	Kusabue
Pi-b	X	8	Kanto-IL1,IL14	B_4	BL-1

Reference: Horisue et al. (1984)

Institution: National Agriculture Research Center, Yatabe, Tsukuba, 305 Japan.

Induced mutants from *Oryza glaberrima* strain GMS

Most varieties of *O. glaberrima* have red pericarp and sensitive to photoperiod. Plants with colorless pericarp and non-sensitive to photoperiod were selected from W492 (non-sensitive) x C7432 (white grain) and other 16 crosses, in 1970. The data showed that white grain was monogenic recessive. From the bulk of selected plants, a strain, GMS, was selected, and was used for EMS (0.05M, 5 hrs) treatment in 1974. Mutants were selected from ca. 7,000 M_2 plants in 1976, and the final selection was made in 1979 (M_4). The mutant genes listed were found to be allelic to the respective genes of *O. sativa*. In addition, round kernel (E108), spotted leaf (E111), and early-flowering (E112) were obtained, for which the genes remain unidentified.

Gene aymbol	Character	Linkage group	Chromo-some	Strain	Remarks
wx	glutinous endosperm	I	6	E106=GMS-2	EMS induced M_4
lg	liguleless	II	11	E109=GMS-4,8-1	do.
Rc^+	White pericarp	IV	10	E105=GMS-1	Selection from hybrid
d-1	daikoku dwarf	VI+IX	2	E107=GMS-11-2	EMS induced M_4
bc-1	brittle culm-1	XI	5	E110=GMS-85-4	do.

Reference: Oka (1977), Sano (1977, 1979).

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E. RESEARCH NOTES

I. General genetics

1. Induced semidwarf mutants

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In the last 14 years several semidwarf mutants have been induced in rice cultivars in California. The most useful mutant was released in 1976 as the semidwarf cultivar Calrose 76. It originated as a single gene semidwarf mutant from the very well-adapted tall cultivar Calrose. Genetic studies showed that Calrose 76 possessed a single recessive gene for semidwarfism, designated sd_1. The sd_1 gene reduces plant height about 25% through approximately proportional reductions in lengths of the top five internodes; panicle length remains essentially unchanged.

In practice Calrose 76 has been more important as an adapted semidwarf donor than as a cultivar per se. It has been the source of semidwarfism either directly or indirectly for five additional cultivars released from the cooperative industry-state-federal breeding program in California: M7, M-101, S-201, M-301 and M-302. Height of the California semidwarfs is generally about 90 cm, compared to 120-130 cm for the previous tall cultivars. The cumulative evidence indicates that the semidwarfing gene increases rice yields 15%, and when the semidwarf cultivars are used with intensified cultural practices, farm yields increase about 25%.

Genetic studies have shown that the induced mutant gene sd_1 is allellic to the major semidwarfing gene in Dee-geo-woo-gen (DGWG) and the widely grown Green Revolution cultivars derived from DGWG. Thus, in F_2 generations of crosses between sd_1 and DGWG types, no truly tall recombinants have been recovered, although considerable variation exists in height of the F_2 semidwarfs (Foster and Rutger 1978; Mackill and Rutger 1979).

Allelism tests have shown that at least three independent, recessively inherited semidwarf genes were induced in the tall cultivar Calrose: the sd_1 locus present in Calrose 76, the sd_2 locus in CI11033, and the sd_4 locus in CI11034. However, neither the sd_2 nor the sd_4 source has been as agronomically useful as the sd_1 source. The sd_4 source reduces height only 15 cm and has an additional pleiotropic effect for a 20% reduction in seed size.

After the three independent semidwarfing genes sd_1, sd_2, and sd_4 were identified, subsequent genetic studies concentrated only on determining if new mutants were allelic to sd_1. To date, seven additional semidwarfs have been found to be non-allelic to sd_1. Again, none has been as agronomically useful as the sd_1 source. Overall, semidwarf mutants have been induced in ten different rice cultivars in U.S.A.

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2. Semidwarfing genes of high-yielding rice varieties in Japan

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Two distinct sources of semidwarfism have contributed to a break-through in the yield level of rice in 1960's in Japan. They are Reimei, a mutant induced from Fujiminori in northern Japan (Futsuhara 1968), and Shiranui and its sister lines derived from a native dwarf, Jikkoku in southern Japan (Okada et al. 1967). These varieties were widely used as parents in cross-breeding programs and many short-statured high-yielding varieties have been developed.

Semidwarfism has also been introduced into high-yielding rice varieties in the tropics and other areas since 1960's. The Green Revolution is a direct achievement of an intensive use of the tropical semidwarfs. On the other hand, cumulative evidence has indicated that most of the short-statured varieties possess the same gene for semidwarfism, as a Taiwan-native variety Dee-geo-woo-gen (DGWG) was the common gene source (Hargrove 1979; Mackill and Rutger 1979; Chang and Li 1980). Short-statured mutant lines induced from tall native varieties, selected for high-yielding potential, also had the dwarfing gene at the same locus (Hu 1973). In conceiving breeding strategies for high-yielding varieties in Japan, it is of primary importance to investigate whether or not the Japanese semidwarf varieties have the same gene as DGWG.

The allelism test of semidwarfing genes in the Indica and Japonica groups is not always easy because of hybrid sterility and transgressive segregation for delayed maturity. We have overcome this difficulty by use of isogenic lines. By transferring the semidwarfism from Taichung native 1 having the DGWG gene and another semidwarfism from Shiranui with the gene of Jikkoku into a Japanese tall variety, Norin 29 through 4 times of backcrosses, two series of near-isogenic lines were obtained, which were desingated as SC 2 and SC 3 (Taichung Native 1/5* Norin 29) and SC 4 and SC 5 (Shiranui/5* Norin 29).

To investigate the genetic bahavior of the semidwarfism, the near-isogenic lines were crossed with Norin 29 and the F_1 and F_2 plants were observed for culm length at National Institute of Agricultural Sciences in 1981. The long culm of Norin 29 was found to be partly dominant since the F_1 plants had shorter culms than Norin 29. The F_2 clearly segregated into 3 tall : 1 short types indicating that the semidwarfism of DGWG and that of Jikkoku were each controlled by a recessive gene. Then, SC 4 and 5 were crossed with SC 2 and 3. The F_1 plants had as short culms as of the parents and the F_2 showed a narrow range of variation in culm length around the F_1 and parental mean. This indicates that DGWG and Jikkoku have the same semidwarfing gene.

Furthermore, the semidwarfism of Reimei is known to be controlled by a single gene with incomplete dominance (Futsuhara 1968). When Reimei was crossed with SC2, the F_1 plants had slightly shorter culms than of Reimei as SC 2 was shorter than Reimei, and the range of F_2 segregation was between the parental values. This suggests that Reimei also has the same semidwarfing gene as DGWG although it has modifiers which increase the culm length.

It is known that Calrose 76, an induced semidwarf mutant grown in California, also has the same gene (foregoing note by J. N. Rutger). Suh and Heu (1978) have shown that the semidwarfism of a Korean variety Tongil (IR 8//Yukara/Taichung Native 1) is controlled by a single recessive gene, *d-t*, which is linked with the marker genes such as *A* (anthocyanin activator), *Pp*(brown pericarp), *Pn*(purple node), and *Pau*(purple auricle) of linkage group III with recombination values of 24.8%, 35.1%, 40.9% and 42.9%, respectively. It is of particular interest to find that all the genes carried by semidwarfs of economic importance are at the same locus as that of DGWG despite the differences in genetic background. There may be a potential danger of reducing genetic diversity

by the frequent use of the same gene. Semidwarfing genes of economic use at non-allelic loci are now being searched for.

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3. Semidwarfing genes in rice germplasm collection

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A single recessive gene (sd_1) from 3 independent Chinese sources—Dee-geo-woo-gen, Ai-jiao-nan-te and Ai-zai-zhan, has led to the development of semidwarf rice varieties in Taiwan and mainland China, several Asian Countries, and at IRRI. This gene has lent great impetus to rice production increases in many Asian countries.

Ever since IRRI began its research operations in 1962, we have been interested in the genetic control of the improved plant type and the sources of semidwarfing genes. This article summarizes our decades of search for other sources of semidwarfs in the rice germplasm collection of IRRI.

We have identified about 145 short-statured accessions from the IRRI germplasm collection and have made more than 200 crosses to date. Each of them was crossed with Taichung (Native)1, IR8, IR20 or IR36 to test the allelic relationship of their semidwarfing gene(s) with that of sd_1.

From a concurrent planting and comparison of the two parents, F_1 plants and F_2 progenies, the gene systems in the new semidwarfs were classified into 3 categories:

I. Identical with sd_1 locus: F_1 plants and all F_2 plants are semidwarfs, although the effect of modifying genes may be detected.

a) Purbachi; derivatives of Ai-zai-zhan and Ai-jiao-nan-te (China). b) Induced Mutants of I-kung-bau and Keh-tze (Taiwan). c) C53-39, Khunnaywayin(S) and (P), Khunni Shay, Nga-Kywe (Burma). d) Culture 147, Culture 155, T141/Baok 360, ARC 5929 and other ARC semidwarfs, Crm 13-3241, BM 13, CRHP8, Jikkoku/Shiranui (52-37; 52-102), Shiranui/Seraup Kechili 55-296, Shiranui/Gionchiew 61-8, CNM 25, C12329, IET 2895, New Sabarmati, Barmda 21, 23 and 828, KH 863 (India). e) "California 2" (Philippines). f) Calrose 76, M9,

M101, M302, S201 and LA 110 (U.S.A.).

II. Sharing a compound locus with sd_1: F_1 plants are semidwarf; great majority of the F_2 plants are semidwarfs; only a small number of F_2 plants (less than 1%) are intermediate-tall or tall. a) Fukunishiki, Kochihibiki and Reimei (Japan). b) Gora (NCS 16), Synthetic Sativa and TR17 (India). c) Sekarmandi (Indonesia).

III. Non-allelic gene(s): F_1 plants are taller than either parent: distinct segregation for plant height is observed in the F_2 populations.

a) CI 9649/CI 9722, CI 9858, CP231/SLO-17 (B5580A1-15), Double Dwarf 1, Intermediate Dwarf, Short Straw Dawn, Short Straw Starbonnet (U.S.A.). b) CN 242d_3, Tainan-5-mutants 72-534, 72-536; Tainan-5-mutants 73-66, 73-75, 73-111, TNA 761-1-148-157, Tainan 6 mutants 772039, 772040 (Taiwan). c) 2243-85F and IRAM 2165 (Ivory Coast). d) Culture 854, Culture 956, d_6, d_7, d_8, d_{10}, Kalimonch Dwarf Mutant, Mutant 65, P-3 dwarf (India). e) Nadula Dwarf (Fiji). f) Fanny Dwarf (France). g) GS 1649A (China via Thailand). h) K8 mutant (Sri Lanka). i) Hoyoku (Japan).

The above tabulation, however, does not include many semidwarfs whose pedigrees may be traced to one of the known Chinese semidwarf sources (sd_1).

Plant height, being a quantitative trait, is subject to environmental influence of temperature, photoperiod and soil fertility. Many short-statured varieties and lines from temperate zones tend to be shorter and earlier at IRRI than in their home habitat. Hence, the classification of the progenies may be affected by genotype x environment interactions.

Genetic incompatibility between ecogeographic races may also lead to aberrant segregation. Cytoplasmic-nuclear interaction may be detected in some reciprocal crosses.

Additional genes inhibiting tall height may affect the expression of F_2 plants which were reported to have the sd_1 gene.

Some of the accessions may have been outcrossed or mislabelled when handled by workers in a different country. Some "dwarfs" are more likely semidwarfs.

The sd_1 gene appears to belong to a readily mutable locus so that many spontaneous and induced mutations had occurred at the same site. Its compound nature needs to be further studied by expanded F_2 populations, say, over 10,000 F_2 plants per cross.

Among those semidwarfs who have gene(s) non-allelic to sd_1, the great majority of them were extremely poor in agronomic characters, i.e., weak growth vigor, shorter height than is desired, poor tillering, unattractive panicles, poor panicle exsertion, poor grain filling, hard threshability, and miniaturized grains. Only the following strains appeared relatively better: CP231/SLO-17 (Acc. 6993), Culture 854, Culture 956, D66, P-3 dwarf, Tainan 5 mutants (72-534, 72-536), Reimei, and Hoyoku. But none of them can compare with IR8, TN1 or IR36 in growth vigor and grain yield under Los Baños conditions.

4. Establishment of differential varieties for pathogenicity test of rice blast fungus

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Varieties with different single resistance genes are ideal differential varieties for pathogenicity test of the pathogen (Flor 1945). In Japan, the first differential varieties for rice blast were chosen by Goto and co-workers (1961, 1964). Yamasaki and Kiyosawa (1966) initiated gene analysis using

seven fungus strains selected in the light of Goto's work and detected three resistance genes, *Pi-a*, *Pi-i* and *Pi-k*. Since then, 13 genes have been identified (Kiyosawa 1972). Since 1960's, the author has tried to develop lines with a single gene from varieties having more than one gene. For example, resistance gene *Pi-ta* was isolated from the hybrid between Pi No. 1 having *Pi-a* and *Pi-ta* and Norin 8 which has no resistance gene (Kiyosawa 1966). Line K 1 with *Pi-ta* was thus obtained (Kiyosawa 1967). Also, line K 60 with *Pi-k*^{p} was obtained from K 2 x Shin 2; K 2 having *Pi-a* and *Pi-k*^{p} was derived from Pusur x Norin 22. *Pi-k*^{p} was first found in Pusur, a variety from Pakistan (Kiyosawa 1969). K 59 with *Pi-t* was obtained from BL 10 (*Pi-b Pi-t*) x Kanto 51 (*Pi-k*) (Kiyosawa 1972).

The strains with different single resistance genes thus obtained, as listed in Table 1, are useful as differential varieties to classify fungus isolates into groups differing in pathogenicity. In addition, another set of differential varieties was established by Yamada et al. (1976, included in Table 1). By the use of these differential varieties, it has become possible to estimate the structure of populations of the blast fungus in terms of frequency of virulence and avirulence genes. However, it is possible that some of these differential varieties have more than one gene if fungus strains of alien origin are tested.

Table 1. Differential varieties and their resistance genes

Differential varieties of		Gene	Code number	Literature
Kiyosawa	Yamada *et al.*			
Shin 2	Shin 2	*Pi-k*^{s}	1	Kiyosawa 1969
Aichi Asahi	Aichi Asahi	*Pi-a*	2	Kiyosawa *et al.* 1967
Fujisaka 5	Ishikari-shiroke	*Pi-i*	4	Yamasaki and Kiyosawa 1966
Kusabue	Kanto 51	*Pi-k*	10	Yamasaki and Kiyosawa 1966 Kiyosawa 1968
Tsuyuake	Tsuyuake	*Pi-k*^{m}	20	Kiyosawa 1978
Fukunishiki	Fukunishiki	*Pi-z*	40	Kiyosawa 1970
K 1	Yashiro-mochi	*Pi-ta*	100	Kiyosawa 1967, 1969
Pi No. 4	Pi No. 4	*Pi-ta*^{2}	200	Kiyosawa 1969
Toride 1	Toride 1	*Pi-z*^{t}	400	Yokoo and Kiyosawa 1970
K 60		*Pi-k*^{p}	.1	Kiyosawa unpublished
BL 1		*Pi-b*	.2	Yokoo et al. 1978
K 59		*Pi-t*	.4	Kiyosawa 1972

Note: Race number is determined by adding the code number of varieties which show virulent reaction. For example, the race number of isolate which shows virulent reactions to Shin 2 and Kusabue is 1 + 10 = 11.

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5. Multiple alleles at the *Xa-1* and *Xa-kg* loci for resistance to bacterial leaf blight

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The mode of resistance and allelic relationships of genes for the resistance to bacterial leaf blight, caused by *Xanthomonas campestris* pv. *Oryzae* (Ishiyama 1922) Dye 1978, were investigated in a Japanese (Kogyoku) and three IRRI varieties (IR28, IR29 and IR30). The IRRI varieties are resistant to bacterial groups I (strain T7141) and V (H75304) at all stages of plant growth, whereas the Japanese variety shows resistance at mature stage only. Varieties resistant at all stages of plant growth are rare. No varieties are known which show resistance to bacterial groups II, III and IV at all stages of growth.

A test of the F_1 and F_2 plants from crosses between a Japanese susceptible variety Toyonishiki and the IRRI varieties indicated that the resistance to each of the groups I and V was controlled by a single major gene, respectively, and the reactions at the seedling and mature stages to each group were controlled by the same gene. The F_1 and F_2 plants from crosses between Kogyoku and the IRRI varieties were also tested for resistace at both seedling and mature stages. Kogyokyu, susceptible at seedling and resistant at mature stage, is known to have two resistance genes, *Xa-1* for resistance to bacterial group I (Sakaguchi 1967) and *Xa-kg* for resistance to group V (Ogawa et al. 1978). The F_1 plants of all three crosses were resistant at both seedling and magure stages, and the F_2 populations segregated into 3 resistant and 1 susceptible types at the seedling but were

wholly resistant at the mature stage. This indicates that the genes of the IRRI varieties for the resistances to groups I and V were allelic to, and dominant over *Xa-1* and *Xa-kg*, respectively. They were then symbolized *Xa-1*^{h} and *Xa-kg*^{h} respectively.

Xa-1 and *Xa-kg* are closely linked (recombination 2%, Ogawa et al. 1978), and *Xa-1* is also linked with *Ph* for phenol staining (Sakaguchi 1967). These genes are located on chromosome 11. From the segregation data of crosses of IRRI varieties with Toyonishiki, recombination values were estimated as follows: *Xa-1*^{h} — *Xa-kg*^{h} : 2.0 ± 0.65; *Xa-1*^{h} — *Ph* : 2.8 ± 0.77; *Xa-kg*^{h} — *Ph* : 3.7 ± 0.87.

Varieties having *Xa-1*^{h} and *Xa-kg*^{h} which are resistant at all stages of plant growth, cannot serve as the initial source of infection, so far as bacterial groups I and V are concerned. Their resitance is also effective against *kresek* phase of bacterial blight which causes the complete wilting of infected tillers at the seedling stage. The breeder can evaluate the resistance of the materials segregating for these genes in the nursery.

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6. Chromosomal location of *Xa*$_4$ gene

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A dominant gene for resistance to bacterial blight of rice was identified by Petpisit et al. (1977) and it was designated *Xa*$_4$. This gene gives resistance to bacterial blight at all stages of plant growth and has been widely used in the breedling program (Khush 1981). In fact all the bacterial blight resistant IR varieties are homozygous for *Xa*$_4$.

In order to determine the chromosomal location of *Xa*$_4$, we crossed IR29 (*Xa*$_4$ *Xa*$_4$) with 11 of the 12 primary trisomics which were susceptible to bacterial blight. The F_1 progenies (trisomic as well as disomic) of all the trisomics except triplo 7 were resistant. The disomic F_1 plants amongst the progeny of triplo 7 x IR29 were resistant as expected. However, the trisomic F_1 plants of this cross were moderately susceptible. We interpreted these results to be due to dosage effects of *Xa*$_4$ gene. If *Xa*$_4$ is located on chromosome 7, triplo 7 plants would be *Xa*$_4$ + +, and one dose of *Xa*$_4$ would not be enough to convey resistance. To verify this hypothesis we tested an F_2 population from the triplo 7 F_1 plant. Out of 575 F_2 plants, 183 were trisomic and 392 were disomic. Amongst the trisomic fraction 50 plants were resistant and 133 were susceptible or moderately susceptible. This agrees with 2:7 ratio (χ^2:0.141) expected on the basis of dosage effects of a dominant gene (Khush et al. 1984). In the disomic fraction of this cross 301 plants were resistant and 91 were susceptible. These data do not agree with the 5:4 expected ratio. The deviation may be the result of misclassification of some plants.

The F_2 or backcross populations from the ten other trisomic heterozygotes segregated in a normal 3:1 or 1:1 ratios. These results indicate that *Xa*$_4$ is located on chromosome 7.

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7. Genic analysis for resistance to brown planthoppers in rice

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The brown planthopper, *Nilaparvata lugens* Stal. (abbr. BPH), is one of most serious pests of rice throughout Asia. The inheritance of BPH resistance has been investigated by many workers since Athwal et al. (1971). To date, four genes, *Bph-1*, *bph-2*, *Bph-3* and *bph-4*, have been found (Athwal et al. 1971; Lakshminarayana and Khush 1977). We also have been engaged in genic analysis for the resistance.

First, to determine whether or not two or more resistance genes can be combined in a variety, allelism tests were made. The results indicated that, as reported previously (Sidhu and Khush 1978; Ikeda and Kaneda 1981), *Bph-1* and *bph-2* are linked closely, and so are *Bph-3* and *bph-4*. The two linkage groups, however, are independent.

Second, to identify the chromosomes on which the BPH resistance genes are located, each of the trisomic lines obtained from Kyushu University were crossed with Kanto PL 1 or Kanto PL 4 (having *Bph-1*), Rathu Heenati (having *Bph-3*) and Babawee (Having *bph-4*). In the cross with trisomic line E, which has chromosome 11 in triplicate, the F_2 ratio for *Bph-1* significantly differed from 3 : 1 and agreed with a trisomic ratio of 2 : 1. *Bph-1* was thus located on chromosome 11 (linkage group II). Linkage tests showed that *bph-2* was linked with *d-2* belonging to linkage group II, the recombination value being 39.4%. Both *Bph-1* and *bph-2* segregated independently of *lg*, *P1-1* and *d-11* which belong to linkage group II but are distant from *d-2*.

Similarly, *Bph-3* and *bph-4* were found to be located on chromosome 7 (to which no linkage group is assigned yet); trisomic ratios of 2 : 1 for *Bph-3* and 1 : 17 for *bph-4* were found in crosses with trisomic line C having chromosome 7 in excess (Table 1).

Furthermore, three cultivars with unknown resistance genes, Andaragahawewa, PTB 34, and PTB 21, were crossed with resistant and susceptible testers to identify their resistance genes. The results showed that Andaragahawewa and PTB 34 had *Bph*-1 and *bph*-2, respectively. PTB 21 had been known to have a dominant and a recessive gene, one of them being either *Bph-1* or *bph-2* (Lakshmirarayana and Khush 1977). Our data indicated that the second gene of PTB 21 was either *Bph-3* or *bph-4*. Then, 12 F_3 lines from Kochihibiki/PTB 21//Asominori were tested with BPH biotypes I, II and III. The result showed that one of the two genes was *bph-2*. Accordingly, it was concluded that PTB 21 had *bph-2* and *Bph-3*. Although this variety had two resistance genes, but it had no new resistance gene.

Table 1. F_2 ratios for resistance to BPH in the crosses of trisomic lines with Kanto PL 4 (*Bph-1*), Rathu Heenati (*Bph-3*), and Babawee (*bph-4*)

Resistance gene	Cross	No. of F_2 plants R	S	Total	χ^2 (3 : 1 or 1 : 3)
Bph-1	Trisomic E/Kanto PL4	337	182	519	28.1**[a]
	Control (disomic)	981	350	1331	1.2
Bph-3	Trisomic C/Rathu Heenati	78	42	120	7.6**[a]
	Control (disomic)	164	64	225	1.4
bph-4	Trisomic C/Babawee	8	306	314	84.4**[b]
	Control (disomic)	203	634	837	0.3

** Significant at 1% level. a — fitting 2 : 1, b — fitting 1 : 17.
Crosses of other trisomic lines, A, C, G, and H with *Bph-1*, H with *Bph-3*, and A, E, F, and L with *bph-4*, all gave a good fitness to 3 : 1 or 1 : 3 ratio.

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8. Heading-time genes of rice, E_1, E_2 and E_3

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The three heading-time genes, E_1, E_2 and E_3, independent of one another and delaying the heading time of rice, were detected 30 years ago through the intercrosses among three varieties, Gimbôzu, Aikoku and Kyotoasahi (Syakudo et al. 1954; Kawase 1961). From those varieties and intercrosses, seven lines possessing different genotypes for the three genes, EG1 to EG7, were then developed (Table 1).

Making use of these lines as testers, the three genes have been studied on their attribues, and the genes responsible for heading dates of many Japanese varieties have been genetically analysed and identified. The recent experimental results are summarized as follows:

1) Growth-cabinet experiment clearly showed that each of E_1, E_2 and E_3 was a photoperiod-sensitivity gene which did not affect basic vegetative growth. It also showed that the degree of photo-sensitivity, which evenually determined the magnitude of delay in flowering under natural conditions, was ranked as $E_1 \gg E_3 > E_2$ (Table 1).

2) The response of E_1 to a light break (1 hour, over 300 lux) during short-day treatment far exceeded those of the other two genes.

3) The heading-delaying effects of E_2 and E_3 under natural condition were significantly lower than that of E_1. However, E_3 showed a synergistic effect when it coexisted with E_1, and E_2 also did when it coexisted with both E_1 and E_3 (Table 1).

4) So far as the 15 varieties tested are concerned, all varieties grown south of Tohoku district possessed E_1 (Yamagata 1984).

5) Yokoo et al. (1980) reported the presence of a photo-sensitivity gene, *Lm* (late maturity), belonging to linkage group I. In spite of its similarity in action, this gene was found to be independent of E_1, indicating the necessity for further studies of the genetic mechanisms controlling heading time in rice.

Table 1. Basic vegetative growth (BVG), photoperiod sensitivity, and days to heading under natural condition of seven lines having different genotypes for E_1, E_2 and E_3

Line	Genotype[a]	BVG[b]	Photoperiod[c] sensitivity	Days to heading[d] in natural daylength
EG 1	$E_1e_2e_3$	37	58	112
EG 2	$e_1E_2e_3$	38	46	97
EG 3	$e_1e_2E_3$	38	52	101
EG 4	$E_1E_2e_3$	40	60	117
EG 5	$e_1E_2E_3$	39	54	105
EG 6	$E_1e_2E_3$	37	81	121
EG 7	$E_1E_2E_3$	41	92	131

a) Genotype for E_1 E_2 and E_3, all lines being homozygous.
b) Days from sowing to heading under short day (10 h) at 30°C.
c) Difference in days to heading between a long day (14 h) and a short day (10 h) at 30°C.
d) Sown on May 6, 1980.

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II. Sterility and varietal differentiation

9. Inheritance of fertility restoration in a rice cross

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IR54, a rice variety developed at IRRI and released in 1980, was identified as an effective restorer of cytoplasmic genetic male sterile lines (viz., Zhen Shan 97A, V20A, V41A) possessing 'WA' cyto-sterility system. We studied the inheritance of its fertility restoration ability in the cross: Zhen Shan 97A/IR54. Progenies of Zhen Shan 97A (P_1), IR54 (P_2), Zhen Shan 97A/IR54, (F_1, F_2)and Zhen Shan 97A/Zhen Shan 97A/IR54 (BC1 F_1) were analyzed with regard to pollen and spikelet fertility. The F_1 showed pollen and spikelet fertility similar to the restorer parent, IR54, indicating that restoration ability was dominant and the cytoplasmic-genetic sterility system of Zhen Shan 97A was sporophytic in nature. With regard to pollen fertility, determined by staining with 1% IKI solution, the F_2 population segregated into fully fertile (like IR54), fertile (70—95%), partially fertile (1—70%) and completely sterile (like Zhen Shan 97A) giving a good fit to digenic ratio 9:3:3:1 (Table 1). The backcross segregation conformed to 1:1:1:1 ratio. Segregation for spikelet fertility in F_2 and backcross generations (Table 2) conformed to the results on pollen fertility. It appears that the restoration ability of IR54 is governed by two independent major genes, one of them has a stronger fertility restoration ability than the other, such that if both genes are present, fertility is like the restorer line, IR54; if the gene with stronger fertility restorattion ability is present alone fertility is somewhat reduced, but if the gene with weaker restoration ability is present alone, plants show partial fertility ranging between 1—70% (for pollen) and 1—30% (for spikelet). The plants possessing the double recessive genotype are completely sterile like Zhen Shan 97A.

Table 1. Frequency distribution for pollen fertility (%) in parental F_1, F_2 and BC_1 F_1 populations of the cross Zhen Shan 97A/IR54

Population	Pollen fertility (%) class						Total	χ^2	P value
	0	0.1-10	10-30	30-70	70-95	95-100			
Zhen Shan 97A (P_1)	10						10		
IR54 R (P_2)						10			
F_1 (P_1/P_2)						10	10		
F_2 (P_1/P_2)	30	4	9 (72)	59	84	273	459	3.23[a]	0.38
BC_1 F_1 ($P_1//P_1/P_2$)		2	15 (17)	21	6 (27)	24	92	2.34[b]	0.50

[a] Segregation ratio 9:3:3:1; [b] Segregation ratio 1:1:1:1.

Table 2. Frequency distribution for spikelet fertility (%) in parental F_1, F_2 and BC_1 F_1 populations of the cross Zhen Shan 97A/IR54

Line	Spikelet fertility (%) class							Total	χ^2	P value
	0-1	1-10	10-30	30-50	50-70	70-80	80-100			
Zhen Shan 97A (P_1)	10							10		
IR54 (P_2)					4	2	4	10		
F_1 (P_1/P_2)				2	3	2	3	10		
F_2	27	22	69 (91)	107	159	52	23 (234)	459	7.78[a]	0.06
BC_1 F_1 ($P_1//P_1/P_2$)	30	17	8 (25)	13	17	3 (24)	4	92	6.70[b]	0.09

[a] Segregation ratio 9:3:3:1; [b] Segregation ratio 1:1:1:1.

10. Genetics of fertility restoration and biochemical basis of male sterility-fertility restoration system in rice

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Inheritance of fertility restoration was studied in F_2's of ten crosses of the Chinese male sterile lines, viz., Zhen Shan 97A (97A)— and V20A with a set of complete restorers through chi-square analysis. The mode of inheritance of fertility restoration appeared to vary with the restorers. One to three genes appeared to control the restoration (Table 1). While three restorers IR26, IR50 and Pusa 37—3 restored the fertility of V20A monogenically, in case of Pusa 245-51-1 two genes interacting in a complementary fashion (9F:7S) seemed to be involved. In crosses involving different restorer lines three genes appeared to control the fertility restoration. Appearance of partial fertile segregants in crosses with complete restorers suggested the probable role of modifiers in fertility restoration.

Soluble protein and esterase isoenzyme patterns in matured anthers and spikelets (at meiotic stage) of male sterile and maintainer lines were studied through isoelectric focussing technique. Soluble protein and esterase isoenzyme patterns of matured anthers of male steriles and maintainers differed qualitatively and quantitatively. Also, these biochemical parameters differed between matured anthers and spikelets. Nevertheless, comparison of soluble protein and esterase isoenzyme patterns of spikelets of male sterile and maintainer lines revealed no differences between them. Therefore, presence or absence of certain specific soluble protein and esterase isoenzyme bands might have a bearing on pollen abortion and consequent male sterility. Absence of differences between spikelets (at meiotic stage) of male steriles and maintainers suggested that possibly the differences tended to appear late during the process of pollen development.

Amino acid analysis of matured anthers of male sterile, maintainer and restorer (IR36) lines revealed that proline content in anthers of maintainer/restorer lines was 3 to 6 times more than in the sterile anthers. On the other hand, we observed appreciable increase in the level of aspartic acid in the sterile anthers. No difference was, however, found between anthers of maintainer and restorer lines for any of the amino acids analyzed. This indicated that proline and aspartic acid in the sterile anthers may be in some way related to the phenomenon of male sterility. This relationship, however, needs further investigation.

Table 1. Genetics of fertility restoration of two Chinese cytoplsmic-genetic male sterile lines.

S. No.	Crosses	Number of F_2 plants scored	Degree of restoration*			Genetic ratio	Level of probability
			Fully fertile	Partial fertile	Sterile		
1.	V20A x IR26	437	313	24	100	3:1	0.2-0.3
2.	V20A x Pusa 37-3	505	332	39	134	3:1	0.05-0.1
3.	V20A x IR50	475	344	25	131	3:1	0.5-0.7
4.	97A x Pusa 245-51-1	200	88	22	90	9:7	0.7-0.9
5.	97A x IR19793-25-2-2-2	343	139	8	196	27:37	0.7-0.9
6.	97A x Mijingem	139	16	38	85	27:37	0.3-0.5
7.	V20A x NDC 28	518	33	184	301	27:37	0.7-0.9
8.	V20A x IET 4141	518	282	67	169	45:19	0.1-0.2
9.	V20A x NDC 50	495	317	25	153	45:19	0.5-0.7
10.	97A x IR9761-19-1	200	123	5	72	45:19	0.05-0.1

*Fully fertile and partial fertile classes were merged into one class to enable X^2 analysis.

11. A probable new male sterile line with cytoplasm from a Boro rice of Uttar Pradesh, India

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In search of the alternative sources of sterile cytoplasm, 51 direct and reciprocal crosses were made at IRRI with 16 advanced breeding lines of IRRI and 23 *indica/japaonica* derivatives from Korean rice breeding program in 1980. Most of the F_1s were normal but partial sterility was observed in crosses involving IR46, IR10154-23-3-3, FR 43 B and IR13426-19-2. Reciprocal differences for sterility were significant in these cross-combinations.

In the backcross generations, segregation for sterility occurred which ranged from 1.7 to 98.3 percent in the cross UPRB 31/IR46. As a result, this cross was studied further and backcross F_2 was grown during Kharif, 1982 at Agricultural Research Institute, Patna, India. There were 70 fertile and 170 sterile plants. Few sterile plants with white anthers were detected late in the season so that planned backcrosses could not be made and open pollinated seeds were collected. During Kharif, 1983, 6 plant progeny rows each having 40 plants were grown. Two progenies had as high as 80 percent sterile plants and sterile plants were 100% pollen sterile. Further backcrosses have been made to maintain and multiply these plants. This male sterile line is being named as Patna CMS-1.

12. Geographical distribution of the genes for black hull coloration

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The F_1 plants of crosses between distantly related rice varieties, as well as the wild relatives of cultivated rice, often show black hull coloration. It was reported that the black hull coloration was controlled by one dominant gene (Kuang et al. 1946; Jodon 1964), or two complementary genes (Chao 1928; Mitra and Ganguli 1937; Kuriyama and Kudo 1967), or three complementary genes (Nagao and Takahashi 1954; Rao and Seetharaman 1973). The present author confirmed that the

complementary genes controlling this character were three, symbolized as *Bh-a, Bh-b* and *Bh-c*. The *Ph* gene for phenol reaction was also found to be responsible for this character (Kuriyama and Kudo 1967; Rao and Seetharaman 1973), and to correspond to *Bh-c*. The frequency of *Ph* is high in the Indica and low in the Japonica type (Oka 1953); phenol reaction can be used for distinguishing between the two types with an about 10% probability of mis-classification (Morishima and Oka 1981). Kinoshita and Takahashi (1976) suggested that the distributions of *Bh-a* and *Bh-b* were also localized geographically in relation to the Indica-Japonica differentiation.

The presence of dominant or recessive allele at the *Bh-a* and *Bh-b* loci in a given variety can be known by observing the F_1 plants from crosses with test strains having *Bh-a* + *Bh-c* (=*Ph*) and + *Bh-b Bh-c* (=*Ph*), respectively, and that for the *Bh-c* locus simply by phenol reaction of the grain. By this method, a total of 294 varieties from different parts of the world were examined. The result showed that among the varieties sampled, the frequency of *Bh-a* was high (79.6%) while that of *Bh-b* was much lower (15.6%); the frequency of *Bh-c* (=*Ph*, representing the Indica type) was 25.5%. *Ph* and *Bh-b* were recombined at random, but the association between *Ph* and *Bh-a*, as well as between *Bh-a* and *Bh-b*, significantly deviated from random assortment. Most varieties with *Ph* had *Bh-a*, while about 1/3 of varieties with ph (Japonica) had its recessive allele (*bh-a*, shown by + in Table 1).

The geographical distribution of these genes is shown in Table 1. The frequency of *Bh-a* was highest (100%) in the varietal groups from India, Sri Lanka, Pakistan, Burma, Nepal and Bhutan, and was lowest (27%) in the varieties from Hokkaido, Japan. In contrast, the frequency of *Bh-b*, which was generally low in other regions, was quite high (78%) among Hokkaido varieties.

Table 1. Geographical distribution of genotypes for three genes controlling black hull coloration (in %)

Region	*Bh-c* (= *Ph*)				+ (= *ph*)				No. of var. s tested
	Bh-a *Bh-b*	*Bh-a* +	+ *Bh-b*	+ +	*Bh-a* *Bh-b*	*Bh-a* +	+ *Bh-b*	+ +	
Japan, Hokkaido			4.6		13.6	9.1	59.1	13.6	22
Japan, other parts		1.4			1.4	59.5	2.7	35.1	74
China, Korea & Taiwan		62.8			7.0	25.6	2.3	2.3	43
Indochina & Philippines		7.1				85.7		7.1	14
Indonesia		21.7				69.6		8.7	23
India, Sri Lanka, Pakistan & Burma	12.9	40.3			6.5	40.3			62
Nepal & Bhutan	6.3	31.3			6.3	56.3			16
USSR & East Europe					33.3	55.6	11.1		9
U.S.A.	4.0				8.0	52.0		36.0	25
Others					16.7	83.3			6
Total	3.4	21.8	0.3		6.1	48.3	5.8	14.3	294
Expected from random combination	3.2	17.1	0.8	4.4	9.3	50.0	2.4	12.8	X^2 = 35.6[a]

a — Comparison between observed and expected numbers of varieties, significant at 1% level.

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13. The genetic basis of hybrid chlorosis found in a cross between two Japanese native cultivars

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We incidentally found a case of hybrid chlorosis in the F_2 population of a cross between two Japanese native cultivars, J-147 and J-321. Its first symptom was a change in color of the third or fourth leaf-blade to yellowish. The chlorotic plants died within 30 days after germination, hence no seed was obtainable from them.

The F_2 population segregated into 849 normal and 63 chlorotic plants, giving a good fit to the 15 : 1 ratio. The F_3 lines showing segregation ratios of 1 : 0, 3 : 1 and 15 : 1 numbered 72, 35 and 37, respectively. This F_3 ratio fitted 7 : 4 : 4, which was expected on the assumption of two independent recessive genes (Table 1). The data thus indicated that there was a set of duplicate genes independent of each other whose double-recessive combination causes chlorosis. They were symboled *ch-1-d* and *ch-1-a*, tentatively.

Table 1. Segregation ratios of F_2 plants and F_3 lines into normal and chlorotic phenotypes in J-147 x J-321

Generation	No. of plants or lines				χ^2	P
F_2,	Normal	Chlorotic		Total		
Observed	849	63		912		
Exp. (15 : 1)	855	57		912	0.67	>0.3
F_3,	1 : 0	3 : 1	15 : 1	Total		
Observed	72	35	37	144		
Exp. (7 : 4 : 4)	67.2	38.4	38.4	144	0.69	>0.7

Sato and Hayashi (1983) reported the presence of a set of complementary lethal genes, *L-2-a* and *L-2-b* causing F_1 weakness, and that the distibution of *L-2-a* was suggestive of the phylogenetic relationship among varietal groups and the mode of dissemination of rice varieties. The distribution of the hybrid chlorosis genes may also be useful for such studies.

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III. New genes and mutants

14. Inheritance of two anatomical characteristics

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An extra sclerenchymatous band is found in the stem of some special rice varietites but not in others. Two rice varieties FR13A and Intan have such bands. Its occurrence was investigated in four crosses between varieties having the band and those lacking the band (Ratna, Jana, CRM 13 named 'Sattari'). The main stems from five F_1 and 100 F_2 plants of each cross were cross-sectioned for observation. All the plants showed the extra band although its continuity as a circular band and its thickness varied among crosses. The F_2 populations showed, in terms of the presence or absence, a 9 : 7 ratio indicating that two complementary dominant genes were involved. The mode of variation among plants also suggested segregation for some modifiers. The complementary genes were symbolized Esb_1 and Esb_2.

The fuscoid cells are those with big vacuoles occurring on either side of the vascular bundle in the leaf blade. Such cells are commonly found in bamboo but are rather rare in rice. A salt-tolerant variety, Phulbuh, having such cells was crossed CR 1039(M) which lacks such cells. The F_1 and F_2 data showed that the occurrence of the fuscoid cells was controlled by a dominant gene, which was symbolized *Fc*.

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15. A. "Fish-hook" mutation in rice

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A striking recurved and sharply pointed modification of the lemma appeared uniformly in an F_5 dwarf line. "Hooked", "parrot beaked" and "claw shaped" lemmas have been reported, but this is perhaps more extreme. The lemma tips will hook into clothing and support an entire panicle. This mutation could function for seed dispersal in wild rice. It would be a useful genetic marker if it segregates in a clear-cut manner.

The florets did not open in response to hot water treatment, probably becasuse the lemma and palea clamp together tightly. However, the seed set was fairly good. A cross with an IR 8 derived dwarf was obtained. The F_1 was a somewhat sterile non-dwarf with very slender spikelets which were not hooked. The F_2 was classified as: 47 hooked (some or all spikelets), 128 with the lemma tilted toward the palea but not recurved, and 10 normal apiculus.

It appears that "hooked" is controlled by a recessive gene; it was not expressed in the F_1 and constituted 1/4 of the F_2 population. However, this does not account for the intermediate "tilted" class. Duplicate dominant genes for "tilted" are suggested by the 15 : 1 ratio of "tilted" to "normal". Assuming that "hooked" is epistatic to "tilted", a combined ratio of 45 "tilted" : 3 "normal" : 16 "hooked" results. Agreement with expected is very close, but F_3 progeny tests were not conducted.

16. A big-grain gene, *Lk-f*, found in a Japanese local variety "Fusayoshi" and its character expression

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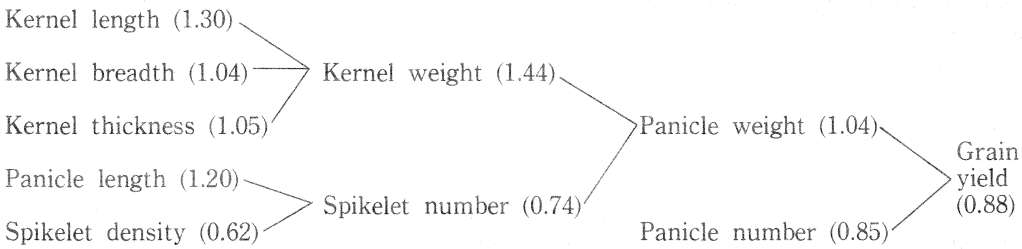
The grain size of rice is usually controlled by polygenes. As an exceptional case, a major gene controlling spikelet length was found in Japanese native variety, Fusayoshi. This variety had a

kernel (brown rice) length of 6.8 mm (kernel weight being 32 mg), while othe Japanese varieties have a kernel length of about 5 mm (kernel weight 20—24 mg).

The mode of inheritance of grain size was studied in the F_2 to F_5 populations of crosses between Fusayoshi and several other normal-grained varieties. The F_2's segregated into 1 short : 2 medium (=F_1) : 1 long grain classes, indicating that Fusayoshi had an incompletely dominant gene for long grain. The gene was symboled *Lk-f* tentatively. It was found to be linked with a gene for awn development, the recombination value being 7.4 to 8.4% and 7.6 ± 0.57% in pooled data. *Lk-f* may be used as a marker.

Pairs of isogenic lines for the *Lk-f/lk-f* locus were established from heterozygous F_7 lines of Shin 2 (normal) x Fusayoshi to examine the effects of the big-grain gene on yield component traits. In terms of *Lk-f/lk-f* ratios in various traits, the results were summarized as follows:



17. Inheritance of reduced culm number type and its character expression

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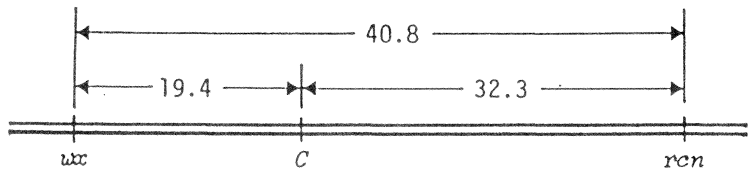
A mutant with a reduced culm number was found in the progenies of AC-11 (strain regenerated from A-5 Akamuro by anther culture) irradiated by gamma rays. In the F_2 population of its cross with the original strain, A-5 Akamuro, it was demonstrated that a single recessive gene, *rcn*, was responsible for both the reduced culm number and dwarfness under field conditions. Linkage analysis demonstrated that *rcn* was linked with the genes belonging to the first linkage group such as *wx* (glutinous endopserm) and *C* (chromogen for anthocyanin). The order of gene loci was obtained as follows:



The mutant gene, *rcn*, was epistatic to the dwarfing genes, *d-2* (ebisu dwarf), d_3, d_4, d_5 (bunketsu waito or tillering dwarf). *d-6* (ebisumochi dwarf), and *d-10* (toyohikari-bunwai tillering dwarf). The mutant plants showed a perfect seed set and differed from similar mutants showing complete or high sterilities which had been induced by gamma irradiation (Futsuhara and Yamaguchi 1963).

The expression of reduced culm number and dwarfism trait was affected by temperature conditions. Although the mutant produced only one or two tillers under field conditions, its growth was nearly normal when grown in a vinyl-house. When irrigated with cool running water during the growing period, the tillering and growth of the mutant were much suppressed and the reduced

culm number and dwarfism were expressed more clearly. This mutant would be useful in physiological and nutritional studies as an indicative material.

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Futsuhara, Y. and H. Yamaguchi, 1963. A radiation-induced mutant of less tillering in rice. Jpn. J. Breed. 13: 183-185.

18. Linkage relationship of long palea in rice

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Rao and Misro (1968) reported a floral variant of rice, long palea, where palea invariably outgrows the lemma. It was found to be due to dominant complementary genes. The second report on the occurrence of long palea was by the author (Thakur 1971). In an F_2 of a cross between two normal palea varieties, Ac 1224 and 7107 (a marker type, received from Mr. N. E. Jodon of U.S.A.) plants with long palea were obtained. This long palea was found to be a recessive trait. All F_3 progenies raised from long palea F_2 plants bred true. The F_1 plants of long palea and normal palea parents had normal palea and the F_2 segregation confirmed its recessive nature. A gene symbol *lp* was suggested.

This gene was found linked with *g* (extra-sterile glume) of linkage group IV (Thakur and Roy 1975). In order to clearly establish its linkage relationship, five long palea lines were crossed with representative markers of three linkage groups. Long palea segregated independently of *Cl* (clustered grains) and *C* (Chromogen gene) of group I, *pl* (purple leaf), *lg* (liguleless) of group II and *Rd* (red pericarp) of group III. In the F_2 of a cross between 7435 and Ext. 20-4 where long palea and rolled leaf segregated in coupling phases, linkage between long palea and rolled leaf, *rl* was observed. The χ^2 value (72.6) for independent segregation was highly significant and crossover value between these two markers was 11.8%.

The new linked genes (*lp* and *rl*) are tentatively assigned to group IV due to earlier report of linkage between *lp* and *g*. The allelic relationships of this *rl* gene with other *rl* genes reported earlier in Japonica rice need to be investigated.

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19. Allelic relationships of *Hg* and *Lh*

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A dominant gene *Hg* (hairy glume) was described by Nagao et al. (1960) which conditions presence of longer trichomes (hairs) on the surface of glumes, leaf margins and auricles of rice plant. Professor M. H. Heu gave us another mutant with similar morphology which he called *Lh* (long hair). The F_1 progenies from the cross of these two mutants had the mutant phenotype. An F_2 population of 158 plants consisted of mutant plants only. It is thus obvious that *Hg* and *Lh* are

allelic. The gene symbol *Hg* has priority and should be retained for this locus.

Reference

Nagao, S., M. Takahashi and T. Kinoshita, 1960. Genetical studies on rice plant, XXV. Inheritance of three morphological characters, pubescence of leaves and floral glumes, and deformation of empty glumes. J. Fac. Agric. Hokkaido Univ. 51: 299-314.

20. New mutations at old loci

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The lazy (*la*) mutant of rice is a well known marker of linkage group VIII. During last few years, we obtained five new lazy mutants which were induced through mutagenic treatments in different varieties of rice by three scientists (Table 1). These five lazy mutants were crossed with RGS 14 (our lazy tester stock) as well as with IR36 (non-lazy cultivar). The five F_1s with IR36 were normal indicating that all the five new lazy mutants are recessive. All five F_1s with RGS14, however, were lazy, thereby showing that the new mutations occurred at the previously known *la* locus.

Table 1. New lazy mutants of rice tested for allelic relationships with *la* (RGS 14)

RGS No.	Parent variety	Muitant obtained from
54	Balilla (Mutant No. 19)	R. Marie, INRA France
56	Cesariot (Mutant No. 28)	R. Marie, INRA France
73	Americano (Mutant No. 16)	R. Marie, INRA France
234	Norin 18	K. Pavithran, Calicut, India
259	CR115-32	R. N. Misra, CRRI, India

21. Search for new *g* loci unfruitful

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The recessive mutant *g* of rice, which conditions long empty glumes instead of rudimentary glumes of the grain, is a very useful marker of linkage group IV. Many rice varieties in our germplasm bank have long empty glumes. In search of new loci, conditioning long empty glumes, we studied the allelic relationships of the genes for long glumes in these varieties with the known *g* gene (RGS 10). A total of 49 varieties from 14 countries (Table 1) were crossed with IR36 had rudimentary glumes and with RGS 10. All the F_1's with IR36 had rudimentary glumes thus indicating that long empty glumes in all of these varieties are under recessive gene control. The F_1's of all of these varieties with RGS 10 had long empty glumes thereby showing that the recessive genes conditioning long empty glumes in all these varieties are allelic to *g*. Since these varieties come from many different countries, they are unrelated to each other. Thus the different mutational events must have occurred at the same locus as independent events. Our search for a new locus conditioning long empty glumes was unfruitful.

Table 1. Varieties from the IRRI germplasm bank with long empty glumes analyzed in this study

Acc. No.	Variety Name	Country of Origin
1349	P.I. 160764-2	China
1358	P.I. 160769-1	China
1454	P.I. 160863-1	China
1535	Fei Oh Chan	China
1537	Pien Chan Ying Tao	China
1583	Chang Ch'sang Hsu Tao	China
2809	Paiyautsuru 4	Taiwan
4000	Gungen-lang-shui-pe	Philippines
4212	Boenar	Indonesia
5999	Pankhari 203	India
6574	Pankiraj 258	Banglasesh
8268	Pappaku	Taiwan
8519	DZ 180	Bangladesh
8739	UCP 38	Bangladesh
11875	Kh. Nganh Tamay	Laos
15165	C	Ivory Coast
15900	Ebagagona	Senegal
17065	Napatsupai	Taiwan
17909	Ketan Manggaran	Indonesia
22422	ARC 12889	India
23240	Slab	Cambodia
23267	Srau Slap	Cambodia
23527	Khao Pick Deng	Laos
24182	Ba Ponar	Vietnam
25218	Luttu	Indonesia
25599	Pulut Kalesa	Indonesia
27038	Ase Pulu Haji	Indonesia
27050	Ase Pulut Cambang	Indonesia
28493	Tsao-fei-lai-feng	China
30593	Payon	Sierra Leone
30733	Jebowalogi (A2-102)	Liberia
30740	Kartiwegee (A2-76)	Liberia
30795	Maniqui (A2-95)	Liberia
30809	Netemah (A2-98)	Liberia
30864	Liberian Coll. D1-31	Liberia
31335	Liberian Coll. B-72	Liberia
31576	Fori Pakri	Bangladesh
33757	Taungpyanyin	Burma
33758	Taungpyanyin	Burma
35091	Pakhai Raj	India
38132	Pankhiraj 26-551	Bangladesh
41335	ARC 14035	India
42829	ARC 13994	India
43240	ARC 15858	India
46511	Pankhasail	India
46512	Pankheraj	India
47039	2-IS13-C77	Ivory Coast
50855	Kopike I (531)	Ivory Coast
53392	Fei-lai-feng	China

22. Mutant genes controlling starch synthesis in rice endosperms

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Starch is generally composed of two kinds of polysaccaride, amylose and amylopectin. In rice, genes controlling the proportion of amylose to amylopectin have not been identified although the *wx* is known to suppress amylose production. In maize, several genes which change the proportion of the two types of starches are known. The present authors found genes for low and high amylose contents among lines induced from Japanese cultivars; the low amylose mutant was obtained from the progeny of Norin 8 treated with ^{32}P (beta ray), and the high amylose mutant from Kinmaze treated with NHU.

The low amylose mutant was characterized by 'dull' endosperm. The starch was analyzed by gel filtration on a Sephadex G-75 column of starch components after debranching with *Pseudomonas* isoamylase. The content of amylose (Fr. I) in the dull mutant was about half of that of normal Norin 8. The contents of 'intermediate fraction' and Fr. II (longer unit chains of amylopectin) were similar to those in the waxy (glutinous) mutant and normal, and the content of Fr. III (shorter unit chains of amylopectin) was higher than that of normal, covering the reduction of amylose. However, the Fr. III/Fr, II ratios did not differ much among the normal, dull, and glutinous endosperms, suggesting that the distribution of unit-chain length of amylopectin remained unchanged in the mutant lines (Okuno et al. 1983).

The amylose content in F_2 and B_1F_1 endosperms was determined by Technicon Autoanalyzer with single grains. The F_2 endosperms from a cross between normal and dull segregated into 3 normal : 1 dull, and the B_1F_1 endosperms from dull/normal//dull segregated into 1 normal : 1 dull. The F_2 of a cross between dull and waxy mutants segregated into waxy, dull and normal types and the ratio was assumable to be 4 : 3 : 9 although the variation was continuous. The data thus indicated that amylose production in the dull mutant was controlled by a recessive gene, *du*, which was independent of *wx*. The normal, dull and waxy lines were intercrossed in different combinations, and the parental and reciprocal F_1 seeds were compared. The *wx* gene decreased amylose content in proportion to dosage in the endosperm, but *du* showed no such dosage effect.

Table 1. Properties of isoamylase-debranched endosperm starches from a high amylose mutant (EM-16) and control (Kinmaze)

Line	Distribution of components (%)				Fr. III/	Chain length at peak of	
	Fr.I	Int.Fr.	Fr.II	Fr.III	Fr.II	Fr.II	Fr.III
Kinmaze	20.4	3.7	16.7	59.2	3.5	38	14
EM-16	27.7	10.1	26.7	36.0	1.4	42	16

Furthermore, five mutant lines with an increased amylose content were found among those with floury endosperms, which had been induced from Kinmaze. Their amylose contents were 29.4 to 35.4 %, about twice as high as that of the normal line. One of them, EM-16, was used for analysis by gel filtration on a Sephadex G-75 column of starch components after debranching with *Pseudomonas* isoamylase. It showed an increased proportion of longer unit chains of amylopectin (Fr. II) as compared with the control (Table 1). The X-ray diffractogram of starch granules from the normal showed a type-A pattern which was typical of cereal starches, while that from the high amylose mutant showed a type-B pattern. The onset temparature of gelatinization of the mutant was much higher (63° — 69°C) than that of the normal line (52°C). The endosperm cells of the mutant were loosely packed by irregularly round-shaped starch granules, while those of the normal line were densely packed by polyhedral starch granules. The high amylose mutant seemed to have similar starch properties to those of the amylose-extender (*ae*) lines of maize. The F_2 seeds from a cross between normal and high amylose lines segregated into 3 normal : 1 high. The high amylose content was controlled by a recessive gene, which was symboled *ae* tentatively.

Reference

Okuno, K., H. Fuwa and M. Yano, 1983. A new mutant gene lowering amylose content in endosperm starch of rice, *Oryza sativa* L. Jpn. J. Breed 33: 387-394.

23. Endosperm mutants of rice induced by N-methyl-N-nitrosourea treatment of fertilized egg cells

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Several kinds of induced mutants for embryo or endosperm properties have been reported in rice recentry (Toda 1979; Amano 1981; Satoh and Omura 1981; Okuno et al. 1983; Yano et al. 1984). We maintain about 400 endosperm mutants of rice with several thousand morphological or physiological mutants, most of them induced by N-methyl-N-nitrosourea (MNU) treatment of fertilized egg cells. This method gives a high mutation frequency and little chimera formation (Satoh and Omura 1979). The spectrum of endosperm mutants made available in rice is now as rich as that in corn (Satoh and Omura 1981).

The frequency of various embryo and endosperm mutants induced by one-hour treatment with 0.75 mM MNU at different stages of fertilized eggs are given in Table 1. The frequency varied among types of mutation, being highest in the white core mutant, possibly reflecting the number of genes controlling each trait. The results of allelism tests supported this assumption. The mutation frequencies for certain types seemed to differ accordidng to the stages of treatment.

Table 1. The mutation frequency for embryo and endosperm properties induced by 0.75 mM MNU treatment for one hour at different hours after flowering

Mutant	Treatment stage (hour after flowering)																
	6	9	10	11	12	13	15	16	17	18	19	20	21	22	23	24	Total
Waxy				1			1	2		1		1	3	3			12
Dull	2	2	1		2	1	1		1		2		1		1	1	15
High amylose					1												1
Sugary				2						2		1		1	1		7
Shrunken-1[s]						1							1		1		3
Shrunken-2		3	1	1											1	2	8
Floury		2		1	2		3	1	1	1	1	1	1	4	1	1	20
White core	3	5	4	3	2	3	3	8	6	4	5	1	5	7	1	1	61
Giant embryo					1	1	2	3	2		1		1	1	1		13
No. of M_1	321	358	292	201	391	322	339	322	356	268	271	302	497	520	285	584	5619

Gene analysis for these mutants indicated that most of them were controlled by a single recessive gene with the exception of two floury mutants, one controlled by a single dominant gene and the other by two recessive duplicate genes (Satoh and Omura 1981). All glutinous mutants examined were allelic to *wx* located on chromosome 6. All dull mutants except for one showing an intermediate glutinous-ordinary property, were controlled by genes independent of *wx*. There were at least four dull loci, and one of them, *du-1*, was located on chromosome 7. Genes for three high amylose mutants were at the same locus. Most of sugary mutants were controlled by the same gene, *su*, which was located on chromosome 12. There were at least two loci for shrunken mutants, one being located on chromosome 3 (Yano et al. 1984). Two loci were recognized for giant embryo mutants, one belonging to chromosome 10. There were many different loci for floury or white core mutants, one of which was located on chromosome 5.

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IV. Regulation of gene action

24. Differential regulation of waxy gene expression in rice

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The evidence being accumulated shows that the genome of higher organisms appears to contain substantial amounts of genetic factors regulating the timing, place, and quantity of production of various gene products leading to diversified phenotypes. In order to examine the effects of different alleles on the gene expression at the waxy locus, the *Wx* gene product which controls the synthesis of amylose was isolated from endosperm starch of rice plants and analysed by electrophoretic techniques. The major protein (about 60,000 daltons) was absent in most of waxy strains and increased with the number of *Wx* alleles in triploid endosperms, suggesting that the major protein was the *Wx* gene product. In addition to *wx* alleles which result in the absence or drastic reduction of the *Wx* gene product and amylose, differentiation of *Wx* alleles seemed to have occurred among non-glutinous rice strains. At least two *Wx* alleles, Wx^a and Wx^b differing in efficiency in the production of the major protein as well as of amylose were detected.

Of special interest is the presence of different alleles, Wx^a and Wx^b, which regulate the quantitative level of the gene product. The two alleles may be regarded as the result of a mutation at a regulatory site(s) at or near the structural gene. Thus, biochemical approaches to the study of *Wx* protein in rice may throw more light on not only an important quality trait but also regulatory mechanisms of gene expression in rice. I intend to investigate whether the level of *Wx* protein is well correlated with amylose content in endosperm starch by using various induced mutants.

25. Unusual segregation patterns found at the *m-Ef* locus

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An early flowering isogenic line of T65 (Taichung 65) with gene $Ef\text{-}1^a$ (formerly symbolized E^a, hereinafter abbreviated as E^a), T65(7)E^a, was obtained from recurrent backrosses (7 times) of T65 with a native variety from northern China, Tatung-tsailai, used as the donor parent (Tsai 1961; Tsai and Oka 1965). Three isogenic lines with E^a plus *m-Ef* (formerly symbolized m^a, hereinafter abbreviated as *m*, since m^a and m^b were found to be identical), T65(7)E^am^a, T65(7)E^am^1, and T65(7)E^am^2, were also isolated from the backcrossing experiment. The three sib-lines showed a heading time much earlier (about 10 days) than that of T65E^a which was about 8 days earlier than T65 (with *ef-1* and m^+-*Ef*) in both the winter (first-crop) and summer (second-crop) seasons almost similarly; the *m* allele emphasizes the heading-promoting effect of E^a. However, the heading time of lines with *m* alone (without E^a or with *ef-1*) was earlier than that of T65 only a few days in winter, and did not differ from that of T65 in summer (Tsai and Oka 1966). The *m* locus was found to be linked with *Rc* (red pericarp), the recombination value being 23%; it belongs to the 4th linkage group (Tsai 1984).

The F_2 of T65(7)E^a × T65(7)E^am^a segregated for *m* into 1 early (E^am): 2 medium: 1 late (E^am^+) type, and the early type bred true in the F_3. The F_2 of T65(7)E^a × T65(7)E^am^1, 149 plants in total, also segregated similarly, but a part (6 of 28) of the early-flowering segregants did not breed true; the 6 F_3 lines, which were expected to be homozygous for earliness (E^am), segregated into early and late types, the ratio being approximately 8:1. Among the F_4 lines derived from 2 of the 6 segregating F_3 lines, those from 25 early-flowering F_3 plants bred true for earliness, but those from 5 late-flowering (E^am^+ type) F_3 plants showed a 1 early : 2 medium : 1 late ratio, suggesting that the late segregants from E^am plants had the m^+ allele.

The F_2 of T65(7)$E^a m^a \times$ T65(7)$E^a m^2$, both parents being early tuypes, produced 6 late-flowering off-types ($E^a m^+$ type) out of 280 plants. The F_3 lines from 3 of the F_2 off-types segregated into 6 early and 51 late plants, and those from the other 3 F_2 off-types segregated into 11 early and 47 late plants. Although the parental lines had no m^+ allele, the F_2 offtypes appeared as if they were heterozygous for the *m* locus (E^a/E^a m/m$^+$). About 2/3 of F_4 lines derived from 3 early- and 16 late-flowering plants of an F_3 segregating line showed a 1 early : 3 late ratio in each of them.

With regard to the occurrence of early- and late-flowering off-types due to some genic changes at the E^a locus, the present author has suggested intralocus recombination of subunits assuming that E^a was a complex locus, as the frequency of off-types was lower (1% or less; Tsai 1976). In the present case, however, the genic changes have a higher frequency and the *m* locus appears as if it is mutable. In families showing unusual segregation for heading time, a few morphological off-types (dwarfing and chlorophyll anomaly) were also found. It may be suggested that a controlling element or 'transposon' is attached to the *m* locus, as suggested in maize by McClintock (1951) and Fedoroff (1983). The evolutionary significance of the mutability of loci controlling heading time may also be noticed.

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V. Isoenzymes

26. Genic analysis for isozymes in rice

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Isozyme analysis in rice was pioneered by Chu (1967) and was extended to a wide range of materials by several workers as reviewed by Endo and Morishima (1983). Generally, wild species are more variable than cultivated species. So far, 14 enzyme species were studied and 40 loci were proposed (Second 1982). However, formal genic analysis was limited to some 12 loci. The 9 loci which were identified by deliberate crossing experiments by using the starch gel system are listed in Table 1. For the techniques employed, the reader is referred to Pai et al. (1973, 1975) and Second and Trouslot (1980). In addition, 4 loci encoding esterase were detected on acrylamide gels by Nakagahra (1977).

Each allele at the 9 loci listed specifies a single band, except for *Acp-1* alleles each specifying a set of three major and three minor bands. An inter-locus hybrid band is formed between *Pgi-1* and *Pgi-2*. *Pgi-1* and *Est-2* were linked (13% recombination) with each other and with *wx* (35% and 22% recombination, respectively); they were thus located on chromosome 6. In addition, temperature sensitive variants were detected for certain loci (Second 1982). Furthermore, the presence of regulatory genes was proposed to interpret organ specificity and temporal patterns of isozyme activity (Pai et al. 1973; Endo 1981a,b).

Table 1. Data for isozyme genes identified by formal genic analysis

Enzyme	Symbol[a]	Type	No. of alleles	Null form	Linkage with:	Reference
Acid phosphatase	*Acp-1*	dimer	8	Present	*Pox-2* (31—34%)	Pai et al. (1975) Pai & Fu (1977)
	Acp-2	monomer	3	"	*Acp-1*	Pai et al. (1975)
	Acp-3	monomer	2	"		"
Peroxidase	*Pox-1*	dimer	4	"		Pai et al. (1973) Pai & Fu (1977)
	Pox-2	monomer	2	"	*Acp-1* (31—34%)	Pai et al. (1973)
Catalase	*Cat-1*	tetramer	2	Unknown		Second & Morishima (1980)
Phosphoglucose isomerase	*Pgi-1*	dimer	4	"		"[b]
	Pgi-2	dimer	4	"	*Est-2* (13%) *wx* (35%)	"[b]
Esterase	*Est-2*	monomer	3	Present	*Pgi-2* (13%) *wx* (25%)	Authors' unpubl. data

a. *Px-1* and *Px-2* in original papers are symboled *Pox-1* and *Pox-2; Cat-A, Pgi-A* and *Pgi-B* in original papers are symboled *Cat-1, Pgi-1* and *Pgi-2,* respectively.
b. Partly due to authors' unpublished data.

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27. Geographical distribution of esterase genotypes of rice in Asia

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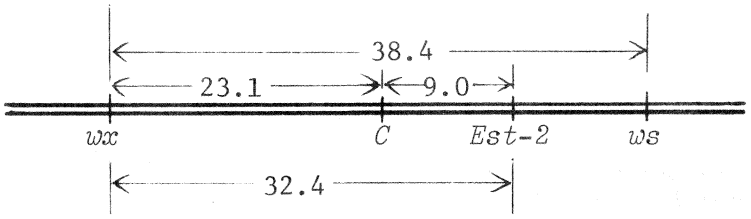
Esterase variations of Asian rice cultivars are determined by genes at 4 loci, *Est-1, Est-2, Est-3* (Nakagahra 1977), and *Est-4* (Unpublished). Alleles so far found are 2 at *Est-1* (*Est-1* and *Est-1*^{nul}), 3 at *Est-2* (*Est-2S, Est-2F,* and *Est-2*^{nul}), 2 at *Est-3* (*Est-3S* and *Est-3F*), and 3 at *Est-4* (*Est-4S, Est-4F,* and *Est-4*^{nul}). *Est-2* is located on Chromosome 7 (1st Linkage group) with the following gene sequence and linkage intensities (Nakagahra and Hayashi 1976).



There was no evidence for linkage between *Est-1* and *Est-2* or between *Est-2* and *Est-3* (Nakagahra 1977). The geographical distribution of genotypes for these enzymes was reported earlier with 1,190 indigenous rice cultivars (Nakagahra 1978). Since then, the present author and coworkers have explored different regions of Asia to obtain more material, and have studied a total of 2,752 native varieties.

With regard to the *Est-1, Est-2,* and *Est-3* loci, 12 genotypes were found as follows:

	Est-1	*Est-2*	*Est-3*		*Est-1*	*Est-2*	*Est-3*
1	1	2S	3F	7	nul	2S	3F
2	1	2S	3S	8	nul	2S	3S
3	1	2F	3F	9	nul	2F	3F
4	1	2F	3S	10	nul	2F	3S
5	1	nul	3F	11	nul	nul	3F
6	1	nul	3S	12	nul	nul	3S

The frequencies of these genotypes in 13 regions of Asia are shown in Fig. 1. North China (NC) and Japan (JP) were dominated by genotype 6, India-Sri Lanka (IN), Nepal (NE) and Bhutan-Northeastern India (AS) by genotype 1, and Vietnam (VT), South China (SC) and Malaysia (ML) by genotype 3. The frequencies of genotypes 1, 3 and 6 showed geographic clines clearly. Diversity was highest in the area covering Burma (BU), Thailand (TH), Laos (LA), and Yunnan Province of China. This area may be regarded as a diversity center for rice.

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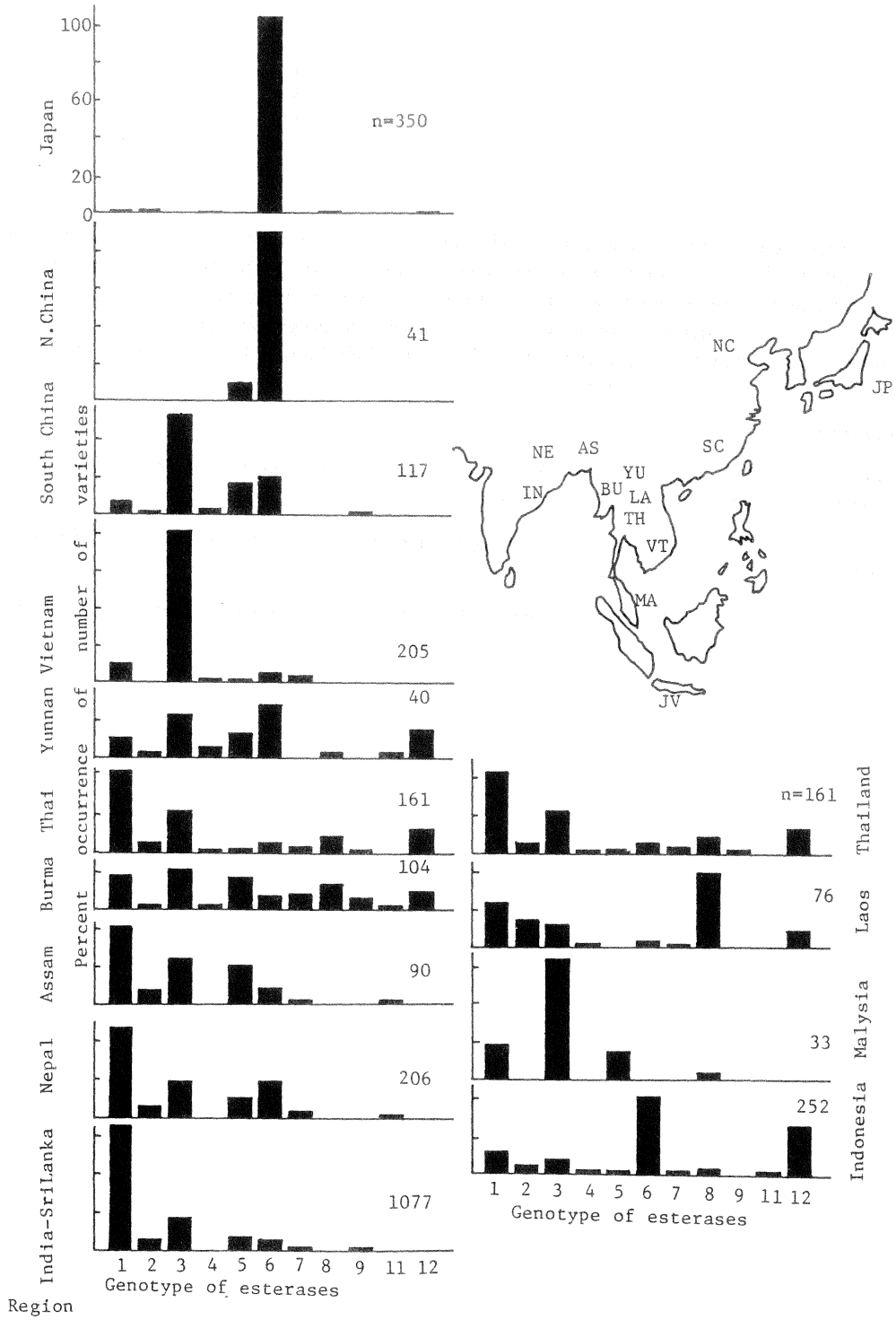

Fig. 1. Geographic distribution of esterase genotypes in native rice varieties in Asia

VI. Chromosomes

28. Chromosome pairing in a haploid rice

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A triploid plant isolated from a population of diploid Indica variety T 1242, on selfing, yielded a halploid plant which was used in this study. Young spikelets at proper stages were fixed in 1 : 3 acetic alcohol with a trace of ferric chloride and stored in 70% alcohol at low temperature. Slides were prepared by the simple acetocarmine smear technique and were made permanent by n-butyl alcohol method.

Out of 106 pollen mother cells examined at diakinesis, 96 (90.6%) exhibited chromosome association of 8 I + 2 II, followed by 10 I + II (10, 9.4%). None of the PMCs showed 12 I. The consistent synapsis indicates partial homology between the chromosomes. This is in conformity with the earlier observation of haploid rice by Hu (1957, 1960) and also with the report on chromosome association in a triploid rice by Rao and Reddi (1971).

The disjunction at anaphase I was highly irregular. The plant was completely pollen and seed sterile.

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29. Chiasma studies in genus *Oryza*

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Mode of reproduction and growth habit exert a great influence on chiasma frequency and hence genetic recombination in plants. Darlington (1937) and Mather (1943) pointed out that perennial and cross pollinated species in general exhibit lower chiasma frequency in comparison to their related annual self-pollinated relatives. Since the evolutionary change in many genera is from perennial to annual habit, most of the primitive perennial species exhibit lower chiasma frequency in comparison to the more evolved annual forms.

The present study of chiasmata frequency at diplotene and metaphase I in eleven diploid species of *Oryza* is in agreement with the above generaliations and reveals that the perennial species like *O. rufipogon, O. barthii, O. australiensis, O. granulata* and *O. collina* have lower chiasma frequency than their related annual species such as *O. nivara, O. sativa, O.glaberrima* and *O. cubensis*.

In the present study (Table 1), the chiasma frequency of *O. barthii* was found to be 1.31 and 1.11 at diplotene and metaphase I stages, respectively. Das (1961) while comparing the chiasma frequency of *O. rufipogon* and *O. barthii* also found that the latter species has exceptionally low chiasma frequency. In genus *Oryza,* series *sativae, O. barthii, O. rufipogon* and *O. cubensis* are the perennial wild species which are mostly cross-pollinated (Sharma 1964). The evolutionary trend in this series is towards self-pollination and annual growth habit. The chiasma frequency data

Table 1. Chiasma frequency at diplotene and Metaphase I in *Oryza* species

Species	Chiasmata at diplotene			Chiasmata at Metaphase I		
	No. of PMCs studied	Mean Xta per bivalent	Mean Xta per cell	No. of PMCs studied	Mean Xta per bivalent	Mean Xta per cell
O. australiensis						
(SC 452)	13	2.33	27.96	16	1.18	14.21
O. meyeriana (SC 306)	4	1.62	19.34	20	1.51	18.21
O. officinalis						
(SC 308)	21	2.63	31.56	18	1.83	22.0
(SC 279)	11	2.36	28.32	22	1.96	20.82
(SC 268)	8	2.83	33.96	12	2.32	27.84
O. collina	7	1.90	22.9	14	1.58	19.1
O. barthii	43	1.31	15.8	23	1.11	13.3
O. rufipogon						
(SC 145)	11	2.71	28.6	29	1.82	21.9
(SC 140)	—	—	—	29	2.54	30.5
(SC 159)	14	2.41	24.5	14	1.96	23.5
O. nivara						
(SC 31)	—	—	—	18	2.40	28.8
(SC 51)	21	2.56	30.75	—	—	—
O. cubensis	14	2.61	24.32	10	2.43	29.16
O. breviligulata	—	—	—	8	2.48	29.76
*O. glaberrima**						
(EC 21932)	12	3.06	36.72	16	2.63	31.56
*O. sativa**						
(A-18)	17	2.48	29.76	14	2.47	29.70
(T141)	13	4.30	51.60	22	3.31	39.88
(Norin-20)	11	2.66	31.92	20	2.41	29.92

* Self-pollinated, cultivated species.

obtained here are in accordance with this trend. Among these wild perennials, *O. barthii* maintains maximum primitive characters and is fully self-incompatible. *O. rufipogon* which is relatively more advanced exhibits higher chiasma frequency than *O. barthii.* Similarly, *O. cubensis* which is a weak perennial and has progressed in the direction of annual growth habit, exhibits higher chiasma frequency. Other annual species of this complex exhibit higher chiasma frequency which is indicative of their highly evolutionary advanced condition. Primitive species like *O. australiensis, O. collina* and *O granulata* exhibit lower chiasma frequency.

It is obvious from the foregoing observations that in the genus *Oryza,* the general evolutionary trend is towards development of mechanisms enhancing effective genetic recombination (higher chiasma frequency) and change from perennial to annual growth habit.

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VII. Linkage groups, trisomics and translocations

30. Trial construction of cytological map in rice

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Since twelve linkage groups corresponding to the haploid chromosome number of rice were proposed by Nagao and Takahashi (1963), the relationships between linkage groups and chromosomes have been examined by the use of segmental interchanges and primary trisomics (Iwata and Omura 1971a,b, 1975, 1967; Sato 1976, Sato et al. 1982). Interchange homozygotes used in these experiments were all determined cytologically and the chromosome numbering system proposed by Nishimura (1961) was followed. The results showed that three linkage groups, VI, IX and XII were associated with the 2nd choromosome, two groups, V and VII, were assigned to the 1st chromosome, and that the remaining seven linkage groups corresponded to seven other chromosomes, respectively. Accordingly, three linkage groups remained to be established. A new linkage group corresponding to the 7th chromosome was recently identified (Sato and Shinjo, in preparation). The remaining two groups will be established in near future. Several genes have been found to be located on the 4th and 12th chromosomes, to which no linkage group had been assigned (Yoshimura et al. 1982).

The construction of cytological maps is a problem left for studies in the future. The point of interchange is a useful cytological market. To determine the point of interchange and the position of centromere, Sato et al.(1980) observed pachytene chromosomes of hybrids between two reciprocal translocation homozygotes with different interchanged segments on the same chromosome, which show a configuration of six paired chromosomes, in comparison with those of their simplex heterozygotes. By examining the ratios of interchanged segments, the breakage points could be located on respectively chromosomes. The centromeres were represented by a small block of heterochromatin in the pachytene chromosomes, although it was not easy to determine the position when two or more heterochromatin blocks occurred on a single chromosome. Yet, a study of configurations of six paired chromosomes could bring about a provisional determination of the positions of centromeres. Thus, the cytological map for nine chromosomes indicating the point of interchange, centromere and several marker genes were constructed (see Fig. 2, (B) Current Linkage Map, p.19).

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31. Cytological identificaiton of extra chromosome in trisomics and location of the brittle-culm (*bc*) gene

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There trisomic lines, R78-14-8, R77-16-3 and R77-24-2, made available through the courtesy of the International Rice Reseach Institute, were investigated by using the new technique for root-tip chromosomes developed by Kurata and Omura (1978). The extra chromosomes embodied in the trisomic lines were chromosomes X, VII, and IV, respectively; the chromosomes were numbered according to their length in descending order. R77-16-3 had two pairs of nucleolar chromosomes. This confirmed Liou's analysis of pachytene chromosomes.

Each of the three trisomic lines were crossed to six marker stocks carrying *d-1* (Daikoku dwarf), *Dn* (dense panicle), *lg* (liguleless), *gl* (glabrousness), *g* (long empty glume), and *bc* (brittle culm), respectively. Since the F_1 plants had a low fertility due to the effect of an extra chromosme and Indica (trisomic) × Japonica (marker) crosses, only 6 cross-combinations yielded F_2 populations large enough for studying segregation ratios. One of them, R77-16-3 × *bc*, gave a ratio significantly deviating from 3:1 and fitting the trisomic ratio. This indicated that *bc* was located on chromosome VII.

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32. Use of primary trisomics of rice for associating linkage groups with respective chromosomes

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Twelve linkage groups corresponding to the haploid chromosome number of rice were suggested by Nagao and Takahashi (1963). However, these linkage groups have not been associated with the cytologically identifiable chromosomes and their independence has not been tested. We established all the twelve possible primary trisomics in the background of a disease and insect resistanct and widely grown indica variety, IR36. The extra chromosome of each of the trisomics was identified at pachytene stage of meiosis following the numbering system of Shastry, Ranga Rao and Misra (1960). Accoridng to this system the longest pachytene chromosome was numbered as 1 and shortest as 12. We studied the segregation of 22 marker genes in the trisomic progenies. In all we tested 120 out of 264 possible combinations involving 22 genes and 12 trisomics. On the basis of modified trisomic ratio technique we were able to identify marker genes for all the 12

chromosomes. Three linkage groups (VI, IX, and XII) were associated with chromosome 5 and linkage groups VII and V were associated with chromosome 9. New linkage groups for chromosomes 6, 8 and 10 were established. The relationships between the chromosome numbering systems of Shastry, Ranga Rao and Misra (1960), Nishimura (1961), Kurata and Omura (1978) and the linkage groups of Nagao and Takahashi (1963) are shown in Table 1. The table also shows the relationships between our trisomics and those of Iwata and Omura (1975). Detailed paper on these investigations will appear in volume 106 of Genetics.

Table 1. Relationships between various systems of numbering chromosomes, trisomics, linkage groups, and marker genes of rice

Chromosomes			Trisomics		Linkage groups	
Shastry, Ranga Rao and Misra (1960)	Nishimura, (1961)	Kurata and Omura (1978)	This study	Iwata and Omura (1975)	Nagao and Takahashi (1963)	Maker genes
1	3	K1*	1	O*	III	*eg, lax*
2	8	K2*	2	N*	X	*tri*
3	6	K6	3	B	I	*wx, ws*
4	5	K3*	4	M*	XI	bc_1, ch_1, *dl*
5	2	K9	5	L	VI, IX, XII	gh_1, nl_1, gl_1
6	4	K5	6	A	—	spl_1, rl_1
7	10	K11	7	F	IV	*g*
8	12	K7	8	D	—	v_8, *su*
9	1	K10	9	H	VII, V	dp_2, drp_2, *I-Bf*
10	7	K12	10	C	—	*pgl, fl*
11	9	K8	11	G	VII	*la*, z_2
12	11	K4	12	E	II	*lg, Pl*

* added by editor as per Iwata, Satoh and Omura (No. 34)

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33. Establishment of a complete trisomic series from a Japonica rice variety

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A series of 12 different types of trisomics had been isolated earlier from a Japonica variety, Nipponbare (Watanabe and Koga 1975). However, the investigation of karyotypes of extra

chromosomes by Kurata et al. (1981) revealed that four of the trisomics had the same extra chromosome (K10). Thus, these four were reclassified to be identical and the trisomics for the three longest chromosomes (K1, K2 and K3) were not present in our series. The extra chromosomes of the 9 types were identified by crossing them with marker stocks (Iwata and Omura 1975, 1976; Iwata et al. 1984). The relationships between Nagao and Takahashi's (1963) linkage groups and chromosomes numbered for designation of interchanged segments by Nishimura (1961) were established by using the trisomics and reciprocal translocation lines (Iwata and Omura 1971a, b, 1975, 1976; Iwata et al. 1984; Kinoshita et al. 1975; Sato 1976; Sato et al. 1973, 1975, 1982; Yoshimura et al. 1982).

The trisomics having chromosomes K1, K2 and K3 (chromosomes 3, 8 and 5, respectively, of Nishimura's designation), which were lacking in our series, were discovered recently, as types O, N and M, from the progeny of triploid plants of Nipponbare. The results from crossing experiments with these new trisomics are summarized in Table 1. In crosses with type M, marker genes *dl, ch-2* and *v-2* located on chromosome 5 (K3) showed trisomic ratios either in BF_1 or in F_2. Similarly, the N and O types were found to have chromosome 8 (K2) and 3 (K1) as extras, respectively. The respective correspondence of chromosomes 3, 8 and 5 to K1, K2 and K3 was thus established.

Table 1. Segregation for some marker genes located on chromosomes 3, 5 and 8 in BF_1 or F_2 of crosses with M, N and O types of trisomics

Cross combination	Segregation mode			χ^2		
Trisomic F_1 marker	Dominant	Recessive	Total	1 : 1	2 : 1	3 : 1
(M type × *d1*) × *d1*	17	6	23	5.26*	0.54	—
(M type × *ch-2*) × *ch-2*	19	9	28	3.57	0.02	—
(M type × *v-2*) × *v-2*	34	10	44	13.09***	2.23	—
(N type × *gh-2*) × *gh-2*	132	41	173	47.86***	7.22**	
(N type × *gh-2*) selfed	136	9	145	—	—	27.31***
(N type × *d1 gh-2*) × *gh-2*	209	60	269	82.53***	14.72***	—
(N type × *d1 gh-2*) × *d1*	349	335	684	0.25	65.72***	—
(N type × *ch-2*) × *ch-2*	220	180	400	4.00*	24.50***	—
(N type × *v-1*) × *v-1*	70	61	131	0.68	10.32***	—
(O type × *lax v-6*) × *v-6*	66	21	87	23.28***	3.31	—
(O type × *lax v-6*) selfed	549	34	583[a)]	—	—	114.24***
(O type × *lax v-6*) selfed	113	3	116[b)]	—	—	31.08***
(O type × *d-18*) × *d-18*	96	30	126	34.57***	5.14*	—

a) Segregation for *v-6*. b) Segregation for *lax*.
In this cross, only $v\text{-}6^+$ plants were used to observe the segregation for *lax*.
*, ** and ***: Significant at 5%, 1% and 0.5% levels, respectively.

The morphological features of the trisomic series are summarized in Table 2. The M type is completely self-sterile, but it set some seeds when pollinated by fertile disomic plants. The relationships among the trisomics, chromosomes and linkage gropus are given in the note by Iwata, Satoh and Omura that follows.

Table 2. Morphological features of 12 primary trisomics derived from a *japonica* cultivar, Nipponbare

Type	Short name	Morphological features
A	Pale	Pale green leaves at heading stage, fertile
B	Awned	Somewhat rough and lax panicles, awned spikelets
C	Small grain	Fine stature, bushy, small grain
D	Erectoides	Dark green leaves, erect panicles, short grain
E	Spreading	Open tiller, more or less narrow grain
F	Rolled leaf	Semi-rolled leaves, imperfect panicle emergence
G	Pseudo-normal	Nearly the same morphological features as disomics
H	Large grain	Dark green leaves, large grain, excess of nucleolar chromosomes
L	Short panicle	Short in height, short panicles, small grain
M	Sterile	Dark green leaves, short in height, perfectly sterile
N	Smooth glume	Dark green leaves, small and smooth glume, highly sterile
O	Grassy	Pale green and droopy leaves, bushy, small and narrow grain, highly sterile

(References — See the next note, No. 34)

34. The relationships between chromosomes identified cytologically and linkage groups

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Since the establishment of 12 linkage groups corresponding to the haploid chromosome number of rice by Nagao and Takahashi (1963), we have been investigating the relationships between the linkage groups and chromosomes mainly by using trisomics and reciprocal translocations (Iwata and Omura 1971a, b, 1975, 1976, 1984; Iwata et al. 1984; Kurata et al. 1981; Sato 1976; Sato et al. 1982; Yoshimura 1982). Recently we have established the relationships between all the chromosomes and linkage groups. The results from different series of studies are summarized in Table 1.

Table 1. Relationships among chromosomes, trisomics and linkage groups in rice

Chromosome		Trisomics		Linkage	Marker genes
RT	Karyotype	*Japonica*	*Indica*	group	
1	K10	H	Triplo 9	VII	*Bp*, Dn, dp-2*, drp-2**
				V	*I-Bf*
2	K9	L	Triplo 5	VI	*bgl*, d-1, nl-2*, ops*, v-10(t)**
				IX	*nl-1, ri, spl-7*, spl-8**
				XII	*gl*
3	K1	O	Triplo 1	III	*A, ch-5*, ch-6*, d-10*, d-18, eg*, fs-2, lax, Pn, Rd, rl-2*, spl-6*, v-6*, shr-1**
4	K5	A	Triplo 6	-	*d-B*, nal-2*, rl-1, spl-1**
5	K3	M	Triplo 4	XI	*bc-1, ch-1*, ch-2*, ch-3*, ch-7*, d-K-2*, dl*, drp-3*, drp-4*, fc*, op*, rl-3*, spl-3*, st1*, v-1, v-2*, v-5*, v-7*, z-3**
6	K6	B	Triplo 3	I	*C, ch-4*, Cl, dp-1*, spl-4*, v-3*, ws*, wx*
7	K12	C	Triplo 10	-	*du*, fl*, pgl*, Rf-1, rk-2*
8	K2	N	Triplo 2	X	*bl-1, bc-3*, d-K-1*, d-K-4*, d-W*, gh-2*, gh-3*, spl-2*, tri*
9	K8	G	Triplo 11	VIII	*d-C*, D-K-3*, d-t*, la, sp, v-4*, z-1*, z-2*, v-9(t)**
10	K11	F	Triplo 7	IV	*d-6, g, ge*, Rc, rfs*, spl-5*, v-11(t)**
11	K4	E	Triplo 12	II	*d-2, d-11*, lg, nal-1*, Ph*, Pl, rk-1*, ylm**
12	K7	D	Triplo 8	-	*d-51*, su*, ur-2(t)*, v-8*, z-4**

RT — Arbitrarily numbered on the basis of studies of reciprocal translocations by Nishimura (1961).
Karyotype — Numbered according to the length of somatic prometaphase chromosomes in descending order by Kurata and Omura (1978).
Trisomics, Japonica — Derived from Nipponbare, classified morphologically (Iwata *et al.*1970, 1984).
Trisoics, Indica — Estblished by Khush *et al.* (1984).
Marker genes — Shown by symbols used at Kyushu University, Dept. of Plant Breed.
Underline indicates genes whose location was determined by trisomic analysis. * indicates genes described by the staff of Kyushu University.

In the table (1st column), the chromosomes are numbered 1—12 tentatively according to Nishimura's (1961) system which is based on studies of reciprocal translocations. They are also numbered K1—K12 (2nd column) according to the length of somatic prometapohase chromosomes in descending order on the basis of karyotype analysis by Kurata and Omura (1978). The trisomics derived from triploid plants of Nipponbare (Japonica) are classified into types A—O by morphological features (Iwata *et al.* 1984). Those derived from an Indica strain, made available through the courtesy of Dr. G. S. Khush (Triplo lines; Khush et al. 1984), are numbered according to the length of extra chromosomes at pachytene in descending order. The linkage groups, I—XII, are those of Nagao and Takahashi (1963). The relationships among these different series were determined by segregation patterns for marker genes as listed in the table, particularly by trisomic analysis.

Linkage groups VII and V were associated with chromosome 1 which is the extra chromosome of the H-type trisomics. Genes *Bp, Dn* and *dp-2* (linkage group VII) and *I-Bf* (V) showed trisomic ratios in crosses with the H-type trisomics (Iwata and Omura 1975). Similarly, linkage groups VI, IX and XII were found to be associated with chromosome 2 which is the extra chromosome of the L-type trisomics (Iwata and Omura 1976). The linkage relations between *I-Bf* and genes belonging to linkage group VII still remain unknown. Linkage relations among genes of linkage groups VI, IX and XII were confirmed by both conventional and reciprocal translocation methods (Yoshimura et al. 1982; Sato et al. 1982).

None of the Nagao and Takahashi's linkage groups could be assigned to chromosomes 4, 7 and 12. However, several genes were located on them through trisomic analysis (Iwata and Omura 1975; Iwata et al. 1984; Yoshimura et al. 1982; Sato and Shinjyo 1978). Their linkage relations still remain unknown. The linkage groups assigned to respective chromosomes are shown in Fig. 1.

Many of the mutant genes listed in Table 1 were induced by irradiation or application of chemical mutagenes, but additional marker genes are necessary for preparing detailed linkage maps. The numbering system of rice chromosomes is not standardized as yet. It may be based on the length of chromosomes, but there is no complete agreement between the order of length of chromosomes at pachytene stage and that at somatic prometaphase. The adoption of an acceptable system would depend upon discussions among rice geneticists in the future.

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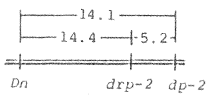
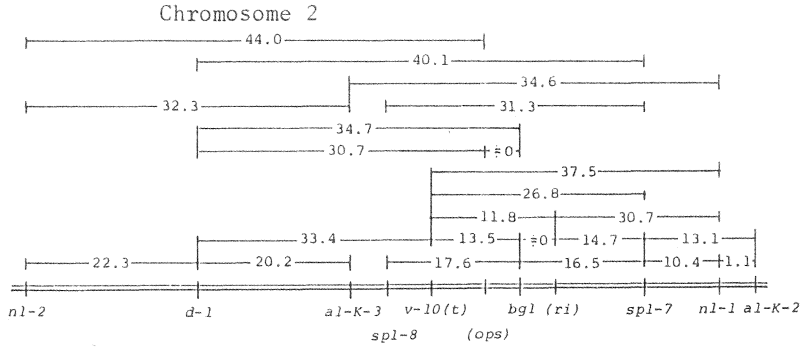
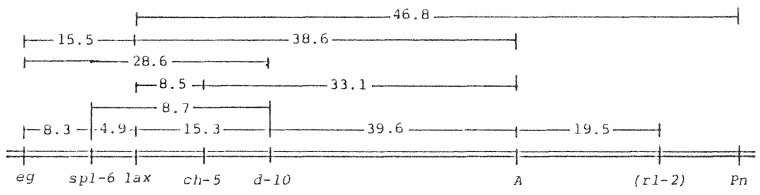
Chromosome 1



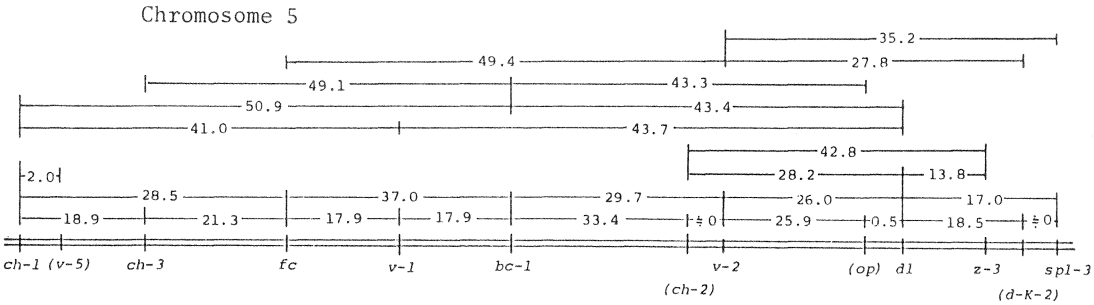
Chromosome 2



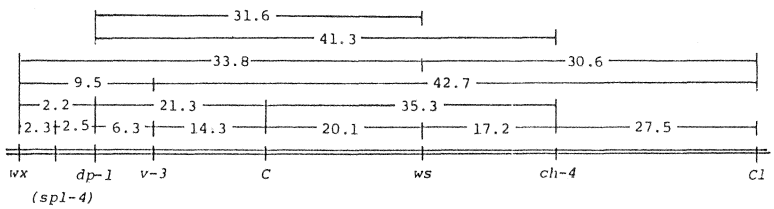
Chromosome 3



Chromosome 5



Chromosome 6



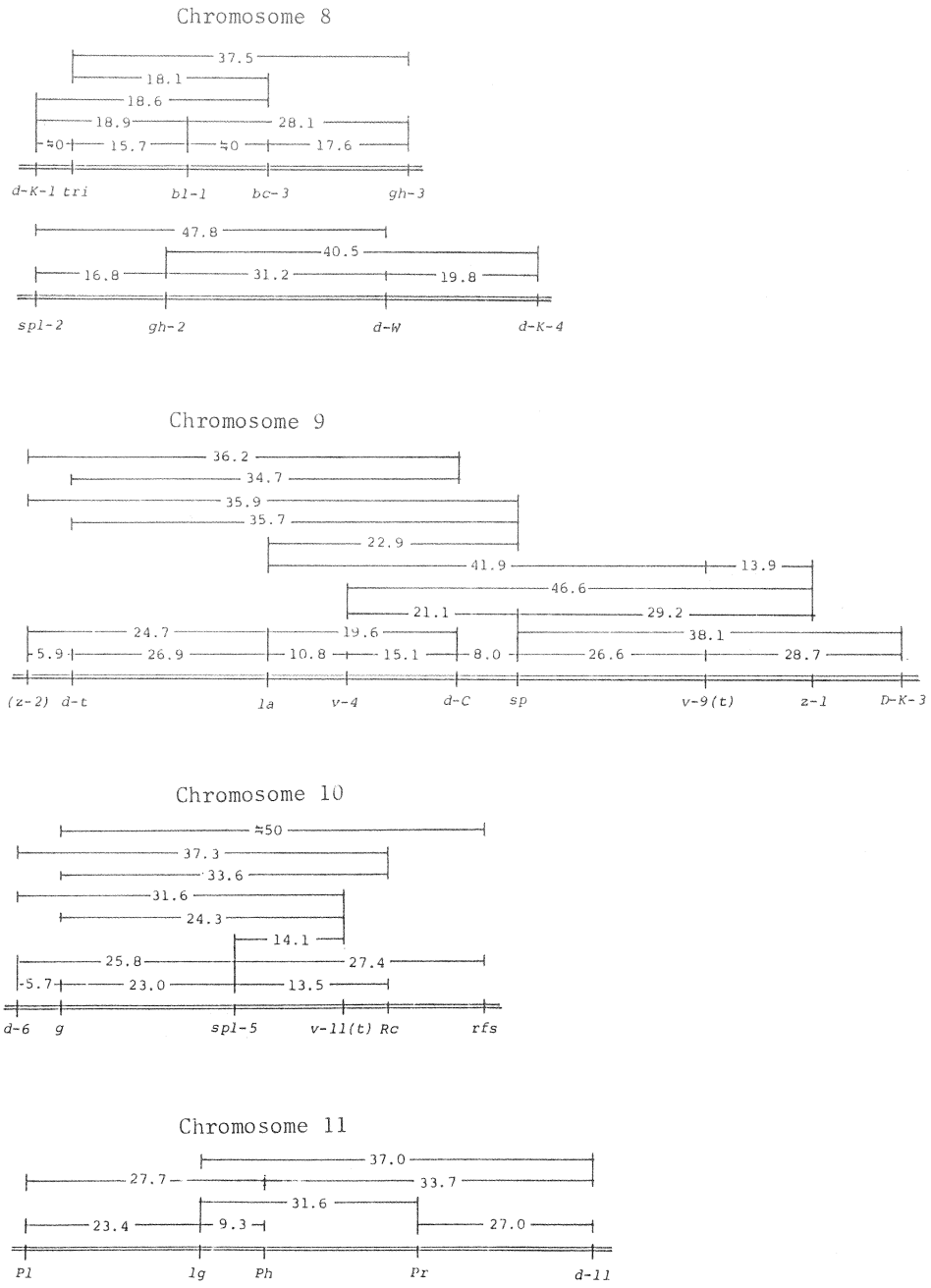


Fig. 1. Linkage maps for 9 chromosomes. Gene symbols follow those traditionally used at Kyushu University.

VIII. Technical notes

35. Embryo rescue of interspecific hybrids and its scope in rice improvement

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Wide hybridization in cereals is a significant plant breeding tool for the incorporation of desirable characters from wild into the cultivated species. Several accessions of three diploid wild species of rice, e.g., *O. australiensis* (Domin), *O. officinalis* (Wall), and *O. brachyantha* (Chev. et Roehr) are resistant to all biotypes of brown planthopper (BPH). In order to transfer genes for BPH resistance from these wild species we crossed them with three improved plant-type BPH susceptible lines of *Oryza sativa* L., e.g., IR1529-680-3-2, IR25587-109-3-3-3-3 and IR31917-45-3-2.

We used 3 accessions of *O. australiensis,* 5 accessions of *O. officinalis* and 1 accession of *O. brachyantha* as male parents in crosses with three breeding lines of cultivated rice. Due to cross incompatibility between the parents we obtained very low seed set and few hybrid seeds we obtained, were poorly developed. Most of the hybrid embryos started degenerating two weeks after pollination because of the incompatibility between the genomes.

To overcome the problem of degeneration of interspecific hybrid embryos we resorted to embryo rescue work. Spikelets, after 14 days of pollination, were taken and surface sterilized in sodium hypochlorite solution (35%) supplemented with 2 drops of Twin-20. After washing them in

Table 1. Embryo rescue of interspecific hydrids

Hydric combination	Embryos cultured	Embryos germinated	Percentage of germination
IR1529-680-3-2 x *O. australiensis* (1)	27	31	77.8
IR1529-680-3-2 x *O. australiensis* (2)	34	13	38.2
IR1529-680-3-2 x *O. australiensis* (3)	36	27	75.0
IR1529-680-3-2 x *O. officinalis* (1)	148	83	56.1
IR1529-680-3-2 x *O. officinalis* (2)	174	78	44.8
IR1529-680-3-2 x *O. officinalis* (3)	98	72	73.5
IR1529-680-3-2 x *O. officinalis* (4)	6	3	50.0
IR1529-680-3-2 x *O. officinalis* (5)	8	5	62.5
IR1529-680-3-2 x *O. brachyantha*	37	29	73.5
IR25587-109-3-3-3-3 x *O. australiensis* (1)	8	4	50.0
IR25587-109-3-3-3-3 x *O. australiensis* (2)	5	4	80.0
IR25587-109-3-3-3-3 x *O. australiensis* (3)	7	5	71.4
IR25587-109-3-3-3-3 x *O. officinalis* (1)	41	21	51.2
IR25587-109-3-3-3-3 x *O. officinalis* (2)	19	12	63.2
IR25587-109-3-3-3-3 x *O. officinalis* (3)	45	28	62.2
IR25587-109-3-3-3-3 x *O. officinalis* (4)	13	6	46.2
IR25587-109-3-3-3-3 x *O. officinalis* (5)	15	12	80.0
IR31917-45-3-2 x *O. australiensis* (1)	16	8	50.0
IR31917-45-3-2 x *O. australiensis* (2)	14	6	42.9
IR31917-45-3-2 x *O. australiensis* (3)	9	5	55.6
IR31917-45-3-2 x *O. officinalis* (1)	59	39	66.1
IR31917-45-3-2 x *O. officinalis* (2)	71	49	69.0
IR31917-45-3-2 x *O. officinalis* (3)	7	3	42.9
IR31917-45-3-2 x *O. officinalis* (4)	22	14	63.6
IR31917-45-3-2 x *O. officinalis* (5)	13	4	30.8
IR31917-45-3-2 x *O. brchyantha*	5	4	80.0

sterilized water, the delicate young embryos were excised and isolated under a stereomicroscope in an asceptic condition on a laminar flow bench. The isolated embryos were cultured asceptically on 1/4 MS medium and were incubated in the dark (25±1°C) until germination. The seedlings were kept in light incubation room up to three leaf stage and transferred to soil after growing in liquid culture medium for 10 days. The germination of hybrid embryos ranged from 38%—77%, 46%—80% and 31%—80% in the interspecific hybrids of three varieties, respectively (Table 1).

The hybrid plants of the three interspecific crosses are now growing. We shall examine their cytological behavior and pollen and seen fertility and study the possibility of gene transfer from these wild species to the cultivated rice.

36. A rapid method for identifying different dwarfing genes in rice

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A breakthrough in rice production has been attained through the development of semi-dwarf varieties. The semi-dwarf varieties in addition to having lodging resistance, have high nitrogen responsiveness. TN1 from Taiwan and IR8 from IRRI, Philippines, inherited their common recessive dwarfing gene from Dee-geo-woo-gen (DGWG). After the success of IR8 the breeders depended heavily on this source of short stature. This has resulted in narrow germplasm base of the world's rice crop, as far as dwarfing gene is concerned. Studies are underway for identifying new dwarfing genes. Allele tests between the new genes and the DGWG gene require 3-4 growing seasons as crosses must be made and F_1 and F_2 populations must be examined for plant stature. In this study a new approach to identify a dwarfing gene different from DGWG is reported.

Variety IR8, PR106, TN1, Basmati 370 and IR127—80-10—1 were planted in a single row in rice experimental area, Punjab Agricultural University, Ludhiana (India) in the later part of the rice planting season, i.e., on 15th July, 1982 in two replication. The row to row and plant to plant distance was 30 × 20 cm, respectively. Freshly prepared 100 ppm, solution of GA_3 was sprayed on five plants of each variety, in each replication, at the booting stage. Final observations on culm elongation after GA_3 spray were taken at full maturity of all the varieties. The results with respect to control and sprayed plants are given in Table. 1.

The data show that three varieties, e.g., IR8, TN1 and PR106, which have the same dwarfing gene from DGWG, responded similarly to the exogenous supply of GA_3. All these varieties showed a response of about 30 percent increase in culm elongation over their respective controls. Even the traditional tall variety Basmati 370 indicated a response of 14 percent increase in plant height. On the other hand plant height of IR127-80-10-1 at maturity showed no increase after the exogenous GA_3 spray (Table 1). This lack of response to GA_3 clearly shows that the dwarfing gene in IR127-80-10-1 is different from the DGWG gene present in other three dwarf varieties.

The dwarfing gene of IR127-80-10-1 ws found to be different from DGWG dwarfing gene by genetic analysis. The height of F_1 plants of the cross between TN1 and IR127-80-10-1 was 135 cm and compared to 83 cm and 94 cm for TN1 and I127-80-10-1, respectively. The F_2 of this cross showed transgressive segregation, the range being 60 cm to 165 cm. IRRI (1967) also reported that the dwarfing gene in CP-SLO (one of the parents of IR127) was different from the DGWG gene.

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Table 1. Plant height of different varieties before and after GA^3 application

Variety	Plant height (cm)		Percent increase in height over control
	Treated	Control	
IR8	86	65	32
TN1	88	70	26
PR106	88	68	30
Basmati 370	190	166	14
IR127-80-10-1	76	75	1

These results indicate that GA_3 responsive and non-responsive dwarf plants can be identified easily in the segregating populations of the crosses between varieties having CP-SLO gene and the DGWG gene. All the plants having least response to GA_3 would be having a dwarfing gene from CP-SLO and the ones having DGWG would respond to GA_3 application. Such an approach in rapidly identifying the different dwarfing genes can be extended to other dwarfing sources in rice also.

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37. High recovery of useful hybrid mutants in a lowland variety of rice

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A well adapted lowland rainfed variety of rice called Janki was treated with 0.4% EMS during rabi 1979—80. In M_1 generation 28 mature plants were obtained from a total of 1000 treated seeds. In M_2 generation very high spikelet sterility was observed and 1212 plants were harvested in bulk. In M_3 & M_4 the population was promoted by bulk method except that the plants looking like Janki were rejected. In the M_4 population, observations were recorded on 3120 plants. Tremendous genetic variability was observed with unusual combination of the mutant characters (Table 1).

Table 1. Hybrid-mutants of Janki with red and white rice in various plant height classes in M_4 generation

Plant height class (cm)	No. with	
	Red kernel	White kernel
1. up to 90	75	10
2. 91—106	155	14
3. 107—122	456	26
4. 123—138	439	22
5. 139—154	87	5
Total No. of plants	1212	77

In this method of breeding, it was expected that the sterility in M_2 would promote high rate of outcrossing. Mutants and the normal plants were expected to cross pollinate each other. This situation has led to the recovery of very high number of hybrid-mutants. It may be noted that Janki is a released and adopted variety for a water regime of about 1 metre depth in the state of Bihar in India. It has also done exceedingly well in several Eastern States of India and countries like Vietnam. The variety has excellent submergence tolerance, kneeing ability and resistance to

rice tungro virus and problem soils, but has coarse grains and red kernels. Several mutants have white kernel and fine grains in the background of semi-dwarf to semi-tall plant height and varying degrees of photoperiod sensitivity. These mutants are of great practical value. The breeding method followed is cost efficient and involves very little time and funds.

38. An improved technique for staining rice pachytene chromosomes

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Microscopic analysis of the pachytene chromosomes of rice was first carried out by Shastry et al. (1960), and this technique was further used by Sen (1963), Wu (1967), Khan (1975) and others with some modifications. These workers found that rice pachytene chromosomes were not easily stained by 1% aceto-carmine after the plain acetic alcohol fixation, and some of them added a trace of ferric chloride to there fixatives (Shastry et al. 1960; Das and Shastry 1963; Misra and Shastry 1967; Ranganadhacharyulu and Yesoda Rai 1974; Khan 1975; Reddi and Reddi 1977; Dolores et al. 1979; Sato et al. 1980). This treatment gave a deeper staining than the use of fixatives without iron (Yao et al. 1958; Bouharmont 1962; katayama 1966). In many cases, fixed materials were soon moved to 70% alcohol, but Khan (1975) kept his materials in the fixative with iron for two months or even longer and obtained improved staining of pachytene chromosomes.

Another difficulty has been insufficient spreading of the pachytene chromosomes. Good spreading seemed to depend on the choice of an appropriate stage of cell division and sufficient swelling of the cell. Pollen mother cells fixed in a fixative with iron do not swell well upon heating.

Wu (1967) developed a double mordant technique, by which these difficulties were circumvented. In this technique, a trace of ferric hydroxide is added to 1% aceto-carmine and to the fixative to which a trace of ferric chloride is also added. Not only chromosomes but cytoplasm also are overstained, especially when the slide is gently heated, and the residual stain can be removed by adding several drops of 45% acetic acid to one side of cover slip and blotting the excess fluid from the other side. Swelling of the cell and differential staining of chromosomes are then carried out simultaneously by heating the slide again evenly and gently up to a point just before boiling. No intended pressure needs to be applied directly to the cover slip except turning the slide upside down over a piece of blotting paper as soon as the heating is completed.

The pachytene chromosomes prepared in this way are well spread and reasonably differentiated (Chen et al. 1982). Among those who had studied rice pachytene chromosomes, Ranganadhacharyulu et al. (1974) was the only ones who used 45% acetic acid to remove excess stain.

The technique developed by Kurata et al. (1981) is quite different from the ones mentioned above. By their technique, anthers of proper size are treated with 75 mM KC1 or 0.5 mM uridine and macerated in a mixture of pectinase and cellulase. After being rinsed with distilled water, the anthers are smashed in a drop of fixative (3 parts methanol + 1 part acetic acid), flame-dried and Giemsa stained. Their micrographs have shown well differentiated configurations of chromosomes with prominence of centromeres. This technique may also be highly recommended for analysis of rice pachytene chromosomes.

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39. Utilization of microspore-derived plants for genetic analysis in rice

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This communication summarizes our results on genetic analysis of rice plants produced from anther culture (Chen et al. 1982, 1983). Four traits each controlled by a recessvie gene, glutinous endosperm (*wx*), narrow leaf (*nal*), ligulelessness (*lg*), and long glume (*g*), were selected as markers for the analysis. Previous studies showed that *nal* and *lg* were in linkage group II and their distance was approximately 19 map units (Yen et al. 1968); *wx* is in linkage group I and *g* in linkage group IV (Takahashi 1964). The anthers of hybrids heterozygous for two unlinked (*wx* and *lg*; *lg* and *g*) and linked (*nal* and *lg*) genes were cultured according to the method described by Chen (1977, 1978). Chromosome numbers of the plants obtained from anther culture were determined and the haploid and diploid plants were subjected to genetic analysis.

Progeny tests showed that all diploids but one were homozygous for both loci. The homozygous diploids were likely of microspore origin, where the haploid chromosome complement of microspores was doubled spontaneously during *in vitro* development. The exceptional plant was homozygous for lg^+ but was heterozygous for the waxy locus. Indirect evidence suggested that a

mutation at the *wx* locus in a spontneously doubled haploid cell during culture might have resulted in this exceptional plant.

Chi-square tests of the haploids and homozygous diploid plants further revealed the following facts: 1) In plants produced from all hybrids, the ratio of the dominant to recessive states of each character fitted the expected 1:1 gametic ratio, indicating that there has been no competition among the microspores with different genotypes during *in vitro* development. 2) In plants derived from heterozygotes for two unlinked genes, the frequency distribution of the four genotypic classes fitted a 1:1:1:1 ratio. 3) In plants derived from heterozygotes for two linked genes (*nal* and *lg*), the ratio of the four genotypic classes departed significantly from the 1:1:1:1 ratio, and the pooled data for haploids and diploids yielded a recombination value of 9.3 ± 1.47 percent between *nal* and *lg*. This value was quite close to that estimated from the F_2 data by the author (8.5 ± 1.33%) although it differed from a 19% value reported by Yen et al. (1968).

These results indicate that meiotic events, such as segregation, independent assortment, and recombination of linked genes, occurring in the donor plants can all be detected in the microspore-derived progeny. The significance of this work would be that it provides a theoretical basis for the utilizatin of another culture for rice breeding and demonstrates the feasibility of using the gametophyte derivatives of higher plants for gene mapping. Because fertilization is bypassed in the procedure, this new method of gene mapping may have some special advantages.

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40. Flavonoids as biochemical markers in the genus *Oryza*

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Our aim is to describe a new biochemical and molecular approach for studying polymorphism in *Oryza*. Flavonoid compounds are particulaly convenient for this purpose as they are widely distributed among plants and are chemically stable. They show a structural diversity due to differences in oxygenation, methylation and glycosylation processes. Most of the plant samples studied are those analyzed earlier for enzymatic diversity (Second 1982, 1983).

The leaf material to be sampled is immersed in an alcoholic mixture and after a fast purification step, the extract is analysed by two dimensional thin layer chromatography (flavonoid

patterns fixed by U.V. photographic process; Gonet 1973), or by high performance liquid chromatography (profiles integrated for the peak heights; Jay et al. 1983). In both cases, the phytochemical data were treated by multivariate analysis.

Two large groups of forms or species were recognized. One of them included the representatives of *O. sativa* complex, and the other was composed of *O. latifolia* complex. In the former, different flavonoid patterns could be corresponded to geographical origins or species. In the latter, it was possible to characterize the different genomes reported in this group. Moreover, *O. ridleyi* and *O. meyeriana* were distinguishable, and in the latter species the Chinese and South Asiatic forms could be separated.

The flavonoid patterns can be easily obtained from dry samples. The reports by the Phytochemical, Group of Lyon on various plants demonstrate the cogency of the biochemical markers in appreciating plant variability and its phylogenetic significance at species or infraspecific level (Jay et Gorenflot 1980; Jay et al. 1983; Reynaud et al. 1982).

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41. Callus induction and growth from different *Oryza* species

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The problems involved in the successful use of the protoplast culture method in rice breeding, namely, stable isolation of protoplasts, protoplast culture, and selection of fused cells, remain unsolved. The anther culture of rice was pioneered by Niizeki and Oono (1968) who obtained haploid and diploid plants from microspores. This technique was used to obtain promising true-breeding lines from F_1 plants at Hokuriku National Agricultural Experiment Station and Kamikawa Agricultural Experiment Station of Hokkaido, in Japan. Niizeki nand Kita (1981) succeeded in obtataining fusion of rice and soybean protoplasts by using polyethylene glycol (PEG)-high pH-high Ca^{++} techniques, but the culture of fused protoplasts and regeneration of plants were unsuccessful.

I attempted to induce callus formation from seeds of 40 strains belonging to 9 *Oryza* species and studied the proliferation of the calli. The seeds were surface-sterilized by immersion in 10%

bleach and were rinsed three times in sterile water. Then, Chu's medium containing 2×10^{-5} M 2,4-D was used for seed callus initiation. The callus growth was maintained by subculturing repeated every 10 days. Callus induction and proliferation were observed 40 days after plating.

All species except *O. latifolia* produced seed calli, but they clearly differed in callus induction and proliferation ability (Table 1). The seed calli from *O. sativa* and *O. glaberrima* showed better proliferation than those from other species. In *O. sativa*, subspecies *japonica* showed better proliferation than *indica*. A wide range of variation in callus growth or proliferation was also observed among *japonica* strains, suggesting that callus proliferation is under genetic control. The wild species generally showed a poor callus growth, the diameter of calli being nearly 0 to 3 mm after 40 days of incubation.

The techniques for protoplast fusion and culture of hybrid protoplasts are being studied. It seems necessary to screen the genotypes for a highly prolific callus growth and to obtain suitable media containing optimum levels of growth regulating substances for successful cell breeding.

Table 1. Comparison of callus growth among *Oryza* species

Species name	No. of strains	Diameter of callus (mm)											Mean
		0	1	2	3	4	5	6	7	8	9	10	
sativa	18					1	3	6	1	3	2	2	6.9
subsp. *japonica*	14						2	4	1	3	2	2	6.6
subsp. *indica*	4					1	1	2					5.1
glaberrima	5					1	1	2	1				5.7
punctata	4		1	2	1								1.9
minuta	1		1										1.2
officinatis	6		3	3									1.2
australiensis	2				2								3.2
latifolia	1	1											0.0
grandiglumis	1				1								2.8
brachyantha	2	2											0.1

Note: Callus diameter after 40 days from plating

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